AIR QUALITY MONOGRAPHS·VOL.3

INDOOR AIR QUALITY

A comprehensive reference book

Edited by
M. MARONI
B. SEIFERT
T. LINDVALL

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A COMPREHENSIVE
REFERENCE BOOK

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Vol. 3 Indoor Air Quality. A comprehensive Reference Book
M. Maroni, B. Seifert and T. Lindvall (Editors)

AIR QUALITY MONOGRAPHS-VOL. 3

INDOOR AIR QUALITY A COMPREHENSIVE REFERENCE BOOK

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Preface

The decision to produce this book arose from the recognition that Indoor Air Sciences have greatly evolved since the pioneering efforts of a few groups of scientists who started work in the seventies. During this evolution, it has become clear that achieving, maintaining, and restoring indoor air quality in buildings are complex tasks involving several key professionals — namely, architects, engineers, medical doctors, hygienists, chemists, biologists, environmental psychologists and others — and their respective scientific fields. The scientific progress in these disciplines has produced remarkable advances in knowledge related to indoor air over the last 20 years. For instance, toxicologists know a great deal about the effects of indoor pollutants; chemists are now able to detect indoor air pollutants at low levels undetectable in the past; engineers are able to model and predict pollution; medical doctors can diagnose specific building-related illnesses; biologists can identify and speciate microorganisms, their residues, and a number of allergenic materials; environmental psychologists can assess environmental perceptions and discomfort. However, most of these specific acquisitions of knowledge have remained separate from each other and confined to the specialists within their respective field of science. As a consequence, existing books are mostly monothematic and there is a substantial lack of comprehensive and integrated views about indoor air quality.

It was thus a challenge to offer the reader a reference book which, in a single volume, would provide a complete panoramic view of every expertise necessary for a comprehensive understanding and management of indoor air quality. Such an ambitious goal was pursued by gathering experts active in different international groups and selecting the material already available which could present all the issues involved.

This strategy was supported by the active involvement of international programmes, institutions, and organizations that had produced important material and publications in this area. The editors (and the contributing authors) are very grateful to all these bodies for having made available essential documents and for consenting to the reproduction of — sometimes integral — extracts of their publications. Among the principal sources used to

compile this book, special mention deserves to be made of the NATO/CCMS Pilot Study on Indoor Air Quality, the European Collaborative Action "Indoor Air Quality and its Impact on Man" (formerly COST, 613), the World Health Organization programme on IAQ, the United States Environmental Protection Agency, the International Academy of Indoor Air Sciences, and the International Society of Indoor Air Quality and Climate.

The reader familiar with the scientific production of these international groups will not find great novelties in the chapters of this book and, perhaps, will have already listened to some of the arguments when they were presented at the Indoor Air Conferences or in other workshops. However, we believe that the "added value" of the enterprise is the combination of materials from sparse (and sometimes difficult to find) sources and the matching of information targeted to hygienists and medical doctors with other information targeted to engineers, architects, chemists, etc. Thus we hope to have succeeded in our goal which was to create a comprehensive multidisciplinary (or interdisciplinary) book.

On account of the characteristics of this book, the editors believe that it will be particularly useful for teaching purposes and as a reference for advanced educational programmes on indoor air at universities and at post-graduate level. Some chapters contain formative and educational messages that should be basic elements in the professional formation of architects, engineers, and public health professionals. Other chapters contain reference documentation, the contents of which even an expert is unlikely to use every day, but which become extremely important when a specific problem (e.g., measurement of a pollutant) is addressed. Therefore, in this respect, the book presents two main facets — basic education and advanced reference material — and its use for educational applications would benefit from proper guidance by the teacher.

Although the preparation of the book has been a tremendous effort for all contributors, it has also been a great professional experience and an opportunity to strengthen our friendship with all those colleagues who have contributed to the enterprise. We are grateful for their patience and determination that has allowed a productive outcome.

The Editors:
Marco Maroni
Bernd Seifert
Thomas Lindvall

Acknowledgements

The Editors wish to thank the NATO-Committee for the Challenges of Modern Society Pilot Study on Indoor Air Quality, the European Collaborative Action "Indoor Air Quality and its Impact on Man", the World Health Organization, the U.S. Environmental Protection Agency, the International Academy of Indoor Air Sciences, the International Society for Indoor Air Quality and Climate and the Nordic Committee on Building Regulations for having supported the preparation of this book by providing technical material and expertise.

In addition, thanks are expressed to the National Institute of Occupational Health of Norway, the National Institute of Public Health of Norway, the Institute of Occupational Health of the University of Milan, the Institut de Recherche en Santé et en Sécurité du Travail du Québec, the Research Triangle Institute at Triangle Park (NC-USA), the Institute for Water, Soil and Air Hygiene of the Federal Environmental Agency in Berlin, Germany, The National Research Council of Italy, for having supported the NATO workshops that have produced a part of the material of this book.

Special thanks are due to ENEL S.p.A. Directorate for Energy Production and Transmission that has generously supported the workshop "Indoor Air Quality for Health" held in Civitavecchia in September 1992, where this book has been drafted and collegially discussed.

The Editors acknowledge the contribution provided by the International Centre for Pesticide Safety in the preparation of the book. Skilful and dedicated efforts have been provided by Dr. M.G. Colombo for the editorial coordination, Dr. V. Bertolini for language and technical supervision, Mr. G. Preti and Mr. F. Visigalli for the preparation of tables and illustrations, Mrs. P. Sartorelli, B. Villa and N. Spedicato for typing the text. Eng. A. Michelazzi, Dr. R. Iachetta, Eng. C. Onestini and Mr. L. Baroni and his staff at ENEL in Civitavecchia are thanked for their contribution to the preparation of the first draft of the book.

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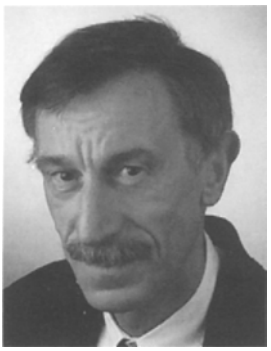
Marco Maroni is a Professor of Occupational Health at the School of Medicine of the Università degli Studi di Milano and Professor of Environmental Hygiene at the School of Architecture of the Politecnico di Milano. He has conducted research in risk assessment of xenobiotics to human health, neurotoxicology, indoor air quality, pesticides. Professor Maroni is author of about 200 scientific papers published in international journals, several monographs and chapters in books. He is also the co-editor of several books.

Professor Maroni has directed the NATO/CCMS Pilot Study on "Indoor Air Quality" and other international research projects. His scientific assignments include: member of the International Academy of Indoor Air Sciences, Vice President of the International Society of Indoor Air Quality and Climate, member for Italy in the Steering Committee of the Collaborative Action Indoor Air Quality and its Impact on Man of the European Union, member of the Advisory Board of the European Center for Environment and Health of the World Health Organization, chairman of the Scientific Committee on Pesticides of the International Commission on Occupational Health, member of the National Commission for Indoor Pollution of the Ministry of Environment of Italy, member of the National Commission for Pesticide of the Ministry of Health Italy.

Professor Maroni is a member of the editorial board of the following international journals: *Archives of Environmental Health, Central European Journal of Public Health of Praha, Indoor Air.*



Name: Bernd Seifert
Date of birth: 1941
Country: Germany

Dr. Bernd Seifert was educated at the Collège Français in Berlin. After studying chemistry from 1960 to 1966 and roman languages from 1966 to 1968 he obtained a Ph.D. in chemistry from Berlin's Technical University (TUB) in 1971. Dr. Seifert started his professional career as an Assistant Professor of Analytical Chemistry at TUB. In 1972, he joined the Institute for Water, Soil and Air Hygiene in Berlin (part of the Federal Environmental Agency) where he is currently Director and Professor, Department of Air Hygiene.

After having devoted much of his early research work to the development of analytical methods for ambient air pollutants, Dr. Seifert's interest turned to indoor air in the late seventies. In 1987, he was President of Indoor Air '87, the 4th International Conference on Indoor Air Quality and Climate held in Berlin, and became Chairman of the EC Collaborative Action "Indoor Air Quality and its Impact on Man".

Currently, Dr. Seifert is President of the International Academy of Indoor Air Sciences and a member of the Editorial Board of the scientific journal *Indoor Air*. He is also an international councillor of the International Society of Exposure and an Associate Editor of the journal *Exposure Analysis and Environmental Epidemiology*. In 1993 he was appointed Chairman of the Subcommittee on Indoor Air of the International Organization for Standardization (ISO).

On many occasions, Dr. Seifert has served as an expert to the Commission of the European Communities and as temporary advisor to the World Health Organization on all kinds of questions related to the field of air quality. Besides these international commitments, Dr. Seifert is engaged in numerous activities at the national level, especially in the work of the German Commission for Clean Air which granted him an Honorary Award in 1994. He is the author, co-author and editor of about 100 publications.

Name: Thomas Lindvall
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Thomas Lindvall, M.D., has a Doctors degree in Medical Sciences, and is a Professor of Hygiene at the Karolinska Institute and the Institute of Environmental Medicine, Stockholm. He has conducted research in sensory physiology and psychology, respiratory physiology and asthma, community noise, ambient air pollution, indoor air quality, occupational ergonomics, thermal stress, building hygiene, epidemiology and questionnaire surveys. He has published about 200 scientific papers, books and book chapters.

Professor Lindvall's scientific assignments include, amongst others: President of the Third International Conference on Indoor Air Quality and Climate, Chairman of the Final Editorial Task Force on the WHO Air Quality Guidelines for Europe, Co-President of the First International Conference on Healthy Buildings, President of the Fifth International Congress on Noise as a Public Health Problem, former Member of the Nordic Committee for Building Regulations (NKBi), Member and Fellow of the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Member of the Steering Committee for the Second International Conference on Healthy Buildings, Chairman of the Nordic Noise Group at the Nordic Council of Ministers, Member of the Steering Committee for the European Collaborative Action "Indoor Air Quality and Its Impact on Man", Founder of the International Academy of Indoor Air Sciences, Founder and former President of the International Society of Indoor Air Quality and Climate. In Sweden, amongst others: Chairman of the Swedish National Project on Environmental Medical Surveillance based on Biological Indicators, Chairman of the Swedish National Project on Risk Assessment, Health and Environment, former Member of the Swedish Environmental Research Board and former Chairman of its Scientific Committee on Community Noise, Chairman of the Swedish National Allergy Research Program Committee, and member of the Board of the Swedish Foundation for Caring and Allergy Research.

Professor Lindvall has been a member of the Editorial Board of the following international journals: *Environment International, Franklin Institute Journal*, and *Indoor Air*.

Authors

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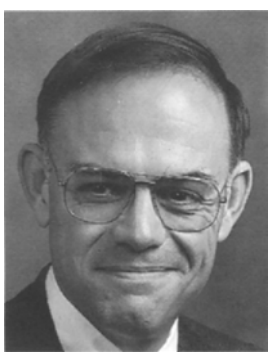
Robert Axelrad is currently the Director of the Indoor Air Division at the U.S. Environmental Protection Agency (EPA). He has been responsible for the development of EPA's indoor air program since 1988. The program emphasizes non-regulatory guidance and training approaches to provide architects and engineers, building owners and managers, building occupants, product manufacturers and the general public with practical information on preventing and resolving indoor air quality problems. He initiated and directed the development of Building Air Quality: "A Guide for Building Owners and Managers", the most widely used indoor air quality problem-solving guidance document available in the U.S., as well as more than 20 other EPA publications and training courses on IAQ. He also initiated and funded a landmark assessment entitled "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorder", which classified second-hand tobacco smoke as a Group A, or known, human carcinogen and developed EPA's policy recommendations and outreach materials on second-hand smoke.

Robert Axelrad serves as the lead Co-Chair on the Interagency Committee on Indoor Air Quality (CIAQ), a coordinating organization of more than 21 Federal agencies, and has served since 1989 as Co-Chair of a NATO Committee on the Challenges of Modern Society Pilot Study on Indoor Air Quality. Previously, Mr. Axelrad worked on hazardous and solid waste, water quality and pesticide issues in both governmental and non-governmental roles. He received a B.A. in Political Science from Pennsylvania State University in 1974.



Name: Michael A. Berry
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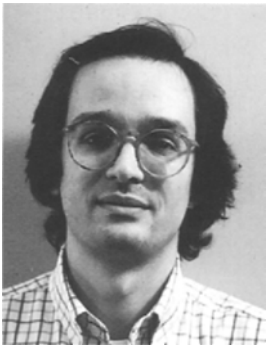
Dr. Michael A. Berry is the Deputy Director of EPA's Environmental Criteria and Assessment Office at Research Triangle Park, North Carolina. His career in public health began in 1963 when he conducted a study of Arab refugee camps in the Middle East. He subsequently worked in Viet Nam (1967–1968),

where he dealt with many public health issues related to the civilian population and refugees. Since 1969, he has specialized in the field of environmental management and protection, focusing on air pollution, the assessment and management of toxic substances, and indoor environments. From 1986 to 1992, Dr. Berry served as EPA's manager for indoor air research.

Dr. Berry is EPA's Campus Executive for the University of North Carolina at Chapel Hill. He is an Adjunct Associate Professor in the Department of Environmental Science and Engineering, School of Public Health, and in the Kenan-Flagler Business School at the University of North Carolina, where he teaches courses in environmental management and policy. He received his Ph.D in Public Health from the University of North Carolina at Chapel Hill. He holds a graduate degree in management from Duke University's Fuqua School of Business and an M.S. and B.S. degree in Mathematics from Gonzaga University. He is a member of the Air and Waste Management Association and a member of the International Academy of Indoor Air Sciences.



Name: Francesco Bochicchio
Date of birth: 1958
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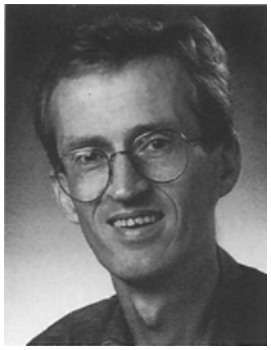
Dr. Francesco Bochicchio was born in Bari (Italy) on 13 December 1958 and lives in Rome (Italy). He obtained a classic high school degree in 1977 and a degree in Physics at the University of Rome in 1984, presenting a thesis on cosmology and elementary particles. Later he dealt with health physics, and attended the Specialisation School on Health Physics at the University of Rome (1985–1987). In October 1987 he started to work as a physics researcher in the Physics Laboratory of the Istituto Superiore di Sanità (National Institute of Health) in the Radioactivity Section directed by Prof. G. Campos Venuti.

Dr. Bochicchio has worked on many aspects of environmental radioactivity and indoor air quality, including exposure estimation, risk assessment and measurement techniques, especially regarding radon, thoron and their decay products. He lectures on natural radioactivity and radon in dwellings at the Specialisation School on Health Physics at the University of Rome (1989 to 1994). He has taken part in international meetings and workshops on environmental radioactivity, health physics and indoor air quality; planning and realisation of the Italian national survey on population exposure to natural radioactivity in dwellings (1988 to 1994); the working group of the NATO/CCMS Pilot Study on Indoor Air Quality (1990 to 1992); the World Health

Organization working group on "Indoor Air Quality: a risk based approach to health criteria for radon indoors" (1993).



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Prof. Dr. ir Bert Brunekreef has an academic degree in Environmental Sciences from the University of Wageningen (1979), and a doctorate (Ph.D) in environmental epidemiology (1985). He is currently Professor in Environmental Health with the Department of Epidemiology and Public Health of the University of Wageningen. His research has focused on health effects of ambient and indoor air pollution, and on respiratory epidemiology in general over the last several years. He has been involved as project leader in most of the projects in the field of environmental health of the Department over the last 12 years. In addition, he spent a year with the Harvard School of Public Health in 1986/1987, collaborating with the Professors B.G. Ferris Jr., F.E. Speizer, J.D. Spengler, D.W. Dockery and J.H. Ware of the Respiratory Epidemiology Program in research on health effects of ambient and indoor air pollution. He is a member of the European Concerted Action (formerly COST 613/2) on Air Pollution Epidemiology, and he served as chairman of the Health Effects Assessment working group of this concerted action. He is also a member of the European Concerted Action "Indoor Air Quality and its Impact on Man" (formerly COST 613). He is currently a councillor of the International Society for Environmental Epidemiology (ISEE), and an active member of the Exposure Analysis (ISEA), the American Thoracic Society (ATS) and the European Respiratory Society (ERS).

Name: Brian Flannigan
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Brian Flannigan is Reader in Microbiology at Heriot-Watt University, Edinburgh, U.K., where his research has been concerned with deteriogenic and toxigenic moulds. The impact of microbial growth in buildings on indoor air quality is a major research interest. Much of his and co-researchers' work on moulds, bacteria and mites in damp housing has been carried

out in liaison with the Building Research Establishment of the U.K. Department of the Environment. He also has a particular research interest in the allergic/toxigenic cause of malt worker's lung, *Aspergillus clavatus*, and is working on development of rapid methods for its detection in airborne particulates. He is a former President of the Biodeterioration Society and of the International Biodeterioration Association, and a member of the IUMS International Commission on Mycotoxicology (1983–1993). He was a member of an expert working group which recently produced a report of Biological Particles in Indoor Environments as part of the EC COST Project 613 on "Indoor Air Quality and Its Impact on Man" and is chairman of an International Society of Indoor Air Quality and Climate task force on moisture problems affecting indoor air quality.

Name: Matti Juhani Jantunen
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Dr. M.J. Jantunen received his degree in Public Health at the University of North Carolina. He is currently Research Professor and Director, KTL-Division of Environmental Health, Kuopio, Finland, and Coordinator, CEC European Concerted Action on Air Pollution Epidemiology (1990–1994).

Dr. Jantunen is a founding member of the Board of Directors of the International Society of Indoor Air Quality (ISIAQ). He was also vice-president of the Indoor Air '93 conference, and currently serves on the Steering Committee of the CEC ECA "Indoor Air Quality and Its Impact on Man", and Steering Committee of the CEC ECA Air Pollution Epidemiology. He has also served on WHO working groups on Indoor Air Quality, particularly, biological contaminants, and man-made mineral fibres and asbestos. His research activities have been on the emissions of solid-fuel power-plants, atmospheric chemistry of organic mutagens, Chernobyl fallout and its dispersion in urban environments, and indoor air quality, especially microbial growth and radon. He has written a textbook on Air Pollution and on Global Climate Change. His scientific publications consist of 48 original articles in peer-reviewed international journals and proceedings; 32 congress abstracts; 54 other scientific and professional articles; and four books (as author or editor).

Name: Helmut Knöppel
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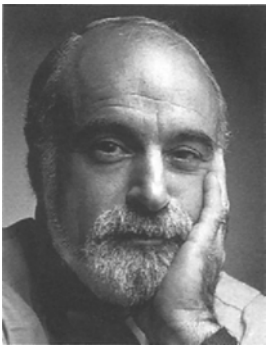
Dr. H. Knöppel obtained his degree in Physics (Diplom-Physiker) at the University of Bonn in 1961, followed by a Doctorate in physical chemistry (Dr. rer. nat.) and worked as a scientific assistant (1961–65). In 1966 he became a scientific officer of the Commission of the European Communities (CEC) at the Joint Research Centre in Ispra, Italy. Since 1972 he has been working in environmental analytical chemistry (determination of volatile organic compounds in air and water). Since 1981 he has been head of the Environmental Chemicals Sector in the Chemistry Division of the JRC in charge of indoor air quality activities.

Since 1983 Dr. Knöppel has been principal scientific officer, responsible for the European Concerted Action "Indoor Air Quality and Its Impact on Man". Since 1990, he has been head of the unit "Environmental Chemicals — Indoor Pollution" in the JRC's Environment Institute; and editorial advisor to the international journal *Indoor Air*. Since 1992, he has been a member of the International Academy of Indoor Air Sciences. He is also a commission representative in WHO and NATO-CCMS Working Groups on indoor air quality.



Name: Hal Levin
Date of birth: 1941
Country: USA

Hal Levin is a research architect in private practice. His firm, Hal Levin & Associates, is located in Santa Cruz, California. He has been consultant to architects and building owners on indoor environmental quality in designs for new buildings and renovations and has investigated problem buildings since 1978. He has extensively advised private organizations, individuals, and state, federal, and foreign government agencies on indoor air quality. In 1988 he developed indoor air quality guidelines for the new EPA headquarters.

Hal Levin received degrees in English and in architecture from the University of California, Berkeley in the 1960s. He returned to UC Berkeley in 1978 as research specialist at the Center for Environmental Design Research where he studied indoor environments and their impact on occupant health and

comfort until 1989. He also taught in UC Berkeley's Department of Architecture and in the Board of Environmental Studies at UC Santa Cruz.

Levin has written more than 65 published articles, papers, or book chapters on indoor environmental quality and is a frequent speaker at courses and seminars both in North America and Europe. He edits and publishes the *Indoor Air Bulletin* providing critical analysis of information on indoor environmental problems based on the latest scientific and technical developments. From its founding in 1988 until 1991 he edited the *Indoor Air Quality Update*. In all, he estimates his published writings on indoor air exceed 2000 pages.

His public service is extensive and includes chairmanship of the Advisory Committee, Monterey Bay Unified Air Pollution Control District, founding member of the Steering Group, American Institute of Architects Committee on the Environment; several committees of ASHRAE including Standard 62, Environmental Health, and as chairman of the Guideline Project Committee 10P, "Criteria for Achieving Acceptable Indoor Environments". From 1977 to 1985 Levin served on the California State Board of Architectural Examiners, and from 1983 to 1985 as its president. Since its founding in 1985, Levin has chaired the ASTM Subcommittee D22.05 on Indoor Air.

Name: Finn E.S. Levy
Date of birth: 1937
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Finn Levy works at the Department of Environment and Occupational Medicine, Clinic for Preventive Medicine at the Ulleval University Hospital. He graduated from the Medical Faculty at the University of Oslo in 1962 and since then has held the following positions: Internship Medical and Surgical Department and general practice 1962–1965; assistant physician at X-Ray Department 1965–1966; Medical Department (General and Cardiologic Department) Ulleval University Hospital 1966–1968, and 1970–1974 (haematology and gastroenterology); University lecturer, Internal Medicine, specially haematology and gastroenterology, for medical students 1968–1970 and 1974–1975; The Deaconness' Hospital, Oslo (gastroenterology, cardiology, haematology, geriatrics & general medicine) (1975–1979); Senior physician, National Institute of Occupational Health, Oslo, Norway (1979–1993). Head of department (1989–1993).

Dr. Levy's main tasks have included: out-patient clinic, information and research. He has worked on the following projects: solvent induced encephalopathy, occupational (allergic) pulmonary diseases, general occupational toxi-

cology, office workers occupational diseases, including problems referring to the use of "self-copying paper", "work with video display units" and other office equipment, and general indoor air quality problems. He is a widely used lecturer on occupational medicine and in particular on indoor environment in non-industrial workplaces. Since September 1993, he has been senior physician and head of the Department of Environment and Occupational Medicine — a new department at Ulleval University Hospital, Oslo, Norway. He is also: a member of the Norwegian Medical Association (since 1962); specialist in internal medicine (1972) and in occupational medicine (1992); founding member of ISIAQ (International Society of Indoor Air Quality); consultant physician for occupational medicine and IAQ matters for the Medical Board of the Norwegian Association for Asthma and Allergy (since 1984); head of the Committee for Health and Indoor Environment (HTI) of the Norwegian Association of Civil Engineers (1986–1990) and thereafter member of their Board for Professional Development Certificate on Indoor Air Quality; and a participant of the NATO/CCMS Pilot Study on Indoor Air Quality since 1989.

Dr. Levy arranged a workshop in Oslo, 1991 and co-authored the workshop report with the same name "Epidemiology and Medical Management of Building-related Complaints and Illnesses" (Finn Levy and Marco Maroni, eds.), National Institute of Occupational Health, Oslo, Norway, 1992. Coauthor of Kjell Aas & Finn Levy: "House and Health, What the Physician Knows About Indoor Environment" (in Norwegian), Cappelen, Oslo, 1992.

He was awarded the ISS-Scandinavia Indoor Climate Prize, Copenhagen 1993.

Name: James P. McLaughlin
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James McLaughlin, B.Sc., M.Sc., Ph.D., is Natural Radiation Research Group Leader at the Department of Experimental Physics, University College Dublin. His present research interests are: radiation exposure and health effects on general population due to natural radiation, with particular interest in the behaviour of radon and its health effects; and the development of techniques, based on image analysis of alpha particle tracks in CR-39, which are used in the retrospective assessment of radon exposure in the indoor environment and for alpha particle autoradiography of human tissue in leukaemia studies. His current activities include: member of the

Environmental Radiation Advisory Committee to the Radiological Protection Institute of Ireland; member and rapporteur of WHO (World Health Organization) Working Group on Radon in Indoor Air Quality; Irish representative on European Collaborative Action on Indoor Air Quality; consultant on radiation protection matters to various utilities and companies.

Name: Lars Møhlave
Date of birth: 1944
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Lars Mølhave, Master of Science (Experimental Atomic Physics and Chemistry), Ph.D. in Public Health, and D.Ms. (Doctor of Medical Sciences, Doctoral Thesis), is an associate professor at the Department of Environmental and Occupational Medicine, Aarhus University, where he is head of the Air Pollution Unit, which investigates human reaction to air pollution in occupational and non- occupational environments. The keywords of this research are: controlled experiments in climate chambers and field investigations, theoretical modelling, investigation of human subjects, (normal subjects, workers, allergic- or asthmatic persons), investigation of human reactions (objective eye, lung, neurologic or skin effects and subjective sensory effects), investigation of exposures (airborne exposure to particulates or vapours of volatile organic compounds (VOCs), thermal exposures) and technical innovations (exposure generation, source control, emission models, ventilation, building materials, exposure measurements).

Lars Mølhave has received the following research awards: National Research Foundation, Research Associateship; R.G. Nevin's Physiology and Human Environment Award for Significant Accomplishment in Man's Response to the Environment, ASHRAE (American Society of Heating, Refrigerating and Airconditioning Engineers) USA; the Environmental Protection Agency's (U.S. EPA) Scientific and Technological Achievement Awards within the category of health effects. He also has the following scientific and technical duties: member of the International Academy of Indoor Air Sciences; elected international councillor of the International Society of Exposure Analysis (ISEA); elected international trustee of the International Society of Indoor Air Quality (ISIAQ); editorial advisory committee of the journal *Indoor Air* and is research consultant to eight national and six international organizations.

His list of publications includes 62 internationally peer-reviewed research publications and journals; 11 peer reviewed chapters for international textbooks, and 151 other publications.

Name: Demetrios J. Moschandreas
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Name: Giacomo Muzi
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Dr. G. Muzi received his M.D. at the University of Perugia and followed this by postgraduate research and clinical training at the Institute of Occupational Medicine, University of Perugia. Since 1992 he has been working as an Associate Professor at the Institute of Occupational Medicine and Environmental Toxicology of the University of Perugia, Italy. His early research was on the mechanism of dioxin toxicity in animals while clinical and epidemiological studies investigated metal toxicity, with particular emphasis on lead absorption in children and *n*-hexane neurotoxicity in workers. In recent years Dr. Muzi has been involved in large-scale research

projects assessing the adverse health effects of indoor pollutants on office and hospital workers in mild climatic areas.

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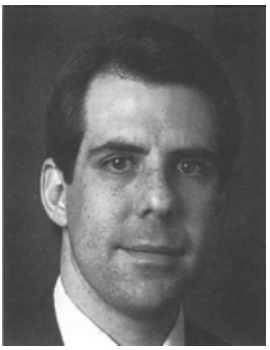
Anthony Pickering is Professor of Occupational and Environmental Medicine at the University of Manchester and Thoracic Physician at the North West Lung Centre, Wythenshawe Hospital, Manchester. He completed his medical training at Guy's Hospital, London, and later trained in the Department of Allergy and Clinical Immunology at the Brompton Hospital, London, under Professor Jack Pepys. His research activities in this department included the design and evaluation of new techniques of bronchial provocation using chemical allergens.

He was appointed as a thoracic physician in Manchester in 1976 where he has continued both his clinical and research activities. He is at present Director of the North West Lung Centre.

His main research interests have been in occupational and environmental medicine. He is currently coordinating a 10-year longitudinal study of the respiratory effects of dust exposure in cotton spinning and manmade fibre workers. Other research interests have included the epidemiology of respiratory and nonrespiratory symptoms associated with airconditioning systems and different types of humidification.



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Terrence K. Pierson is manager of Environmental Risk Analysis at Research Triangle Institute, North Carolina, USA. His research interests focus on the development of risk assessment methodologies and the application of such methodologies within the context of research, regulatory and policy-making activities. He has worked extensively on the development of risk characterization methodologies for both carcinogens and noncarcinogens with respect to exposures in the indoor environment. He has completed risk assessment for a number of consumer products emitting complex mixtures

of volatile organic compounds in the indoor environment including office equipment and new carpets. He has been involved in several NATO/CCMS Pilot Study Indoor Air Quality workshops including serving as an organizer of the workshop entitled "Sampling and Analysis of Biocontaminants and Organic in Non-industrial Indoor Environments" held in the United States in 1992. Dr. Pierson holds a B.S. in biological sciences and psychology from Ohio State University, and a Ph.D. in the environmental planning from the University of North Carolina at Chapel Hill.

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Christian Schlitt attended the Italian site of the European School (European Baccalaureate Certificate) as a German citizen and achieved his pre-doctoral degree in chemistry (laurea in chimica, direction organical-biological) training at the Università degli Studi di Pavia (Italy). For the last three years he has been employed at a local company (analysis of drug integrated fodder) and at the Occupational Health Institute in Milan (laboratory section), and has occasionally collaborated with the International Centre for Pesticide Safety, Busto Garolfo, Italy. He currently specializes in toxicology at the University of Milan, and works, as a grant-holder, at the Indoor Pollution Unit of the Environmental Institute, Ispra (Commission of the European Communities).

Contributors

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Birgitta Berglund received a Ph.D. in psychology in 1971 and in 1993 became Docent in psychology at Stockholm University, Sweden. Since 1980 her chairs there have been affiliated with the Swedish Institute of Environmental Medicine. During 1990-1994 she held a chair, donated by the Swedish Council for Building Research, as Professor of Environmental Psychology at Stockholm University with an affiliation with the Karolinska Institute. In 1994 she was appointed Professor of Perception and Psycho-

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She has published ca. 250 scientific articles in English and has organized 21 conferences among which are "Indoor Air '94" and "Healthy Buildings '88" in Stockholm. Her research areas include, among others, sensory psychology, including olfaction and psychoacoustics, psychological scaling, indoor air quality, community noise and environmentally induced sensory deficits. Her most recognized research results are predictive models for combined effects of sensory stimulations and a method for calibration of psychological scales called 'master scaling'. She has also made substantial contributions to the field of health effects of community noise and perceptual effects due to deteriorated indoor air quality.

The international engagements are numerous as reflected in work for organization as well as a stream of visiting guest researchers and guest students. Currently, she is chairman of the International Commission on Biological Effects of Noise (ICBEN), vice-president of the International Academy of Indoor Air Sciences (IAIAS) and president of the International Society of Psychophysics. She is also actively engaged in the Executive Committee of the International Association of Applied Psychology and on several occasions and in different subject areas, has served as an expert advisor for the World Health Organization and the Commission of the European Communities. She has also held, and is holding, advisory positions in boards of various research funding organizations in Sweden.

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Nadia Boschi is secretary of the International School of Indoor Air Sciences. In 1988 she received her degree in Architecture with a thesis on "Restoration and Preservation of Ancient Buildings" at the Polytechnic of Milan. In July 1994 she obtained her Ph.D. in Technology of Architecture and Environment at the same institution. In 1993 she received a fellowship from the U.S. Environmental Protection Agency, Indoor Air Division. In 1992 she was visiting scholar at the College of Architecture, Virginia Polytechnic Institute, Virginia, USA. In 1991 she was project manager for the European Community, Directorate General XVII for the Videoproduction of "Bioclimatic Architecture — A System Approach". Since 1988 she has carried out work for the Italian Government evaluating historical buildings and has

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Professor Gloria Campos Venuti obtained a degree in physics in 1960 and the habilitation in nuclear physics in 1968. From 1960 to 1978 she was member of a research group in nuclear physics of intermediate energies at the Electrosynchrotron of the Frascati National Center. In 1962, after public examinations, she joined the staff of the National Institute of Health (Istituto Superiore di Sanità, ISS) in Rome. In the period 1965–1971 she was a member of the Commission for Nuclear Safety and Health Protection of the National Commission for Nuclear Energy. From 1968 to 1983 she was professor at the Department of Science of the University of Perugia; and in 1978 and 1979 professor in advanced course in health physics at the Department of Science of the University of Rome. She became director of the Department of Radiation of the ISS in 1977 and held this post until 1980. She has twice been a member of the Scientific Committee of ISS: 1974–1980 and 1985–1988. Since 1979 she has been a member of the EC group for radiation basic standards established by article 31 of Euratom Treaty. She is research contractor of the National Institute for Nuclear Physics and director of the Radioactivity Division, Department of Physics of ISS. She has published about 300 papers in international and national scientific journals and books, on nuclear physics, radiation protection and environmental radioactivity.

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Dr. Paolo Carrer received his M.D. at the Università degli Studi of Milano. After specializing in occupational health, he undertook a masters in occupational health and industrial hygiene with a research project on "Exposure to volatile organic compounds: pollution characterization in office buildings and health risk assessment".

Since 1988 he has been a researcher at the Environmental Toxicology Unit of the Occupational Health Institute of the Università degli Studi of Milano, and since 1993 has been working as a M.D. Assistant in occupational health at Clinica del Lavoro "Luigi Devoto".

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Name: Danilo Cottica
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Dr. Danilo Cottica obtained his chemistry degree at the University of Pavia in 1977. He is currently head of the Environmental Section of the Industrial Hygiene Laboratory of the Foundation for the Institute of Occupational Health of the University of Pavia; vice-president of the Italian Industrial Hygiene Association; contract professor at the University of Pavia for Industrial Hygiene; Italian member of EEC Technical Committees; and the author of over 100 publications on industrial hygiene subjects.

Name: James H. Day
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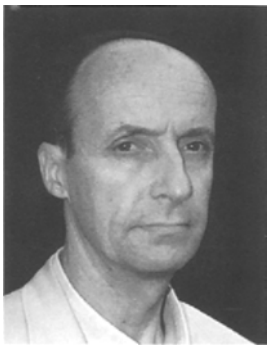
Dr. Day received his M.D. at McGill University in 1959 followed by training at the Royal Victoria Hospital in Montreal. He later undertook research in allergy and immunology at the Scripps Clinic and Research Foundation, La Jolla, California and the Wright Fleming Institute in London, England. He returned to Queen's University in 1967 where he combines clinical practice, teaching and research in asthma and rhinitis including controlled antigen challenge as a means of assessing medication for allergic respiratory disease, hymenoptera sensitivity and health consequences of indoor air quality problems.

Dr. Day is past president of the Canadian Society of Allergy and Immunology and served as chairman of the Public Health Committee Ontario Medical Association, a member of the Science Advisory Board of the International Joint Commission of the Great Lakes and chaired the IJC Task Force on the health effect of non-phosphate detergent builders. He serves as a consultant to the World Health Organization/NATO on indoor air concerns.

He is currently professor of medicine and head of the Division of Allergy and Clinical Immunology at Queen's University, Kingston, Ontario.



Name: Maurizio De Bortoli
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Country: Italy

Dr. M. De Bortoli obtained his licence in Chemistry in 1961. He subsequently (1961–1972) worked on the radiation protection of the population at the Joint Research Centre of EURATOM, the European Atomic Energy Commission, in Ispra, Italy. From 1973 to 1980 he worked on the chemical pollution of the atmosphere (mainly by the use of tracers), in the same place, under the Commission of the European Communities.

Since 1981 he has been working at the Environment Institute of the European Commission (Ispra) in the indoor air pollution research (surveys in homes and offices, emission/adsorption by materials in environmental test chambers) and in the co-ordination of the European Collaborative Action "Indoor Air Quality and Its Impact on Man".

Name: Gaute Flatheim
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Having obtained his M.Sc. degree in Mechanical Engineering from NTH, Technical University of Norway in 1963, Gaute Flatheim started the consulting engineering company Siv.ing. Gaute Flatheim A/S in January 1965, and later Flatheim A/S in 1989. He qualifies as an expert in Indoor Environment Consultancy, working with schools, kindergartens and office buildings. Gaute Flatheim is EURO-engineer and a lecturer in HVAC and environmental subjects in Norway and all over the world. He is involved in an ongoing research project "Indoor Environment in Schools" and in developing the Norwegian concept for "Next Century's Office Buildings". He is a member of a wide range of committees dealing with HVAC and Indoor Air Quality. Flatheim A/S is specializing in quality plans for building owners and planners.

Name: Silvana Piermattei
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Name: Olli A. Seppänen
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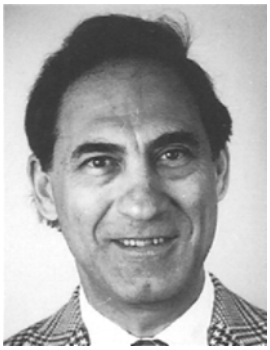
Country: Finland

Olli A. Seppänen has been Professor of Heating, Ventilating and Air Conditioning at Helsinki University of Technology since 1982. He worked as a manager of building services, Ekono INC, Seattle (USA) from 1980 to 1982 and later as a manager of building research, Ekono Consulting Engineers, from 1975 to 1980. He has been President of the 6th International Conference on Indoor Air Quality and Climate (1993) and is President of the International Society of Indoor Air Quality and Climate from 1994. His publications are: one book, approximately 50 papers and 100 articles.



Name: Michael J. Suess
Date of birth: 1933

Dr. Michael J. Suess completed academic studies in Israel and the USA in civil, hydraulic, sanitary and nuclear engineering and radiation sciences, and obtained a doctoral degree in environmental health from the Graduate School of Public Health, University of Pittsburgh. He is a licensed engineer in Israel and the USA, a Diplomate of the American Academy of Environmental Engineers, an invited life member of the International Academy of Indoor Air Sciences, and a founding member of the International Society for Indoor Air Quality and Climate.

Dr. Suess joined the World Health Organization Regional Office for Europe in 1968. As Regional Adviser for Recognition and Control of Environmental Health Hazards, he has managed inter-country projects in the fields of water, air, waste, new technologies, radiation and energy, and has organized over 90 scientific meetings and developed numerous manuals and reference handbooks. In 1975 he initiated the WHO international programme on indoor air quality and conducted ten related working groups, resulting with some major and ground-breaking reports. Dr. Suess has published over 100 scientific peer-reviewed papers and reports, and edited several reference books on water examination, air quality protection, waste management, and radiation protection. He retired from WHO in 1993 and now presides over his own International Consultancy for Environmental Health Hazards and Engineering.

Name: Douglas Walkinshaw
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Country: Canada

Indoor Air Technologies (IAT) conducts indoor air quality investigations and manages indoor air quality-related design, construction, retrofit and commissioning. It also provides the secretariat for the International Society of Indoor Air Quality and Climate (ISIAQ). The principal of the firm, Douglas S. Walkinshaw Ph.D., P.Eng., is well known in the field having served, *inter alia*, as: Coordinator of Indoor Air Quality at the National Research Council (1982–1986) and at Health and Welfare Canada (1986–1988); President of the 5th International Conference on Indoor Air Quality and Climate — Indoor Air '90, Toronto, 1990; member of the American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE) Ventilation Standard Committee (1990–1994); and member of the Canadian General Standards Board Combustion Venting Requirements Committee CGSB-51.71 (1993–1994). Dr. Walkinshaw is the inventor of the ventilated cavity ECHO system for energy efficient prevention of contaminant migration and moisture problems.

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Having obtained his Ph.D. in Biochemistry from the University of Tübingen, Germany, in 1978, Maged Younes joined the Institute of Toxicology, Medical University of Lübeck, Germany, where he remained until 1989. During that period, he conducted research mainly in the field of biochemical toxicology. Dr. Younes qualified as an Expert in Toxicology (Fachtoxikologe) by the German Society of Pharmacology and Toxicology, and was nominated Professor of Toxicology and Biochemical Pharmacology. In 1991 he was nominated head of the Unit on Biochemical Toxicology at the Max von Pettenköfer-Institute of the German Federal Health Office in Berlin. Since November 1991, he has been the manager responsible for the European Chemical Safety Programme of the World Health Organization at the Bilthoven Division of the WHO European Centre for Environment and Health.

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Introduction

1. THE IMPORTANCE OF INDOOR AIR QUALITY

The quality of air inside enclosed spaces has become a matter of growing concern over the last twenty years. This concern was initially triggered by reports of occupants of various indoor environments who complained about a variety of unspecific symptoms, such as irritation or dryness of mucous membranes, burning eyes, headache or fatigue. Because in some cases these symptoms could be related to elevated concentrations of specific pollutants in indoor air, such as formaldehyde, increasing attention was devoted to determining climate conditions and chemical compounds in the air of rooms whenever people complained about bad indoor air quality.

It rapidly became clear, however, that acute reactions to specific pollutants were only one of the reasons for becoming concerned about indoor air pollution. The other was more general and related to the fact that estimates of population exposure to air pollutants had been based exclusively on data from outside air monitoring; thus the quality, duration, and effects of human exposure to air pollutants indoors were likely to have been overlooked and undervalued.

To correctly characterize the quality of exposure to air pollutants, emissions from a variety of sources, including building materials, appliances of various types and consumer products, must be considered. As to the duration of exposure, the time that the population of industrialized countries spends both outdoors and indoors would have to be taken into account.

2. INDOOR AIR QUALITY: AN INTERNATIONAL MULTIDISCIPLINARY EFFORT

In recent years, our knowledge in the field of indoor air quality has been increasing steadily. International conferences, especially the International Conferences on Indoor Air Quality and Climate and comprehensive reports prepared by international and national working groups as well as a number of monographs, have contributed to the compilation of the knowledge available in this field.

This book is the result of a large international effort that has collected part of the production of the NATO Committee for Challenges of Modern Society — Pilot Study on Indoor Air Quality, the European Collaborative Action "Indoor Air Quality and its Impact on Man" (formerly COST 613), the International Society of Indoor Air Quality and Climate (ISIAQ), the World Health Organization, and the U.S. Environmental Protection Agency — Indoor Air Division.

Most of the authors who have contributed, directly or indirectly, to the papers collected in this book belong to the International Academy of Indoor Air Sciences.

The reader will note that the knowledge developed over the last two decades has changed the perception of the importance of indoor air, which is now considered to be one of the major areas of intervention in health-related research and in public health applications.

However, despite these efforts and achievements, much remains to be done in both research and practice.

Up to now, large-scale surveys permitting broader conclusions than case study-type investigations have been scarce and studies directed towards uncovering relationships between indoor air pollution and potential negative health effects are still limited for the majority of pollutants. This is in part due to a lack of financial resources, but is also caused by the complexity of the subject itself. Indoor air research being a multidisciplinary task, a highly skilled team including scientists from various disciplines such as medicine, chemistry, biology, engineering, architecture and building sciences, is generally required.

To deal with special problems, e.g., those related to air-conditioned office buildings ("sick building syndrome"), it may even become necessary to include professionals generally not involved in air pollution studies, such as psychologists and behavioural scientists.

3. HEALTH RISK ASSESSMENT

As was mentioned above, the indoor environment plays a highly important role in the assessment of the risk associated with exposure to pollutants.

Hazard identification, dose–response assessment, exposure assessment and risk characterization are the four elements of risk assessment.

While the concepts of hazard identification and dose–response assessment include the use of biological data, such as absorption of pollutants into the body, pharmacokinetics, bioassays and results from animal exposure experiments, exposure assessment has to consider: (a) the sources of various pollutants, (b) the concentrations to which the emissions of these sources lead, and (c) the exposed population (in terms of both quality and quantity). Although progress has been made with regard to these three topics, they are likely to

continue to be major items on the agenda of indoor air quality research in the years to come.

Even in the case of pollutants that have been dealt with in outdoor air studies for many years, such as nitrogen dioxide and suspended particulate matter, our knowledge of frequency distributions of indoor pollutant concentrations is generally not sufficient to permit a comprehensive exposure assessment for the various population groups. However, a sufficiently developed knowledge of such distributions is essential to a sound exposure assessment.

4. NATURE AND SOURCES OF POLLUTANTS OF INDOOR AIR

Occupants and their activities tend to introduce contaminants into the air of a building. Additionally other contaminants often emanate from building materials and contents or penetrate with outdoor air. Elevated contaminant concentrations are usually reduced in a building by natural or forced ventilation, whereby outside air displaces some of the contaminated air.

Clearly, a high ventilation rate is advantageous in reducing indoor air pollution to a low level. There are, however, a number of factors which can interfere with this simple solution, and make it necessary to consider compromises and alternative indoor contaminant reduction strategies. The most important of these factors are the energy cost of ventilation in terms of heating, cooling or dehumidification, and intentional limitation of ventilation in some locations under certain conditions when the air outdoors may be more polluted than that indoors. In recent years, many countries have taken measures to reduce ventilation rates to conserve energy, but this practice is now under dispute for health economic reasons. Also, over the last several decades, knowledge of the outdoor air pollution sources, the related adverse health effects, and the methods required to improve ambient air quality has greatly advanced. Many countries now have standards or guidelines for ambient air pollutants, the concentration of which generally show a downward trend.

Hence, the impact of outdoor air pollution on the indoor air quality has often been reduced.

The concentration of an air pollutant indoors depends on a number of factors:

1. volume of air contained in the indoor space;
2. rate of production, or release of the pollutant into the indoor space;
3. rate of elimination of the pollutant from the indoor space through reaction, filtration or settling;
4. rate of air exchanged with the outside atmosphere, through infiltration, natural or forced ventilation;
5. concentration of the pollutant outdoors.

In the indoor environment, because of the small mixing volume, even the release of relatively small amounts of a pollutant can produce a high concentration which many affect human health and well-being.

Since in almost all inhabited spaces there is a continuous air exchange with the exterior, all pollutants present in the outdoor air are also found indoors. Important pollutants in this category are: suspended particulate matter, sulphur oxides, nitrogen oxides, carbon monoxide, hydrocarbons, photochemical oxidants and heavy metals. Measurements of these pollutants have been made simultaneously indoors and outdoors by a number of investigators, finding that in many cases — with the exception of several organic compounds — concentrations indoors were similar to those outdoors, with the average ratio of indoor/outdoor concentration falling within the range of 0.5 to 2.

Ultimately, all internally-generated air pollutants are the result of human action or choice. It is useful to divide such pollutants into two categories. In the first category, the pollutants are released only in connection with human activity or even presence, while in the second, they are released from building materials, and furnishings, generally over long periods of time. The number of compounds related to human activity or presence is limited only by the inclination of human occupants to bring them indoors. Thus, compounds which occur in the industrial workplace can be introduced into the nonindustrial environments. Furthermore, products enter the residential environment in connection with hobby or craft activities. In many cases these are carried out without the protective measures required by regulations governing the industrial workplace.

The concentration of indoor pollutants varies in time and in the case of activity-related compounds accumulated exposures depend on the intensity and duration of the activity with which the release is associated.

Smoking is a major cause of indoor air pollution. While smokers are exposed to mainstream smoke, nonsmokers in a space are exposed to increased concentrations of tobacco smoke constituents, especially respirable particulate matter, carbon monoxide, and a number of organic substances known or suspected to be carcinogens.

In addition to smoking, other combustion processes like those related to heating and cooking are at the origin of a large number of indoor air pollutants.

Human presence and activity bring further changes in the indoor atmosphere: metabolic activity reduces the concentration of oxygen and increases that of CO_2, and produces a variety of odours. Respiration, sweating and several other processes increase the amount of water vapour in indoor air. If the ventilation rate is low, concentrations of CO_2 and water vapour may rise above the recommended levels.

A large number of organic pollutants are released from building materials

and products, regardless of human presence or activity. One example is formaldehyde which is released from, e.g., particleboard, medium density fibre board and plywood.

Formaldehyde contained in these products can be released over long periods, depending on temperature and moisture regimes. Another example is asbestos which has been used in the past in a variety of building products for its special properties. Spray materials containing 10–30% asbestos have been used for fireproofing, and for decorative and acoustic purposes, and elevated airborne asbestos fibre concentrations have been found indoors. The use of asbestos is now forbidden in many countries.

The ground on which the building is built may contain volatile pollutants entering into the building via fissures and other openings in the building shell. A well-known example is radon originating from the ground either due to natural geological conditions or to earlier mining activities in the area.

5. PRIMARY CAUSES OF HEALTH PROBLEMS INDOORS

Despite the fact that indoor environments vary from one country (or even region) to the other, due to differences in construction habits, climatic conditions and life-style, the industrialised societies tend to face a growing number of similar indoor air quality problems.

Indoor environmental problems have five common causes, and more than one may be active at any time: an inadequately cleaned or maintained environment, insufficient ventilation, pollutants emitted from sources and activities inside the building, contamination from outside sources, and biological contamination due to a lack of moisture control. These causes can often intensify or add to the stress that occupants suffer from inadequate temperature, humidity, or lighting, or by excess noise. Exposure to pollution indoors also adds to stress of occupant density, job dissatisfaction, lack of personal privacy and control over the environment.

The inadequately maintained environment is the consequence of inattention to the different emissions and by-products of activities indoors and the need for constant ordering.

Poor ventilation sometimes is the result of dirty air filters that need periodic cleaning or replacement. Emissions from cooking or tobacco products always need to be removed. Left alone, they accumulate damaging materials, causing odours, and in some cases increasing cancer risks to humans.

Particles from the outside are constantly being tracked or blown inside. They need to be removed through cleaning and maintenance.

Before the 1974 energy crisis, most buildings were designed to provide maximum comfort to inhabitants. Heating, ventilating, and air conditioning

systems were designed to provide as much as 15 cfm of outside air for each building occupant. Since 1974, buildings have been designed to save energy. The size of spaces for heating and cooling has been reduced, and outdoor air ventilation lowered to 5 cfm. Moreover, many ventilation systems do not effectively distribute air to people in buildings. Inadequate air diffusion combined with reduced ventilation causes pollution levels to grow and, as pollutant concentrations rise, so do health complaints.

As already discussed, many indoor pollutants come from sources inside the building, while others may enter the building with outdoor air or from the soil.

A major cause of illness indoors is biological contamination. It is — or is derived — from living organisms such as bacteria, fungi, viruses, and mites, and from other biological material such as insect parts, that can originate both inside and outside of the built environment.

Biological contamination occurs most often when moisture and food sources for living organisms are not properly managed. Ironically, mechanical ventilation systems are often sources of these contaminants. Biological pollutants such as fungi and bacteria breed in water that has been allowed to accumulate on hard surfaces, for example in humidifiers and cooling coil condensate pans.

They also breed where water has collected on or under cellulose materials such as ceiling tiles, wallpaper, carpeting, insulation, and internally lined duct work. The most publicized of these biopollutants is *Legionnella*, that may cause a fatal disease. In addition to the risk of infections, humans can be sensitized to several different fungi such as *Aspergillus* and *Penicillium*, and experience allergic reactions and a range of health effects involving skin, eye, and the upper and lower airways.

6. INDOOR ENVIRONMENTAL MANAGEMENT STRATEGIES

It is interesting to note that in the same way as the man is responsible for the creation of indoor air problems, the man can also prevent them to a great extent adopting a proper environmental management strategy.

In general, environmental management is the process aimed at keeping harmful substances away from humans or down to levels that will not cause harm. The primary goal of environmental management indoors as well as outside is to reduce exposures of people and materials to harmful substances.

We should be optimistic that we can improve or maintain the quality of the indoor environment as there are many methods to keep pollution to safe levels indoors. These approaches to pollution control are so fundamental and time-tested that they can be recognised as basic principles of managing pollution. They are:

- source management which includes source removal or source modification,
- activity management,
- design intervention and remedial actions,
- dilution, and
- cleaning.

Pollutants are most effectively controlled at their source. Once a pollutant is in the environment, it is more difficult to track down and return to its proper place. Much more energy is expended in controlling pollutants that have spread away from their sources than is spent keeping them there. Therefore, it is important to investigate all options for controlling pollution at its source. For example, some sources could be removed or displaced, others could be altered so that they will stop polluting or emitting a specific pollutant. Or the pollutant could be captured at the source so it cannot diffuse into the environment.

Source removal

The ideal, permanent way to eliminate pollution is to eliminate its source. Unfortunately, almost everything in the environment is a source of chemical emission and obviously, we cannot remove all sources: it may be impractical or too expensive. As an environmental management option, source removal should be considered particularly when the pollutants from the source cause serious harm or damage to the environment or to humans.

Source modification is a powerful option in environmental management. Almost always, a polluting source can be redesigned to pollute less: laundry detergents containing phosphates can be replaced with non-phosphate detergents; carpet-cleaning chemicals containing trichloroethylene can be replaced with less hazardous solvents, etc.

One way to control a polluting source is adding a mechanical device to it that will capture the pollutant before it is emitted to the environment. Source control is a "technological fix" designed to manage emissions before they leave the source. In this case the source itself is not removed from the environment or modified.

Source control is an effective means of managing pollution so long as the control technology does not break down. When it fails, pollution levels in the environment will rise. Moreover, the expense of this technology will vary depending on what is being fixed.

Other techniques for keeping pollution out of the built environment, are activity management, design intervention, and remedial action. In some cases, these approaches are refinements of source removal and source modification. However, when we think of these techniques, we should think of them in terms of:

- how the built environment is designed,

- what activities we allow to go on in the built environment, and
- what we can do to keep down pollutant levels in the built environment when we find a problem in it.

Activity management

Activity management is the process of ensuring that a building is used for the activities it was built to accommodate. Every built environment is designed for a specific human activity. Homes, multi-storey office buildings, laboratories, manufacturing plants, schools, hospitals, churches, warehouses, and barns are designed for specific and different activities. The activities that go on in each of these special environments generate different pollutants and pollutant concentrations. In most cases, built environments are designed to deal with their usual activities and resulting by-products. However, if we perform an activity in an environment not designed to handle it, we can create pollution levels harmful to health.

For example, using large amounts of chemicals in a laboratory may be fully acceptable. The laboratory will probably have adequate ventilation and disposal systems for them. Using the same chemicals in a home, however, may present serious hazards and risks. Similarly, office space is safe for some activities and not for others. It is usually designed for people, desks, paper storage, and phones. If we start manufacturing things in an office, cooking and eating at our desks, or using products that emit excessive amounts of chemicals, we can create a state of pollution that the building was never designed to manage.

Contaminated buildings are often being used for purposes other than those they were originally constructed for. The best way to manage this kind of pollution problem is to control activities within the building.

Design intervention

Design intervention is meant to match the built environment with the activity inside in order to minimize human exposure to pollutants.

Design intervention might include special ventilation systems that carry away harmful by-products. It might also consist of the use of special building materials that do not emit pollutants, such as wood products that do not release formaldehyde, or paints that do not emit large amounts of hydrocarbons. Design intervention also includes building a structure that can be easily cleaned: hard, flat surfaces are easier to clean than textured or fleecy surfaces and thus more economical. Cleanable surfaces are a must, for example, in hospitals and schools especially if the institution will have limited funds for regular and thorough cleaning. Design intervention is very important when designing a new building or when remodelling an old structure for a new use.

Remedial action

Even after a new building has been designed with a keen eye to reducing pollution levels indoors, something often has been miscalculated or overlooked. This is where remedial action finds indication. Remedial action may be as simple as eliminating door mats, or it may involve increasing the air flow in the ventilation system, installing dehumidifiers, or increasing the frequency of cleaning activities. Remedial action may also include altering activities or redesigning part of the built environment.

Once pollution has spread away from its source and into the environment, three options for dealing with it are available: one can live with it, dilute it, or clean it up.

Dilution makes pollutants less concentrated and the less concentrated they are, the less toxic or harmful they become. Pollutants can be diluted either by removing some of them or by re-distributing them out over a larger surface or space.

Using dilution as an option for environmental management always entails risks, because some harmful pollutants may remain at toxic concentrations and harmful pollutants successfully removed from the immediate environment, may continue to cause problems in another environment.

At best, dilution is a way of coping with pollution by keeping it at less harmful concentrations, nevertheless, the optimum place to manage pollution is at its source and source management and removal should receive priority over dilution.

Cleaning is the process of removing pollutants from the environment and putting them in their proper place of disposal. Cleaning occurs after pollutants have entered the environment: the steps of the process are find, identify, capture, contain, remove, and dispose of pollutants. Cleaning is different from diluting, as cleaning means removing. We do not hide or brush aside dusts and wastes and say that we are cleaning. We must remove and dispose of them, too.

7. CONCLUSIONS

We are only beginning to understand now how important indoor environmental quality is to health and well-being. It is clear that the indoor environment needs to be managed as seriously as the outdoor. If this does not take place, the result will be human dissatisfaction, discomfort, illness, social problems and, consequently, reduced productivity.

It has to be remembered that it is easier and less costly to design a healthy building and keep it healthy through effective operation and maintenance than it is to assess, correct, and pay for human suffering in an environment that has deteriorated.

The community should be motivated to properly manage the indoor environment also because unhealthy buildings cost money: it costs money to operate them inefficiently, and it costs money to restore them to healthy environments. But the human costs are even higher: absenteeism increases, and work productivity drops in unhealthy environments; work satisfaction dwindles; people work more slowly when disturbed or annoyed by noise, odours, or other ill-managed features of their workplace; much time is lost in addressing complaints; still more time is spent trying to identify and correct problems.

Fortunately, the indoor environment can be managed, although this requires professional investments at every level. In contrast to the natural environment, the man-made environment can be properly designed, operated, and maintained to meet the needs of its inhabitants. As the society begins to better understand the influence that natural and man-made environments have on human health, the society recognizes a need to better understand our options for managing the indoor environment and a need to better understand procedures to be used to enhance the quality of life indoors.

PART I
Nature, Sources and Toxicity of Pollutants of Indoor Air

The main pollutants of indoor air will be presented and discussed in the following chapters of Part I. For each pollutant there will be a description of the physico-chemical nature, the occurrence and sources, the typical concentrations found indoors and the resulting human exposure. Finally the most important toxicological properties will be discussed, together with the mechanisms by which these compounds may interfere with human tissues.

Chapters 1, 2, 3 and 4 have been prepared by H. Knöppel and C. Schlitt, with contributions from D. Cottica and D. Cavallo. Section 3.4 has been prepared by F. Bochicchio and J. McLaughlin, with contributions from G. Campos Venuti and S. Piermattei. Chapter 5 has been prepared by B. Flannigan.

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Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 1

Inorganic Pollutants[1]

1.1 CARBON DIOXIDE

1.1.1 Physico-chemical nature

Carbon dioxide (CO_2) is a colourless, odourless gas. It is a simple asphyxiant, but it can also act as a respiratory stimulant. [Conversion factors: 1 ppm[2] = 1.80 mg/m^3; 1 mg/m^3 = 0.56 at 25°C and 101.3 kPa (1 atm)].

1.1.2 Occurrence and sources

Man continuously emits amounts of carbon dioxide and water vapour solely from his breath. The significance of carbon dioxide as the leading human exhalation product has already been recognized by Pettenkofer who, considering the hygienic aspects, suggested 0.1% of CO_2 as a limit value for an adequately ventilated room (700 ppm in bedrooms) (Pettenkofer, 1858).

Carbon dioxide represents the main combustion product when considering domestic energy use for cooking and heating purposes. Gas, kerosene or wood-fuelled appliances can generally be considered as the main sources of CO_2, depending on how combustion exhausts find their way into indoor atmosphere.

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1 A part of the text of this chapter has been derived from World Health Organization (WHO), 1987 "Air Quality Guidelines for Europe", WHO Regional Publications, European Series No. 23, WHO Regional Office for Europe, Copenhagen; U.S. Environmental Protection Agency (EPA), 1987 "Preliminary Indoor Air Pollution Information Assessment", Appendix A, EPA-600/8-87/014, pp. 2–18, 19, Office of Health and Environmental Assessment Washington DC; U.S. Environmental Protection Agency (EPA), 1991 "Introduction to indoor air quality", EPA/400/3-91/003; ECA (European Concerted Action "Indoor Air Quality and its Impact on Man", COST project 613), 1989, Indoor Pollution by NO_2 in European Countries, Report No. 3 (EUR 12219 EN), Luxembourg: Office for Publications of the European Communities, p. 8, 1989; Yocom J.E. and McCarthy S.M. (1991) Measuring Indoor Air Quality. John Wiley & Sons, ISBN 0-471-90728-6.

2 All adimensional concentration units in this book refer to V/V units, unless otherwise indicated.

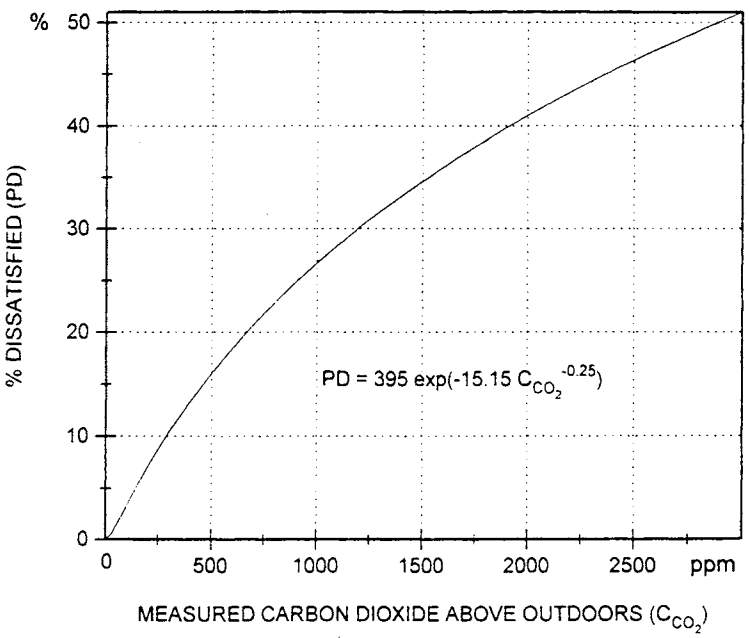

Fig. 1.1: Carbon dioxide as an indicator of human bioeffluents. The curve shows the perceived air quality (% dissatisfied) as a function of the carbon dioxide concentration (COST, 1992).

1.1.3 Typical concentrations and exposures

In homes without CO_2 sources, concentrations might be in the range of 0.07–0.20% (NRC, 1981). Concentrations in homes with kerosene heaters can reach concentrations above 0.3% within several hours, and concentrations greater than 0.5% have been measured (Ritchie and Oatman, 1983). Concentrations of 0.177% to 0.815% were measured by Traynor et al. (1983) in a test house heated by unvented gas heaters. The mean value for the indoor/outdoor ratio is about 1–3.

As an indicator of human bioeffluents, CO_2 has been applied quite successfully for more than a century (Huber and Wanner, 1983). Figure 1.1 shows the percentage of dissatisfied visitors as a function of the CO_2 concentration (above outdoors) for spaces where sedentary occupants are the exclusive pollution sources.

Although CO_2 is a good indicator of pollution caused by human beings, it is often a poor indicator of perceived air quality. It does not acknowledge the perceived pollution from sources not producing CO_2 (Berglund and Lindvall, 1979) and certainly not the non-perceivable hazardous air pollutants such as carbon monoxide, radon, benzene, etc.

1.1.4 Health effects

At concentrations above 1.5% respiration is affected and breathing becomes faster and more difficult. Concentrations above 3% can cause headaches, dizziness and nausea. Concentrations above 6–8% can result in stupor and death (NRC, 1981).

The lowest level at which effects have been observed in both human and animal studies is about 1% (U.S. CPSC, 1983). Structural changes in the lungs of guinea pigs have been observed along with calcification of the kidneys. In humans, effects include increase in respiration, changes in blood pH and pCO_2, and decreased ability to perform strenuous exercise. The significance of these effects is not clear, but a potential increase in respiratory and gastrointestinal illness has been postulated because these effects were observed in submarine crews at concentrations of 0.5–1% (NRC,1981). Table 1.1 summarizes the results of some studies conducted on exposure to CO_2.

1.2 CARBON MONOXIDE

1.2.1 Physico-chemical nature

Carbon monoxide (CO) is a colourless, odourless, and tasteless gas. It is a product of the incomplete combustion of carbon-containing materials. In the blood it reacts with haemoglobin to form carboxyhaemoglobin (COHb). [Conversion factors: 1 ppm = 1.15 mg/m^3; 1 mg/m^3 = 0.87 ppm at 25°C and 101.3 kPa (1 atm)].

1.2.2 Occurrence and sources

Carbon monoxide is one of the most common and widely distributed air pollutants. It is widely generated indoors by unvented combustion appliances, particularly if they are operated in poorly ventilated rooms. Tobacco smoking is also an important source of indoor CO pollution.

1.2.3 Typical concentrations and exposures

Without sources, the concentrations of carbon monoxide in indoor environments are, at best, as high as those of outdoor air. The results obtained by Ott and Flachsbart (1982), who in 1979/1980 determined CO in offices in California where nearly 97% of the values were lower than 10 mg/m^3, could mark an upper limit in comparison with regions of lower outdoor pollution.

TABLE 1.1

Selected studies on the effects of human exposure to carbon dioxide

Exposure concentration and duration	Exposure method and subjects	Effects	Ref.
4%, 2 weeks exposure bracketed by two 2-week control periods	Chamber, 24 subjects	No psychomotor impairment; no decrement in complex task performance by healthy young subjects	Storm and Giannetta (1974)
4.2%, 5 days and 11 days; 3%, 30 days; exposures bracketed by two 3–5 day control periods	Chamber, 12 subjects; total 4 in each of 3 groups	Increased arterial and cerebrospinal fluid bicarbonate; decreased pH; occasional mild headaches and awareness of increased ventilation during first 24 h of exposure; some ectopic foci (cardiac arrythmia) noted during excercise but small sample size hampered interpretation; decreased tolerance to exercise noted	Sinclair et al. (1969)
3%, 5 days bracketed by two 5-day control periods	Space Cabin Simulator; 7 subjects	No changes in ammonia or titrable acidity; no changes in serum electrolytes, blood sugar, serum creatinine, or liver function; no significant changes in excercise or psychomotor studies	Glatte et al. (1967)
1.5%, 42 days 0.7–5%, 50–60 days	Chamber*; Submarines (13 Polaris patrols)	Increases in respiratory minute volume, tidal volume, physiological death space; decrease in vital capacity; respiratory acidosis, increase in pCO_2, decrease in pH; decrease of plasma chloride, red cell sodium increase, potassium decrease; decrease in plasma calcium metabolism, urine calcium, urine magnesium, increase in red cell calcium. In the submarine study a decrease in respiratory and gastrointestinal disease was noted with decreasing CO_2 (and other pollutants)	Schäfer (1979)
1% and 2%, 30 days	Chamber, 2 subjects in each of 2 exposures	At 2% significant increases in pCO_2 in blood and alveolar air, decrease in ability to perform strenuous exercise; decrease in blood pH, increase in pulmonary ventilation; changes at 1% were not considered to be significant; authors conclude that prolonged CO_2 exposure causes acidosis, hypodynamia, and fatigue but effects are reversible	Zharov et al. (1963)

*Similar effects were noted in subjects in both the chamber exposure and submarine exposure.

Measurements in 250 Dutch apartments with unvented hot water appliances (geysers) showed a CO concentration higher than 11.5 mg/m^3 in 1/3 of all cases (Brunekreef et al., 1982). Continuous monitoring of the CO concentration in kitchen air during the use of a gas stove resulted in short-term maximum values up to 17 mg/m^3 (Seifert et al., 1984).

The measurements in smoking zones gave different CO concentrations (depending on the extreme conditions). The upper limit of what may generally be expected in bar and pubs could be taken from Cuddeback et al. (1976) or Sebben et al. (1977) who measured average values of 11.5–23 mg/m^3 with top values of up to 34.5 mg/m^3.

A review of indoor by-product levels of tobacco smoke (Sterling et al., 1981) reported that the difference in CO values between smokers' and non smokers' sections of cafeterias ranged from 0.8 mg/m^3 to about 5 mg/m^3, while the difference between indoor and outdoor values ranged from 0 to about 10 mg/m^3. Tobacco smoking usually makes the largest contribution to the CO body-burden in smokers.

TABLE 1.2

Indoor microenvironments listed in descending order of weighed mean CO concentrations (Akland et al., 1985)

Indoor microenvironment	No. of subjects	CO concentration (ppm)	
		Mean	Standard deviation
Public garage	116	13.46	18.14
Service station or motor vehicle repair facility	125	9.17	9.33
Other location	427	7.40	17.97
Other repair shop	55	5.64	7.67
Shopping mall	58	4.90	6.50
Residential garage	66	4.35	7.06
Restaurant	524	3.71	4.35
Office	2287	3.59	4.18
Auditorium, sports arena, concert hall, etc.	100	3.37	4.76
Store	734	3.23	5.56
Health care facility	351	2.22	4.25
Other public buildings	115	2.15	3.26
Manufacturing facility	42	2.04	2.55
Residence	21543	2.04	4.06
School	426	1.64	2.76
Church	179	1.56	3.35

TABLE 1.3

Distribution of indoor residential CO concentrations for Denver, Colorado, winter 1982–83, 229 observations unweighted

Percentile (%)	CO concentration (ppm)
10	0.5
20	0.8
30	1.0
40	1.3
50	1.8
60	2.3
70	3.0
80	4.2
90	6.2
95	8.5
99	14.5
Maximum	26.7
Mean	2.76
Standard deviation	2.92

Human exposure to CO in the residential indoor environment has been investigated in studies in which subjects carried personal monitors while they were indoors at home, or at work in a public building (Akland et al., 1985). The monitors recorded average CO exposure during intervals of up to one hour. Table 1.2 shows the mean and standard deviations of CO concentrations in the various indoor environments that were studied. Table 1.3 gives the CO distribution found in the air of residences. The results indicate that people may be exposed to CO levels that approach the US National Ambient Air Quality Standard (NAAQS) for the 8-hour and 1-hour averaging times. The 8-hour NAAQS concentration for CO of 10.3 $\mu g/m^3$ was exceeded in 4.3% of the homes. However, these data were collected during the winter and do not represent an annual average.

The mean value for indoor/outdoor ratios is 1 in the absence and 1–5 in the presence of CO sources.

1.2.4 Health effects

Since CO is a gas and does not penetrate the skin, the only route of exposure is via inhalation. Like oxygen, CO is able to combine with haemoglobin

forming carboxyhaemoglobin (COHb). However, CO is about 200 times as effective as oxygen in combining with haemoglobin. This means that when both O_2 and CO are present, haemoglobin will not be available. The transport of oxygen to the tissues may be dramatically reduced, depending on the CO concentration. Once inside the body, CO has a half-life of about 5 hours.

The health effects of CO exposure are generally discussed in terms of the percent COHb in the blood. The level of COHb is directly related to the CO concentration in the air, the duration of the exposure, and the activity level of the individual.

Normally, metabolic processes in the body will result in a COHb level of 0.5–1.0%. Average COHb levels among nonsmokers are 1.2–1.5%. In cigarette smokers the level is about 3–4% on average, but it may be as high as 10% in heavy smokers (WHO, 1987).

Continuous exposure to 30 ppm CO leads to an equilibrium COHb level of 5%; about 80% of this value is reached within 4 hours and the remaining 20% over the next 8 hours. Continuous exposure to 20 ppm CO leads to COHb levels of 3.7% and exposure to 10 ppm leads to COHb levels of 2%. The time for equilibrium to be established is usually 8 hours, but this time can be shorter if a person is physically active (Doull et al., 1980).

CO can have detrimental effects on the heart and lungs, and on the nervous system. At COHb levels of 10%, the major effects are cardiovascular and neurobehavioural. Levels of 2.5% have been shown to aggravate symptoms in angina pectoris patients. No adverse health effects have been reported below 2.0% COHb. In the range of 2 to 3% findings with regard to health effects in the normal population are inconclusive (WHO, 1987).

1.3 NITROGEN DIOXIDE

1.3.1 Physico-chemical nature

There are a number of different nitrogen oxides (NO_x), among which nitrogen dioxide (NO_2) is the most widely considered in indoor air pollution studies. NO_2 is a water soluble red to brown gas with pungent, acrid odour. It is produced during combustion at high temperatures from the combination of nitrogen and oxygen from air. It is an oxidizing agent that is highly irritating to mucous membranes, and causes a wide variety of health effects.

Oxidation of nitric oxide (NO) by atmospheric oxidants such as ozone occurs rapidly, even at low levels of reactants present. The reaction is regarded as the most important route for nitrogen dioxide production in the atmosphere. [Conversion factors: 1 ppm = 1.88 mg/m^3; 1 mg/m^3 = 0.53 ppm at 25°C and 101.3 kPa (1 atm)].

TABLE 1.4

Range of the maximum 1-minute, 1-hour and 24-hour average NO_2 concentration and of the mean NO_2 concentration ($\mu g/m^3$) in twelve Dutch homes (taken from COST Project 613 no. 3, 1989; modified from Lebret et al., 1987)

Location	Maximum concentrations ($\mu g/m^3$)			
	1-min average	1-h average	24-h average	Overall mean concentration
Kitchen	400–3808	230–2055	53–478	36–227
Living room	195–1007	101–879	49–259	32–142
Bedroom	57–806	48–718	22–100	16–104
Outdoors				25–70

1.3.2 Occurrence and sources

Generally NO_2 is emitted from indoor combustion sources. These comprise tobacco smoke, gas appliances kerosene heaters, woodburning stoves and fireplaces. Additionally, outdoor air can act as a source for indoor NO_2 pollution.

Long-term monitoring activities over the last two decades indicate an increase in concentration of nitrogen oxides in urban areas throughout the world. Indoors, sources such as cooking with gas or cigarette smoking may be the main contributors to individual exposure. For example, the mainstream smoke from one cigarette may contain 100–600 μg of nitrogen oxides (see Table 1.4). Although some controversy exists about the relative proportions of nitric oxide and nitrogen dioxide, the latter is present initially, and other compounds are formed later (WHO, 1977).

1.3.3 Typical concentrations and exposures

Nitrogen dioxide in indoor air has been determined in several countries for many years. When comparing literature data, the time over which measurements have been averaged should be noted. Indications of the importance of the "measuring time interval" on the results are given in Table 1.4, in which the influence of the sampling time is shown. The values have been calculated using the same set of real-time data. It is clear that in rooms without any NO_2 source the measuring time interval is less important.

Owing to the widespread use of unvented combustion appliances, nitrogen dioxide concentrations in homes may considerably exceed those found out-

doors. The average nitrogen dioxide concentration over a period of several days may exceed 200 $\mu g/m^3$ when unvented gas stoves are used for supplementary heating, clothes-drying, or when kerosene (paraffin) heaters are used (Goldstein et al., 1979). Maximum 1-hour concentrations in kitchens were found to be in the range of 470-1880 $\mu g/m^3$ during cooking (Wade et al., 1975) and in the range of 1000–2000 $\mu g/m^3$ when, in addition, unvented gas-fired water-heaters are used (Lebret, 1985).

With regard to a special indoor environment it should be noted that it is frequent commercial practice to burn propane or kerosene in order to enrich glasshouse atmospheres with carbon dioxide as well as to provide heat. Under such conditions, concentrations of nitrogen oxides (mainly nitric oxide) often reach 3.5 ppm and remain high because air change is minimized in well sealed glasshouses. Thus, certain crops are exposed to concentrations of nitrogen oxides which far exceed the levels close to busy roads (Law and Mansfield, 1982).

In the environment, nitrogen dioxide exists as a gas. Thus, the only relevant route of exposure to humans is inhalation. Human exposures to NO_2 in indoor environments have been obtained primarily from indoor concentration studies and personal exposure monitoring studies in which the exposure is integrated over both indoor and outdoor microenvironments. Thus it is difficult to construct a data set of NO_2 observations alone obtained while the subject is in the target microenvironment which fits the criterion of exposure to a single pure compound. All studies show that indoor NO_2 levels are lower than the immediate outdoor values when there are no indoor sources (e.g., all-electric homes) due to the reactivity of NO_2 with indoor surface. In the presence of an indoor source the NO_2 levels are invariably higher than those outdoors. If measurements integrated over a several-day period are carried out the influence of infrequently used intermittent sources on the result is likely not to be detected.

The order of magnitude of mean indoor/outdoor ratios for nitrogen dioxide is 0.5–1 without indoor NO_2 source and 2–5 with indoor NO_2 source.

1.3.4 Health effects

NO_2 is a deep lung irritant which has been shown to generate biochemical alterations and histologically demonstrable lung damage in laboratory animals as a result of both acute and chronic exposures. The major health effects have been summarized (WHO, 1987). In laboratory animals, biochemical changes occur at exposure to concentrations of as low as 0.2 ppm for 30 minutes. Long-term animal studies have resulted in emphysema-like structural changes and increased susceptibility to bacterial lung infections. Changes at

the cellular level occur at the time of exposure, but biological effects are delayed, which complicates the understanding of long-term effects.

In humans, 80–90% of NO_2 can be absorbed upon inhalation. Controlled clinical studies have been conducted on susceptible subjects at concentrations in the range of 0.1 ppm to 5.0 ppm. Most studies show that substantial changes in pulmonary function can be demonstrated in normal, healthy adults at or above concentrations of 2 ppm. The evidence at lower concentrations is not as clear. Asthmatics appear to be responsive at about 0.5 ppm, and subjective complaints have been reported at that level. Below 0.5 ppm, small but statistically significant decrements in pulmonary function have been reported in asthmatics. Kagawa and Tsuru (1979) reported decrements in the lung function of asthmatics at concentrations as low as 0.15 ppm, but others have not substantiated these findings.

NO_2 increases bronchial reactivity as measured by pharmacological bronchoconstrictor agents in normal and asthmatic subjects, even at levels that do not affect pulmonary function directly in the absence of a bronchoconstrictor. Since the actual mechanisms are not fully defined and nitrogen dioxide studies with allergen challenges showed no effects at the lowest concentration tested (190 $\mu g/m^3$), accurate evaluation of the health consequences of the increased responsiveness to bronchoconstrictors is not yet possible.

Epidemiologic studies suggest that children who are exposed to combustion contaminants from gas stoves have higher rates of respiratory symptoms and illness than other children. Nitrogen dioxide concentrations in these studies ranged from 0.005 ppm to about 0.3 ppm (U.S. EPA, 1982b; WHO, 1987). In general, these results have not been observed in studies of adults.

1.4 SULPHUR DIOXIDE

1.4.1 Physico-chemical nature

Sulphur dioxide (SO_2) together with other sulphur compounds and particulate matter is a major air pollutant all over the world due to its generation during combustion of fossil fuels. From the health effects viewpoint two different entities are important:

(a) sulphur dioxide, and

(b) the acid aerosols that may result from the oxidation of sulphur dioxide in the atmosphere

Sulphur dioxide: Sulphur dioxide is a colourless gas with a strong pungent odour which can be detected at about 0.5 ppm (NRC, 1981). It is readily soluble in water and can be oxidized within airborne water droplets.

TABLE 1.5

SO_2 emission rates of indoor sources

Sources	Emission rates (mg/h)
Kerosene space heaters	
Radiant	31–109
Convective	34–94
Gas appliances	
Range	1.29–1.66
Oven	0.67–1.09

Adapted from: U.S. Department of Energy (1985).

Acid aerosol: Sulphuric acid (H_2SO_4) is a strong acid that is formed from the reaction of sulphur trioxide gas (SO_3) with water.

[Conversion factors: 1 ppm = 2.62 mg/m^3; 1 mg/m^3 = 0.38 ppm at 25°C and 101.3 kPa (1 atm)]

1.4.2 Occurrence and sources

Indoor SO_2 concentrations will invariably be lower than the respective outdoor concentrations due to the chemical reactivity of SO_2 with interior surfaces and the presence of neutralizing ammonia generated by humans and animals in the indoor environment. In special cases, where sulphur-containing products, e.g. kerosene, are burned indoors in an unvented device SO_2 will be generated indoors, and indoor values will exceed outdoor values. Table 1.5 provides a range of emission rates (in mg/h) of SO_2 for kerosene space heaters and gas appliances.

Rates of SO_2 oxidation depend on temperature, humidity, and concentrations of oxidants and catalytic components in the air. Indoor sources responsible for direct emissions of sulphuric acid are generally not significant except in some occupational environments.

1.4.3 Typical concentrations and exposures

As a result of the reduction of emissions, annual mean levels of ambient sulphur dioxide levels in major West European cities are now largely below 100 μg/m^3. Similarly, there has been a decrease in maximum daily mean values, which are now mainly in the range 200–500 μg/m^3. Peaks over shorter averaging periods, such as 1 hour, may extend to 1000–2000 μg/m^3 or higher especially under unfavourable meteorological conditions.

TABLE 1.6

Two-week average SO_2 levels for U.S. homes equipped with unvented kerosene heaters and/or gas stoves (modified from Leaderer et al., 1984)

Source category location	SO_2 ($\mu g/m^3$)			
	N	Mean	SD	% > 80 $\mu g/m^3$
One kerosene heater, no gas stove				
House average	25	68.4	86.8	24.0
Living room	25	72.4	92.3	28.0
Bedroom	25	62.9	98.0	20.0
One kerosene heater, gas stove				
House average	3	89.9	91.2	33.3
Living room	3	45.6	57.3	33.3
Bedroom	3	134.1	179.4	33.3
Two kerosene heaters, no gas stove				
House average	5	120.4	66.4	40.0
Living room	5	90.1	62.4	60.0
Bedroom	5	150.7	146.5	60.0
Two kerosene heaters, gas stove				
House average	2	110.0	81.5	50.0
Living room	2	118.0	102.4	50.0
Bedroom	2	101.9	60.7	50.0

As an example of SO_2 concentrations observed indoors in the presence of a source, Table 1.6 presents a summary of concentrations measured in U.S. homes with kerosene heaters and/or gas stoves. If there is no SO_2 source indoors the indoor/outdoor concentration ratio is between 0.1 and 0.6, with the lower value being observed at elevated outdoor SO_2 concentrations.

1.4.4 Health effects

Inhalation is the only route of exposure that is of interest in relation to the effects of sulphur dioxide and acidic aerosol on human health. In this context, however, only health effects on the respiratory tract will be considered.

Absorption of sulphur dioxide in the mucous membranes of the nose and upper respiratory tract occurs as a result of its solubility in aqueous media.

The absorption is concentration-dependent, with 85% absorption in the nose at 4–6 mg/m^3 and about 99% at 46 mg/m^3. Only minimal amounts reach the lower respiratory tract (EPA, 1982a; Ericcson and Camner, 1983). From the respiratory tract, sulphur dioxide enters the blood. Elimination occurs (after biotransformation to sulphate in the liver) mainly by the urinary route.

The deposition pattern of acid aerosols within the respiratory tract is dependent on the size distribution of the ambient droplets and humidity. Acidic ambient aerosol typically has a mass median aerodynamic diameter of 0.3–0.6 μm.

Some neutralization of the droplets can occur before deposition, due to the normal excretion of endogenous ammonia into the airways. Deposited free H^+ reacts with components of the mucus of the respiratory tract, changing its viscosity (Holma, 1985). The unreacted part of H^+ diffuses into surrounding tissues. The capacity of the mucus to react with H^+ is dependent on the H^+ absorption capacity, which is reduced in acidic saturated mucus as found, for example, in asthmatics.

In animal experiments (WHO, 1979b) and in occupational exposures to more than 10 000 μg/m^3, concentrations of sulphur dioxide in the range 2600–2700 μg/m^3 give rise to clearly noticeable effects with bronchospasm in asthmatics (Islam and Ulmer, 1979).

The effects of concern in relation to short-term exposures are those on the respiratory tract. There is an extremely large variability of sensitivity to sulphur dioxide exposure among individuals. This is true for normal persons, but especially so if asthmatics are included (Holma, 1985). Asthmatics have very labile airways and resistance is likely to change in response to many other stimuli, including pollens (WHO, 1979b; EPA, 1982a; Ericcson and Camner, 1983).

Effects of repeated and/or long-term exposures

Repeated short-term occupational exposure to high concentrations of sulphur dioxide combined with long-term exposure to lower concentrations can give rise to an increased prevalence of chronic bronchitis, especially in cigarette smokers. A possible contribution of simultaneously occurring sulphuric acid aerosol has, however, not been examined in these studies (Stjernberg et al., 1985).

Several epidemiological studies have associated the occurrence of pulmonary effects in communities with combined exposure to sulphur dioxide and particulates.

A continuum of response to sulphur dioxide exposures at relatively low concentrations has been observed in laboratory investigations on human volunteers. The magnitude of the effects was much enhanced when subjects

increased their breathing rates through exercise. The findings in a wide range of studies among asthmatics are consistent with a linear relationship (Kleinman, 1984) between magnitude of effect (in terms of proportionate increase in airway resistance) and dose of sulphur dioxide delivered to the airways (after allowing for removal of a substantial proportion in the nose or mouth). In a strict sense it would be difficult to define the lowest-adverse-effect level since this effect appears to be a function of the sensitivity of the subject, concentration, duration of exposure (10 minutes being the most usual duration of test exposure), level of activity and mucus rheological properties. It was, nevertheless, considered that effects of concern to the health of exercising asthmatic subjects were demonstrable down to sulphur dioxide levels of about 1000 $\mu g/m^3$, with discernible effects of less certain consequence below that level.

Another aspect, of great importance to public health, is the proportion of the population liable to be affected. Detailed information regarding the proportion of asthmatic or otherwise sensitive people in the community is not available, although estimates of around 5% have been suggested.

Sensory effects

At concentrations of 10 000 $\mu g/m^3$, sulphur dioxide has a pungent, irritating odour. Since the odour threshold of sulphur dioxide is at several thousand $\mu g/m^3$, this criterion is not critical in relation to public health.

The odour threshold for sulphuric acid has been estimated to be 750 $\mu g/m^3$ on the basis of one study and 3000 $\mu g/m^3$ on the basis of another (EPA, 1982).

Acute effects of acid aerosol

Sulphuric acid and other sulfates have been found to affect both the sensory and the respiratory function in humans. Respiratory effects from exposure to sulphuric acid (350 500 $\mu g/m^3$) have been reported to include increased respiratory rate and decreased maximal inspiratory and expiratory flow rates and tidal volume (EPA, 1982a; Ericcson and Camner, 1983). However, other studies of pulmonary function in nonsensitive healthy adult subjects indicated that pulmonary mechanical function was little affected when subjects were exposed to 100–1000 μg sulphuric acid per m^3 for 10–120 minutes.

Asthmatics are substantially more sensitive in terms of changes in pulmonary mechanics than healthy people, and vigorous exercise potentiates the effects at a given concentration. Effects could still be observed at 100 $\mu g/m^3$ in exercising adolescent asthmatics in studies using mouthpiece inhalation. However, the effects were relatively small and disappeared within about 15 minutes. In adult asthmatics undergoing similar protocols, the lowest-observed-effect level was 350 $\mu g/m^3$ (Ericcson and Camner, 1983; Utell and Morrow, 1986).

1.5 OZONE

1.5.1 Physico-chemical nature

Ozone (O_3) is one of the strongest oxidizing agents. It spontaneously decomposes into oxygen with a half-life of 3 days at 20°C. Smell perceptions (Gr. Ozein: 'it smells of something'), which occur even at a 1 to 10^8 dilution, are mostly due to NO_x formation. In the outer layers of the atmosphere (25 km), ozone is formed indirectly by the action of sunlight (at wavelengths lower than 240 nm) on nitrogen dioxide and recombination of the resulting oxygen radical with molecular oxygen. Wavelengths at 240–300 nm decompose ozone to oxygen.

[Conversion factors 1 ppm = 1.96 mg/m^3; 1 mg/m^3 = 0.51 ppm at 25°C and 101.3 kPa (1 atm)].

1.5.2 Occurrence and sources

There are no significant anthropogenic emissions of ozone into the atmosphere.

The presence of hydroxyl radicals and volatile organic compounds in the atmosphere, either of natural or of anthropogenic origin, causes a shift in the atmospheric equilibrium towards much higher concentrations of ozone.

The maximum ozone concentration that can be reached in polluted atmosphere appears to depend not only on the absolute concentrations of volatile organic compounds and nitrogen oxides but also on their ratio.

Examples of indoor sources are air cleaners, UV lighting, photocopying machines and laser printers.

1.5.3 Typical concentrations and exposures

Generally 120 μg/m^3 can be considered to be the upper limit of the 24-hour background outdoor concentration of ozone. In the early morning, some time is needed for the photochemical process to develop. Ozone peak concentrations of 350 μg/m^3 can be reached in the afternoon. Mean hourly ozone concentrations may exceed values of 240 μg/m^3 for 10 hours or more. During the night, ozone is scavenged by nitric oxide. Generally, ozone concentrations in city centres are lower than those in suburbs, mainly as a result of the scavenging of ozone by nitric oxide originating from traffic.

Without specific sources indoors, the main source of ozone in the indoor environment is outdoor air, the concentrations depending on the balance of the factors affecting ozone formation , transport and decomposition. Since in

indoor space, factors producing ozone decomposition are predominant, indoor/outdoor ratios vary from 0.10 to 0.25 for houses with restricted ventilation. An indoor/outdoor ratio of 0.80 has been measured in a building where the air was changed 10 times per hour with 100% outside air (Yocom, 1982).

1.5.4 Health effects

Ozone is a powerful oxidant and, as such, it can react with virtually every class of biological substance. In general, ozone exerts its action mainly through two mechanisms:

(a) the oxidation of sulfhydryl groups and amino acids of enzymes, co-enzymes, proteins and peptides; and

(b) the oxidation of polyunsaturated fatty acids to fatty acid peroxides.

All membranes are composed of both proteins and lipids, and appear for that reason to be an obvious target for ozone. Cells or organelles with a large specific surface (surface/volume ratio) may be extremely vulnerable (EPA, 1986a).

In a large number of controlled human studies, significant impairment of pulmonary function has been reported, usually accompanied by respiratory and other symptoms (EPA, 1986a; Von Nieding et al., 1979). Exposure to ozone generally was 1–3 hours. In most studies humans were exposed once to concentrations ranging from 200 to 2000 $\mu g/m^3$ (EPA, 1986a).

Field and epidemiological studies have indicated a number of acute effects of ozone and other photochemical oxidants. Eye, nose and throat irritation, chest discomfort, cough and headache have been associated with hourly average oxidant levels beginning at about 200 $\mu g/m^3$ (EPA, 1986a; Linn et al., 1982). Pulmonary function decrements in children and young adults have been reported at hourly average ozone concentrations in the range of 160–300 $\mu g/m^3$ (EPA, 1986a; Kagawa and Toyama, 1975; Lioy et al., 1985). Such symptoms may be responsible for the impairment of athletic performance in the range of 240–740 $\mu g/m^3$ (Wayne et al., 1967). In addition, an increased incidence of asthmatic attacks and respiratory symptoms has been observed in asthmatics exposed to similar levels of ozone (EPA, 1986a).

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Chapter 2

Organic Pollutants[1]

2.1 INTRODUCTION

In contrast to inorganic pollutants, organic compounds in indoor air occur in a much larger number of species and not all of those potentially involved in the generation of indoor air problems can even be measured; in the last decade, hundreds of such chemicals have been identified. Although most occur at concentrations which are several orders of magnitude below known effect levels, there is appropriate concern about their effect on human health. Some of the compounds are genotoxic and many exhibit toxic, irritant and/or odourant properties.

From a number of early small-scale studies (Johansson, 1978; Seifert and Abraham, 1982), it could be concluded that the concentration of many organic compounds in indoor air would exceed that in outdoor air. However, the data were insufficient to permit a thorough evaluation of the levels of exposure of the population. A compilation of 307 Volatile Organic compounds (VOC) identified in indoor air by authors from different countries was published in 1986 (Berglund et al., 1986).

Meanwhile knowledge on the occurrence of organic compounds in indoor air has greatly increased because a number of studies have been carried out in different countries. Such studies from the Federal Republic of Germany (Krause et al., 1987; Seifert et al., 1986)), Italy (De Bortoli et al., 1985), the Netherlands (Lebret et al., 1986), and the United States (Wallace, 1987) provided indoor concentration for a multitude of different VOCs in a sufficiently large number of homes to make exposure estimates for the general population.

Field surveys undertaken to determine frequency distributions of concen-

1 A part of the text of this chapter has been derived from World Health Organization (WHO), 1989 "Indoor Air Quality: organic pollutants", EURO Reports and Studies 111, WHO Regional Office for Europe, Copenhagen; U.S. Environmental Protection Agency (ECA), 1991 "Introduction to indoor air quality", EPA/400/3-91/003; World Health Organization (WHO) "Formaldehyde", Environmental Health Criteria, No. 89, Geneva; World Health Organization (WHO), 1987 "Air Quality Guidelines for Europe", WHO Regional Publications, European Series No. 23, WHO Regional Office for Europe, Copenhagen; U.S. Environmental Protection Agency (EPA), 1987 "Preliminary Indoor Air Pollution Information Assessment", Appendix A, EPA-600/8-87/014, pp. 2–18, 19, Office of Health and Environmental Assessment Washington DC.

TABLE 2.1

Classification of indoor organic pollutants

Category	Description	Abbreviation	Boiling-point range (°C)[a]	Sampling methods typically used in field studies
1	Very volatile organic compounds (incl. gases)	VVOC	<0 to 50–100	Batch sampling adsorption on charcoal
2	Volatile organic compounds	VOC	50–100 to 240–260	Adsorption on Tenax, carbon molecular black or charcoal
3	Semivolatile organic compounds	SVOC	240–260 to 380–400	Adsorption on polyurethane foam or XAD-2 [b]
4	Organic compounds associated with particulate matter or particulate organic matter	POM	>380	Collection on filters

a Polar compounds appear at the higher end of the range.
[b] Styrene–olivinylbenzene co-polymer
Taken from WHO, 1989.

trations of organic indoor pollutants, or of population exposures, usually do not consider the whole range of organic indoor pollutants but rather fractions of them. A WHO Working Group has categorized the entire range of organic indoor pollutants into four groups as indicated in Table 2.1 (WHO, 1989).

Groups or categories have been defined by boiling-point ranges, although no sharp limits exist between the four categories. The reason is that in practice the categories are determined or defined by the different methods used to collect organic pollutants from air. Sampling methods most frequently applied in field surveys are given in Table 2.1 for each category. Most of these methods rely on trapping organic compounds on adsorbent materials such as polymers, carbon molecular black or polyurethane foam. Volatility, which correlates fairly well with boiling-point, is the most important factor determining the range of compounds collected on a given adsorbent. However the sampling volume (which may vary with the required experimental or the available analytical sensitivity) and the compound polarities influence the range of sampled pollutants as well (see Section 2.2.1). As a result, categories of compounds cannot be separated by a single boiling-point value.

Sampling methods differ with respect to the required instrumentation,

volume, weight and cost. Therefore, their applicability to large scale surveys is subject to limitations that are different for the different methods and, hence, the different pollutant categories. As a result, concentration and exposure data of organic indoor pollutants are becoming available by category. Sufficient data for a tentative population risk assessment are for the time being available only for VOC.

Available data on SVOCs and POM are scarce with the exception of pesticides, polyaromatic hydrocarbons (PAHs), and polychlorinated biphenyls (PCBs). Due to their widespread diffusion and sanitary significance, attention has been focused on only these three compound classes.

SVOCs have also been defined (Clements and Lewis, 1988) as compounds with vapour pressures at ambient temperatures from approximately 10^{-2} to 10^{-8} kPa (10^{-1} to 10^{-7} torr). Depending upon their individual equilibrium vapour pressures and the temperature of the environment, SVOCs are found in the gas phase, distributed between gas and particle phases, or entirely in the particle phase. Sampling systems must therefore incorporate both filters and sorbent media.

For analytical reasons, some organic compounds cannot be included in the above classification scheme. Because of their reactivity or thermal liability, these compounds (e.g. formaldehyde) cannot easily be recovered from adsorbents or analysed by gas chromatography. Specifically designed sampling and analytical methods are required for their detection and measurement. As an example the 2.4-dinitrophenylhydrazine (2.4-DNPH) method is frequently applied for the detection of formaldehyde and other carbonyl compounds.

The sources of organic compounds in indoor air can be classified into three categories: outdoor air, man and his activities, and materials and equipment.

(a) Outdoor environment

All buildings exhibit a more or less pronounced exchange between outdoor and indoor air. In buildings with natural ventilation this exchange is highest if windows/doors are open, but also takes place, although at a reduced level, if they are closed (infiltration through cracks and interstices). In the case of mechanically ventilated buildings, the ventilation system forces outdoor air into the building shell to guarantee air exchange. Hence, outdoor air cannot be neglected as a source of contamination in indoor air, in particular if it exhibits an elevated level of pollution.

For mechanically ventilated buildings measures should be taken to clean the incoming air as much as possible. However, experience shows that this is frequently not done to the extent needed. Due to malfunctioning of the ventilation system or an unfavourable location of the air inlet (e.g., close to parking garages or loading docks) polluted outdoor air may become a noticeable

contributor to indoor air pollution. This contribution may be especially important under certain meteorological conditions (high pollution episodes).

A special case of indoor contamination originating from outdoors is the migration of gaseous substances into the building from the surrounding soil. Although radon is the most prominent example of such migration, a number of organic compounds may also play a role in the case of buildings constructed on waste sites.

Finally, the outdoor environment may indirectly contribute to indoor air pollution through organic substances originating from drinking water. In fact, chloroform and other volatile halogenated hydrocarbons have been found to reach non-neglectable concentration levels during showering (Andelman, 1985).

(b) Man and his activities

In addition to carbon dioxide and water vapour, human beings emit a large number of organic compounds, many of which contribute to body odour.

Energy production, e.g. the use of kerosene heaters, gas appliances or open fireplaces for cooking and heating, causes the presence of many indoor air pollutants among which volatile organic compounds (VOC) and semi-volatile organic compounds (SVOC).

Moreover, the large variety of existing household and hobby products makes it impossible to give a comprehensive overview of their individual components contributing to indoor air pollution.

Among the most important VOCs are those belonging to the classes of aliphatic, aromatic, and halogenated hydrocarbons (solvents), aldehydes and esters.

(c) Materials and equipment

While most human activities cause short-term intermittent emissions (Seifert and Ullrich, 1987), the use of building and renovation materials in a room generally results in a more or less continuous pollution of indoor air which may last several weeks, months or even years.

In the past, much attention has been paid to formaldehyde which can be emitted from materials such as wood products, carpets, textiles, but also from lacquers, glues, and sealants. Besides formaldehyde, a large number of VOCs are emitted from materials, especially shortly after their installation.

As many paints and lacquers today are produced using water as the basic solvent, they no longer emit important amounts of organic solvents. However, a number of new compounds are now being added to these products (van Faassen and Borm, 1990). As some of these have higher boiling points, they may significantly contribute to the increased occurrence of VOCs with high boiling points and also of SVOCs in the indoor environment.

Under certain circumstances, HVAC installations in air-conditioned buildings may be a vehicle for, or even a source of, organic indoor air pollution. In

the first case, organic substances resulting from emissions or spills in one room may be transported into other rooms if the percentage of recirculated air is high. In the latter case, growth of mould in a badly maintained HVAC system may produce different kinds of pollutants like odoriferous substances and even mycotoxins (Miller, 1990).

As many air-conditioned buildings are being used as offices, a number of organic compounds related to office work and office machines can be encountered in the air of such buildings.

As has been shown (Seifert and Schmahl, 1987; Borrazzo et al., 1990), adsorption of pollutants on walls, material surfaces, etc. may lower concentrations to a certain extent, especially in the case of polar organics. However, desorption may occur at a later stage and thus cause the presence of the pollutant(s) for a longer time in the air (Berglund et al., 1989).

2.2 VOLATILE ORGANIC COMPOUNDS

2.2.1 Physico-chemical nature

The Volatile Organic Compound (VOC) category has been defined (see Table 2.1) by a boiling-point range with a lower limit of between 50°C and 100°C and an upper limit of between 240°C and 260°C, where the higher values refer to polar compounds. Compounds of this category are typically sampled by adsorption on carbon molecular black, charcoal or the porous polymer Tenax.

VOCs encompass substances with widely varying physico-chemical properties. Among these, in particular two are of practical importance: (1) the lipophilic or hydrophilic character of the compounds, typically described by the octanol/water distribution coefficient and the water solubility; and (2) their neutral, basic or acidic character. The latter has to be taken into consideration for the chromatographic separation of VOC mixtures and determines the choice of appropriate chromatographic columns. The lipophilic or hydrophilic character of VOCs influences their retention on solid sorbents as demonstrated in Table 2.2. The table reports a few typical indoor air pollutants the safe sampling volume on Tenax (Health and Safety Executive, 1991) together with their boiling points, the logarithms of their octanol/water distribution coefficients and their water solubilities. The upper part of the table demonstrates that for unpolar lipophilic compounds (high K_{ow}, low water solubility) the safe sampling volume increases with increasing boiling point whereas the lower part of the Table shows how the effect of the boiling point on the safe sampling volume of a compound may be counterbalanced by its hydrophilicity: the lower

TABLE 2.2

Comparison of safe sampling volumes of selected VOCs on Tenax with their boiling points, octanol/water distribution coefficients and water solubilities

Compound	Safe sampling volume[1] on 200 mg of Tenax (l)	bp (°C)	log K_{ow}	Solubility in water (mg/l)
n-hexane	3.2	69	4.00[2]	12.96[3]
n-heptane	17	98	4.5 [2]	3.14[3]
n-octane	80	125	5.15[2]	0.88[3]
n-nonane	700	151	5.65[2]	0.22[3]
2-butanone	3.2	80	(0.79)[4]	(50500)[4]
t-butanol	not recommended	83	0.473[5]	miscible
isobutanol	2.8	108	0.693[5]	75800
1-butanol	5	118	0.823[5]	—
ethoxyethanol	5	135	−0.153[5]	miscible

1 Taken from Health and Safety Executive, 1991.
2 Recommended value: Sangster, J. Phys. Chem. Ref. Data, 1989, 18, 1111–1229.
3 Mean values calculated from: J. Devillier, D. Dominé and M. Chastrette "Data compilation for chemical fate models", final report, under contract no. 3860-89-11 ED ISP F, Ispra (VA): Commission of the European Communities, Joint Research Centre, 1990.
4 Values for 3-pentanone.
5 From CLOGP-3.3 data base, Medicinal Chemistry Project, Pomona College, Claremont, CA.

the K_{ow} or the higher the water solubility, the higher must be the boiling point in order to yield a given sampling volume. For example a boiling point difference of 14°C is not sufficient in order to compensate the difference of the water solubilities between *n*-hexane and *t*-butanol: the retention volume of *t*-butanol on 200 mg of Tenax is insufficient for safe sampling.

2.2.2 Occurrence and sources

Volatile organic compounds are ubiquitous in the indoor environment. The number of VOCs detected in indoor air is usually higher than in outdoor air and has continuously been increasing over the past decade. Already in 1981 over 250 different VOCs had been identified or tentatively identified in indoor air (Jarke et al., 1981). In 1986 the number of identified VOCs amounted to more than 300 (Berglund et al., 1986) and by 1989 over 900 VOCs had been identified (U.S. EPA, 1989).

VOCs are released into indoor air by almost all materials, consumer products, furnishings, pesticides, and fuels. They penetrate indoors together with outdoor

air by ventilation and can diffuse from polluted soil or be carried with contaminated drinking water (typically well-water) into the indoor environment.

Sources of VOCs in the indoor environment and the more important classes of compounds emitted by these sources are reported in Table 2.3.

TABLE 2.3

Sources of volatile organic compounds in indoor air

Sources	Typical contaminants
Consumer and commercial products	
Cleaners and waxes liquid floor wax, aerosol furniture wax, paste furniture wax, aerosol bathroom cleaner, unpressurized aerosol window cleaner, liquid all purpose cleaner, powdered abrasive cleaner, oven cleaners, dishwashing detergent, concentrated spot remover, aerosol and solid room deodorants	– aliphatic hydrocarbons (*n*-decane, branched alkanes) – aromatic hydrocarbons (toluene, xylenes) – halogenated hydrocarbons (tetrachloroethene, methylene chloride, 1,1,1-trichloroethane 1,4-dichlorobenzene) – alcohols – ketones (acetone, methyl ethyl ketone) – aldehydes (formaldehyde) – esters (alkyl ethoxylate) – ethers (glycol ethers) – terpenes (limonene, α-pinene)
Paints and associated supplies	
paints (oil, urethane, acrylic), varnishes and shellac, wood stains, paint thinners, paint brush cleaners, paint removers	– aromatic hydrocarbons (toluene) – aliphatic hydrocarbons (*n*-hexane, *n*-heptane) – halogenated hydrocarbons (methylene chloride, propylene dichloride) – alcohols – ketones (methyl ethyl ketone, methyl isobutyl ketone) – esters (ethyl acetate) – ethers (methyl ether, ethyl ether, butyl ether)
Pesticides	
termite treatment of homes, aerosol all-purpose household pesticides, roach killer (powder, liquid, spray), flea killer (powder, liquid dip, aerosol), mold and mildew inhibitors, houseplant insecticides, moth repellents, rodenticides (rat or mouse killer), fungicides (household disinfectants)	– aliphatic hydrocarbons (kerosene) – aromatic hydrocarbons (xylene) – halogenated hydrocarbons (chlordane, 1,4-dichlorobenzene, heptachlor, chloropyrifos, diazinon) – ketones (methyl isobutyl ketone) – organic sulphur/phosphorous compounds (malathion)

(continued)

TABLE 2.3 (*continuation*)

Sources	Typical contaminants
Adhesives	
rubber cement, plastic model glue, floor tile adhesive, ceramic adhesive, carpet adhesive, all-purpose adhesive	– aliphatic hydrocarbons (hexane, heptane) – aromatic hydrocarbons – halogenated hydrocarbons – alcohols – organic nitrogen compounds (amines) – ketones (acetone, methyl ethyl ketone) – esters (vinyl acetate) – ethers
Cosmetic/personal care products	
perfume, personal deodorants (aerosols, solids), body powder (talc), shampoo and body soaps, rubbing alcohol, hair sprays	– alcohols (propylene glycol, ethyl and isopropyl alcohol) – ketones (acetone) – aldehydes (formaldehyde, acetaldehyde) – esters – ethers (methyl ether, ethyl ether, butyl ether)
Automotive products	
hydraulic fluids, motor oils, gasoline, automotive cleaners, automotive waves	– aliphatic hydrocarbons (kerosene, mineral spirits) – aromatic hydrocarbons (benzene, toluene, xylenes) – halogenated hydrocarbons (tetrachloroethene) – alcohols (ethylene glycol, isopropyl alcohol) – ketones (methyl ethyl ketone) – amines (triethanolamine, isopropanolamine)
Hobby supplies	
photographic chemicals, specialty adhesives, clay dust, wood fillers	– aliphatic hydrocarbons (kerosene, hexane, heptane) – halogenated hydrocarbons (methylene choride, ethylene chloride) – alcohols (benzyl alcohol, ethanol, methanol, isopropyl alcohol) – aldehydes (formaldehyde, acetaldehyde) – ketones (methyl isobutyl ketone, acetone) – esters [di-(2-ethylhexyl)phtalate] (DEHP) – ethers (ethylene glycol ether) – amines (ethylene diamine)

(continued)

TABLE 2.3 (*continuation*)

Sources	Typical contaminants
Furnishings and clothing	
carpets, upholstered furniture, plastic furnite, shower curtains, draperies, blankets, mattresses	– aromatic hydrocarbons (styrene, brominated aromatics) – halogenated hydrocarbons (vinyl chloride) – aldehydes (formaldehyde) – ethers – esters
Building sources	
Building materials	
pressed wood products, gypsum board, construction adhesive, insulating materials, plastic piping, vinyl or plastic wall coverings	– aliphatic hydrocarbons (*n*-decane, *n*-dodecane) – aromatic hydrocarbons (toluene, styrene, ethylbenzene) – halogenated hydrocarbons (vinyl-chloride) – aldehydes (formaldehyde) – ketones (acetone, butanone) – ethers – esters (urethane, ethylacetate)
Heating, ventilating and air-conditioning systems	
furnaces (carbon-based fuels), air conditioner reservoirs	– aliphatic hydrocarbons
Garage	
vehicular exhaust, stored chemicals (gasoline, pesticides, paints, solvents)	– aromatic hydrocarbons (benzene) – chlorinated hydrocarbons – other substituted hydrocarbons
Combustion appliances	
unvented heaters (kerosene, gas), gas cooking stoves, woodburning stoves and fireplaces	– aliphatic hydrocarbons (propane, butane, isobutane) – aldehydes (acetaldehyde, acrolein)
Personal sources	
Tobacco smoke	– over 3800 compounds including: – organic nitrogen compounds (nicotine) – aldehydes (formaldehyde, acetaldehyde, acrolein) – ketones

(continued)

TABLE 2.3 (*continuation*)

Sources	Typical contaminants
Human and biological origin	
animal feces, pets, indoor plants (spores, pollen), metabolic products, pathogens	– aliphatic hydrocarbons (methane) – aromatic hydrocarbons (toluene) – aldehydes (acetaldehyde) – ketones (acetone, 2-hexanone – alcohols (3-methyl-1-butanol) – pesticides
Outdoor sources	
Outdoor air	
industrial emissions, contaminated groundwater, vehicular exhaust	– aliphatic hydrocarbons – aromatic hydrocarbons – halogenated hydrocarbons – aldehydes and ketones – alcohols – esters – ethers – organic nitrogen compounds – organic sulphur/phosphorous compounds
Potable water	
volatilization of VOCs during showering, bathing, other uses of potable water	– halogenated hydrocarbons (1,1,1-trichloroethane, chloroform, trichloroethane
Contaminated groundwater and soil	
seepage into basements	– VOCs

Source: Adapted from U.S. EPA (1987).

Source emission rates

The information on organic compounds emitted from equipment, materials and products is increasing. Unfortunately emissions are often complex: a large number of VOCs may be emitted and the emissions from each of them may vary with time in a different manner. Therefore also the characterization of emissions is a complex task which needs standardized procedures.

Table 2.4 summarizes published "total" emission rates for VOCs of a large number of sources collated by Levin (1991). "Total" VOC emission rates are usually determined by summing up the responses of a gas chromatographic detector (flame ionization detector, FID) for each individual compound emitted

and assuming a common response factor for all compounds. This assumption usually leads to underestimation of emissions of compounds other than hydrocarbons.

TABLE 2.4

Emission rates for floor coverings, 'wet' products and various building materials (taken from Levin, 1991)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Floor Covering				
Carpet	floor and wall covering, textile	83	new	Mølhave (1982)
Carpet	floor covering, synthetic fibres/PVC	120	new	Mølhave (1982)
Carpet	carpet, UF backing (1 h)	411	no seam	Davidson et al. (1991)
Carpet	carpet, UF backing (1 h)	62	new, direct from manufacturer	Davidson et al. (1991)
Carpet	carpet, UF backing (1 h)	98	aged	Davidson et al. (1991)
Carpet	carpet, UF backing (24 h)	202	no seam	Davidson et al. (1991)
Carpet	carpet, UF backing (24 h)	35	new, direct from manufacturer	Davidson et al. (1991)
Carpet	carpet, UF backing (24 h)	26	new, direct from manufacturer	Davidson et al. (1991)
Carpet	carpet, UF backing (140 h)	20	new, direct from manufacturer	Davidson et al. (1991)
Carpet	carpet, UF backing (140 h)	111	no seam	Davidson et al. (1991)
Carpet	carpet, UF backing (140 h)	6	new, direct from manufacturer	Davidson et al. (1991)
Carpet	felt carpet	80	new	Mølhave (1982)
Carpet	latex-backed carpet (336 h) 4-PC	80		Tucker (1988b)
Carpet	carpet, SBR latex backed (144 h)	45	new, direct from manufacturer	Black et al. (1991)
Carpet	carpet	36	<92 days	Wallace et al. (1987)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Carpet	felt carpet (synthetic fibres/plastic backing)	11	new	Mølhave (1982)
Carpet assembly	carpet, adhesive3 on concrete (24 h)	153000	single stick, new products	Black et al. (1991)
Carpet assembly	carpet, adhesive1, pad1 on concrete (24 h)	145000	double stick	Black et al. (1991)
Carpet assembly	carpet, adhesive1, on concrete (24 h)	98000	single stick, new products	Black et al. (1991)
Carpet assembly	carpet, adhesive2 on concrete (24 h)	88300	single stick, new products	Black et al. (1991)
Carpet assembly	carpet, adhesive4 on concrete (24 h)	783	single stick, new products	Black et al. (1991)
Carpet cushion	carpet, pad3 on concrete (24 h)	775	no adhesive, new materials	Black et al. (1991)
Carpet assembly	carpet, pad1 on underlayment (24 h)	549	no adhesive, new materials	Black et al. (1991)
Carpet assembly	carpet, pad1 on concrete (24 h)	136	no adhesive, new materials	Black et al. (1991)
Carpet cushion	cushion P-3 (144 h)	8110	new from factory	Black et al. (1991)
Carpet cushion	cushion P-3 (24 h)	3360	new from factory	Black et al. (1991)
Carpet cushion	cushion P-2 (24 h)	240	new from factory	Black et al. (1991)
Carpet cushion	cushion P-1 (24 h)	123	new from factory	Black et al. (1991)
Carpet cushion	cushion P-1 (144 h)	59	new from factory	Black et al. (1991)
Carpet cushion	cushion P-2 (144 h)	12	new from factory	Black et al. (1991)
Vinyl	vinyl floor covering	22280		van der Wal et al. (1990)
PVC	Central European PVC	7034	1–3 yrs, subject of complaint	Saarela and Sundell (1991)
PVC	floor covering, homogenous PVC	2300	new	Mølhave (1982)
PVC	Finnish PVC-covering	2192	0.5 yrs, unused, from roll	Saarela and Sundell (1991)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
PVC	Finnish PVC-covering	1629	1 yr, unused, from roll	Saarela and Sundell (1991)
PVC	Finnish PVC-covering	1443	<0.5 years, unused 2nd quality from roll	Saarela and Sundell (1991)
Rubber	floor covering, rubber	1400	new	Mølhave (1982)
PVC	English PVC-covering	1122	1 yr, unused, taken from roll	Saarela and Sundell (1991)
PVC	Finnish PVC-covering	273	2–3 yrs, subject of complaint	Saarela and Sundell (1991)
PVC	Swedish PVC-covering	91	1–2 yrs, subject of complaint	Saarela and Sundell (1991)
Vinyl	vinyl tile	45	Age < 98 days	Wallace et al. (1987)
Soft plastic	floor covering, soft plastic	590	new	Mølhave (1982)
Linoleum	linoleum floor covering	220	new	Mølhave (1982)
Linoleum	linoleum	64	30 yrs, subject of complaint	Saarela and Sundell (1991)
Linoleum	floor covering (linoleum)	22	new	Mølhave (1982)
Wood	pine, industrial (1 mo.)	682	0.1 yr, experimental surface coating	Saarela and Sundell (1991)
Wood	birch, industrial	272	0.1 yr, experimental surface coating	Saarela and Sundell (1991)
Wood	pine	264	1 yr, UF-lacquered on site	Saarela and Sundell (1991)
Wood	pine, untreated	216	new, in plastic wrapping	Saarela and Sundell (1991)
Wood	birch, industrial	157	0.1 yr, experimental surface coating	Saarela and Sundell (1991)
Cork	cork	805	0.3 yr, new material	Saarela and Sundell (1991)
Cork	Cork	7	2 yrs, subject of complaint	Saarela and Sundell (1991)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Wet Products				
Adhesive	wall and floor glue (24 h)	271000	water-based EVA	Mølhave (1982)
Adhesive	adhesive, wall and floor (24 h)	270000		Mølhave (1982)
Adhesive	floor adhesive	220000		Tucker (1988b)
Adhesive	carpet adhesive (24 h)	99000		Black et al. (1991)
Adhesive	carpet adhesive (24 h)	90000		Black et al. (1991)
Adhesive	carpet adhesive (24 h)	76600		Black et al. (1991)
Adhesive	carpet adhesive - low VOC (24 h)	698	low VOC formulation	Black et al. (1991)
Adhesive	carpet adhesive (144 h)	17200		Black et al. (1991)
Adhesive	carpet adhesive (144 h)	11900		Black et al. (1991)
Adhesive	carpet adhesive (144 h)	3950		Black et al. (1991)
Adhesive	carpet adhesive - low VOC (144 h)	76	low VOC formulation	Black et al. (1991)
Adhesive	carpet adhesive (7 d)	234		Wallace et al. (1987)
Adhesive	cove adhesive (7 d)	5000	methanol based vinyl adhesive	Wallace et al. (1987)
Adhesive	primer/adhesive (7 d)	6.1	wall primer/ adhesive	Wallace et al. (1987)
Adhesive	floor adhesive (10–100 h)	<5000		Tucker (1988b)
Adhesive	texture glue, PVA, water-based (24 h)	2100		Mølhave (1982)
Seam sealant	carpet seam sealant (1 h)	2960	seam sealant only	Davidson et al. (1991)
Seam sealant	carpet seam sealant (24 h)	249	seam sealant only	Davidson et al. (1991)
Seam sealant	carpet seam sealant (144 h)	10	seam sealant only	Davidson et al. (1991)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Caulk	silicone caulk (< 10 h)	13000		Tucker (1988b)
Caulk	silicone caulk (10–100 h)	<2000		Tucker (1988b)
Caulk	filler, PVA, glue-cement (24 h)	10200		Mølhave (1982)
Caulk	filler, sand, cement, hardener (24 h)	730	water based hardener	Mølhave (1982)
Caulk	latex caulk (7 d)	637	interior/exterior latex caulk	Wallace et al. (1987)
Caulk	sealing agent - putty strips (24 h)	340	new	Mølhave (1982)
Caulk	tightening fillet (24 h)	160	neoprene/ polyethylene	Mølhave (1982)
Caulk	Caulk - plasticized PVC/polyethylene (24 h)	56	new	Mølhave (1982)
Caulk	heat-expanding neoprene (24 h)	17	new	Mølhave (1982)
Caulk	tightening fillet, heat-expanding neoprene	16	new	Mølhave (1982)
Paint	Paint, acrylic latex	430		Mølhave (1982)
Paint	latex paint, 'high profile' (7 d)	249		Wallace et al. (1987)
Paint	latex paint, 'vinyl flat white' (7 d)	3.2		Wallace et al. (1987)
Stain	wood stain (< 10 h)	10000		Tucker (1988b)
Stain	wood stain (1–100 h)	<100		Tucker (1988b)
Varnish	polyurethane wood finish (< 10 h))	9000		Tucker (1988b)
Varnish	floor varnish, 2-part isocyanate (24 h)	4700		Mølhave (1982)
Varnish	floor varnish, clear epoxy (24 h)	1300		Mølhave (1982)
Varnish	floor varnish, acid hardener (24 h)	830		Mølhave (1982)
Varnish	polyurethane wood finish	<100		Tucker (1988b)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Sealant	sealing agent plastic compounds (24 h)	72000		Mølhave (1982)
Sealant	sealing agent silicone compound (24 h)	26000		Mølhave (1982)
Sealant	urethane sealant	0.13		Wallace et al. (1987)
Polish	spray polish for furniture	27100		Colombo et al. (1990)
Wax	floor wax (< 10 h)	80000		Tucker (1988b)
Wax	floor wax (10–100 h)	<5000		Tucker (1988b)
Wax	floor wax paste (initial emission rate)	1880		Colombo et al. (1990)
Various Building Materials				
Fibre board	woodfibre board (12 mm)	120	new	Mølhave (1982)
Fibre board	fibre board, glass fibre polyester reinforce	17	new	Mølhave (1982)
Gypsum board	calcium silicate board	64	new	Mølhave (1982)
Gypsum board	plaster board	26	new	Mølhave (1982)
Gypsum board	plaster board (12 mm, paper coated)	26	new	Mølhave (1982)
Gypsum board	gypsum board	26	age unknown	Tucker (1988b)
Gypsum board	water repellant mineral board	1.5	age unknown, from construction site	Wallace et al. (1987)
Insulation	insulation foam, polystyrene	1400	new	Mølhave (1982)
Insulation	polystyrene foam A	260		van der Wal et al. (1990)
Insulation	insulation foam, polystyrene	120	new	Mølhave (1982)
Insulation	polystyrene foam B	30		van der Wal et al. (1990)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Insulation	polystyrene foam insulation	22	purchased retail, age < 76 days	Wallace et al. (1987)
Insulation	insulation batt (mineral wool)	12	new	Mølhave (1982)
Insulation	fibrous glass insulation	0.8	from construction site	Wallace et al. (1987)
Insulation	duct insulation	0.28	purchased retail	Wallace et al. (1987)
Laminated board	laminated board (plastic)	0.4	new	Mølhave (1982)
Particleboard	Particleboard (new) formaldehyde	2000		Tucker (1988b)
Particleboard	Particleboard (24 h)	952		Black et al. (1991)
Particleboard	Particleboard (144 h)	837		Black et al. (1991)
Particleboard	Particleboard	200	2 yrs	Tucker (1988b)
Particleboard	Particleboard	140	new, from manufacturer	Mølhave (1982)
Particleboard	Particleboard (urea-formaldehyde glued)	130	new, from manufacturer	Mølhave (1982)
Particleboard	Particleboard	130	new, from manufacturer	Mølhave (1982)
Particleboard	Particleboard (urea-formaldehyde glued)	120	new, from manufacturer	Mølhave (1982)
Particleboard	Particleboard	120	new, from manufacturer	Mølhave (1982)
Particleboard	Particleboard	28	age < 98 days	Wallace et al. (1987)
Plywood	Plywood B	1450		van der Wal et al. (1990)
Plywood	Plywood A	900		van der Wal et al. (1990)
Plywood	Plywood C	725		van der Wal et al. (1990)
Plywood	Plywood D	215		van der Wal et al. (1990)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Sheathing	exterior mineral board	0.03		Wallace et al. (1987)
Chipboard	chipboard (age unknown)	130		Tucker (1988b)
Panelling	plywood panelling HCHO	100	new	Tucker (1988b)
Panelling	plywood panelling (teak)	44	new	Mølhave (1982)
Wall covering	vinyl and fibreglass wallpaper	300	new	Mølhave (1982)
Wall covering	wallpaper, PVC foam	230	new	Mølhave (1982)
Wall covering	wall covering, PVC	100	new	Mølhave (1982)
Wall covering	wallpaper (age unknown)	100		Tucker (1988b)
Wall covering	vinyl coated wallpaper B	95		van der Wal et al. (1990)
Wall covering	vinyl wallpaper	40	new	Mølhave (1982)
Wall covering	printed wallpaper	31	new	Mølhave (1982)
Wall covering	vinyl coated wallpaper A	20		van der Wal et al. (1990)
Wall covering	wall covering, Hessian	5.4	new	Mølhave (1982)
Miscellany				
Cement block	cement block	0.54		Wallace et al. (1987)
Cement flag	cement flag	73		Mølhave (1982)
Cable	small diameter telephone cable (new)	60	standard wall to telephone	Wallace et al. (1987)
Cable	large diameter telephone cable (new)	38	bundled wire, computer or network	Wallace et al. (1987)
Pipe	PVC pipe	0.53		Wallace et al. (1987)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Vapour barrier	tar pipe	6.3		Wallace et al. (1987)
Subfloor	concrete subflooring	<5		Black et al. (1991)
Trim	black rubber moulding	103	age < 124 days, from construction	Wallace et al. (1987)
Cove base	vinyl cove moulding	46	age < 98 days	Wallace et al. (1987)
Cove base	vinyl edge moulding	30		Wallace et al. (1987)
Textile	floor and wall covering, textile	1600		Mølhave (1982)
Cleanser	spray cleanser for carpets	50400	initial emission rate	Colombo et al. (1990)
Cleanser	liquid cleanser/disinfectant	34900		Colombo et al. (1990)
Cleanser	liquid floor detergent (initial)	2200		Colombo et al. (1990)
Pesticide	moth cake (p-DCB) @ 23 C	14000000		Tucker (1988b)
Dry-cleaning	dry cleaned clothes (0–1 day)	100		Tucker (1988b)
Dry-cleaning	dry cleaned clothes (1–2 days) Perc	50		Tucker (1988b)
Office chair	high back chair with arms (1 h)	1060	1 h	Strobridge and Black (1991)
Office chair	high back chair with arms (981 h)	100	981 h	Strobridge and Black (1991)
Office furniture	tackable acoustical partitions (HCHO) (1 h)	158	1 h	Strobridge and Black (1991)
Office furniture	tackable acoustical partitions (2.5 h)	74	2.5 h	Strobridge and Black (1991)
Office furniture	tackable acoustical partitions (HCHO) (48 h)	37	48 h	Strobridge and Black (1991)

(continued)

TABLE 2.4 (*continuation*)

Product type	Material or product description (time sampled during chamber test)	Emission rate TVOC ($\mu g/m^2 \cdot h$)	Acquisition age, description Comments	Ref.
Office furniture	tackable acoustical partitions (581 h)	6	581 h	Strobridge and Black (1991)
Workstation	workstation	3200	1 h	Strobridge and Black (1991)
Workstation	workstation	10	912 h	Strobridge and Black (1991)
Workstation	workstation (HCHO)	1470	1 h	Strobridge and Black (1991)
Workstation	workstation (HCHO)	830	336 h	Strobridge and Black (1991)

Modified from Levin (1991) Controlling Sources of Indoor Air Pollution, Indoor Air Bulletin, 1 (6): 1–11.
This table is reprinted with permission from the November 1991 issue of The Indoor Air Bulletin, H. Levin (Ed.). The Indoor Air Bulletin can be contacted at P.O. Box 8445, Santa Cruz, CA 95060, USA. Tel. 408 426624; Fax 408 426 6522.

2.2.3 Typical concentrations and exposures

Already from a number of early small-scale studies (Johansson, 1978; Mølhave and Møller, 1979; Jarke et al., 1981; Seifert and Abraham, 1982) it could be concluded that the concentrations of many VOCs in indoor air would exceed those in outdoor air.

By 1987, a number of field studies aimed at determining frequency distributions of concentrations for a multitude of different volatile organic compounds in the residential environment had been made available from Germany (Krause et al., 1987), Italy (De Bortoli et al., 1985), the Netherlands (Lebret et al., 1986), and the United States (Wallace, 1987). Analysing the data produced by these studies, a Working Group of the World Health Organization (WHO) concluded that, despite the somewhat different objectives and protocols of the studies, there was quite good agreement between the results. The Working Group therefore undertook the task of combining the results of these studies into a consolidated data set which is shown in Table 2.5 (WHO, 1989). The Working Group considered this data set to be fairly representative for "normal" indoor situations and useful for an estimate of population exposures.

TABLE 2.5

Concentrations and distributions of organic compounds in indoor air

Pollutant	Category (1)	Concentration ($\mu g/m^3$)						Sources
		Percentile				Mean	Outdoor[a]	
		10th	50th	90th	98th			
Aliphatic hydrocarbons								
n-hexane	2	4	10	20		10		solvent, fuel component
n-heptane	2	3	5	15	60	10		solvent, fuel component
n-octane	2	2	5	10		5		solvent, fuel component
n-nonane	2	2	5	20		10		solvent, fuel component
n-decane	2	3	10	50	90	20		solvent, fuel component
n-undecane	2	3	5	25		10		solvent, fuel cmponent
n-dodecane	2	2	5	10		5		solvent, fuel component
n-tridecane	2	2	5	10		10	<0.3	solvent, fuel component
n-tetradecane	2		2	10		2	<0.3	solvent, fuel component
n-pentadecane	2–3		1	5		1	<0.3	solvent, fuel component
n-hexadecane	2–3		1	5		1	<0.3	solvent, fuel component
3-methylpentane	2		5	80		5	2	solvent, fuel component
2-methylhexane	2		5	100		5	2	solvent, fuel component
3-methylhexane	2		2	100			2	solvent, fuel component
Terpenes								
limonene	2	2	15	70		30		odorant, detergent
α-pinene	2	2	10	20		10		wax, wood product

(continued)

TABLE 2.5 (*continuation*)

Pollutant	Category ([1])	Concentration ($\mu g/m^3$)						Sources
		Percentile				Mean	Outdoor[a]	
		10th	50th	90th	98th			
β-pinene	2	<1	<1	5		1		wax, wood product
α-terpinene	2	1	5	10		5		wax, wood product
Aromatic hydrocarbons								
benzene	2	2	10	20	30	10	3	fuel component, tobacco
toluene	2	30	65	150	250	80	5	fuel component, solvent
m,p-xylene	2	10	20	40		20	2	fuel component, solvent
o-xylene	2	3	5	10		10	1	fuel component, solvent
ethylbenzene	2	4	10	20	10		1	fuel component, solvent
n-propylbenzene	2	<1	2	6		3	<0.3	fuel component, solvent
isopropylbenzene	2		1	3		1	<0.3	fuel component, solvent
o-methylethylbenzene	2		2	5		5	<0.3	fuel component, solvent
1,2,3-trimethylbenzene	2		2	5		2	<0.3	fuel component, solvent
1,2,4-trimethylbenzene	2		5	20		10	1	fuel component, solvent
1,3,5-trimethylbenzene	2		2	5		5	<0.3	fuel component, solvent
n-buthylbenzene	2		1	10			<0.3	fuel component, solvent
p-methylisopropylbenzene	2		1	10			<0.3	fuel component, solvent
diethylbenzene	2							fuel component, solvent
styrene	2	<1	1	5	10			fuel component

(continued)

TABLE 2.5 (*continuation*)

Pollutant	Category (1)	Concentration (μg/m^3)						Sources
		Percentile				Mean	Outdoor[a]	
		10th	50th	90th	98th			
Chlorinated hydrocarbons								
chloroform	1–2		3	15				drinking water
dichloromethane	2		<10	<10	600			aerosol, paint remover
tetrachloromethane	2		1	20				industrial solvent
bromoform	2							drinking water
1,2-dichloroethane	2							cleaning agent, additive
1,1,1-trichloroethane	2	2	5	20		10		dry cleaning, solvent
trichloroethylene	2	1	5	20	30		<2	solvent, spot remover
tetrachloroethylene	2	2	5	20	70			dry cleaning, solvent
chlorobenzene	2		<0.5	10		1		solvent, textile additive
m-dichlorobenzene	2		<0.5	5			<0.5	deodorant, moth balls
p-dichlorobenzene	2	1	5	20				deodorant, moth balls
1,2,3-trichlorobenzene	2		1	10			<1	dye carrier, insecticide
1,2,4-trichlorobenzene	2		1	15			<1	dye carrier, pesticide
1,3,5-trichlorobenzene	2			5			<1	dye carrier, pesticide
Alcohols								
ethanol	1							solvent, beverages, fuel
propanol	1–2							solvent
n-butanol	2	<1	<1	3		1		solvent
isobutanol	2	<1	1	5		2		solvent

(continued)

TABLE 2.5 (*continuation*)

Pollutant	Category (1)	Concentration ($\mu g/m^3$)						Sources
		Percentile				Mean	Outdoor[a]	
		10th	50th	90th	98th			
pentanol	2							solvent, plasticizer
hexanol	2							solvent, plasticizer
2-ethylhexanol	2	<1	1	5		2		solvent, plasticizer
Esters and ketones								
ethyl acetate	2							solvent
n-butyl acetate	2							solvent
ethylmethyl ketone	2	<2	5	10		1		solvent
4-methyl-2-pentanone	2		2			1		solvent
Aldehydes								
formaldehyde	–[b]		25	60		40		chipboard, urea-formaldehyde insulation
acetaldehyde	–[b]		10	30				cigarette smoke
acrolein	–[b]							cigarette smoke
butanal	–[b]		1	5		1		
hexanal	2	<1	1	5				paper, paints
nonanal	2							paper, paints, flavour
Other compounds								
naphthalene	3		2	5				solvent, moth balls

1 See Table 2.1.
[a] A wide range of values can be found in the outdoor environment; higher values may be found near sources.
[b] These pollutants cannot be placed within the range of categories given in Table 2.1 as different sampling and analytical procedures apply.
Taken from WHO, 1989.

The 50 percentiles of most compounds amount to only a few micrograms per cubic meter and are several orders of magnitude below existing occupational threshold limit values. However, it should be pointed out that much higher concentrations up to tens of milligrams per cubic meter may occur indoors when special products are used or when permanent sources exist in or near a particular building.

In recent years organic compounds of a more polar character with volatilities in the upper range of the VOC class, such as glycol ethers and esters, are increasingly introduced and detected indoors. From Table 2.3 it results that consumer products such as cleaners and waxes, paints and hobby supplies are among their sources. Data on concentration distributions of these compounds in the residential environment are, however, not yet available.

Fewer data have been published on VOC concentrations in the office environment. Measurements in 83 offices (10 buildings) of the European Parliament (EP) in Brussels, Luxembourg, and Strasbourg are summarized in Table 2.6 (De Bortoli et al., 1990). Median, 90 percentile and maximum concentrations are reported for the most prominent and ubiquitous single VOC, for formaldehyde and acetaldehyde, and for total VOC (TVOC, toluene equivalent).

Median values are in the low microgram per cubic meter range. Compared to the values in Table 2.5 concentrations of hydrocarbon compounds are lower by a factor of 4–6, whereas the concentrations of chlorinated hydrocarbons and of aldehydes are comparable.

The distribution of a larger set of about 160 TVOC values measured in EP buildings including the values of Table 2.6 is shown in Figure 2.1 (De Bortoli, 1991). The high end values of the skewed distribution are partially due to infiltration of heavily contaminated air originating from a printing shop and partially due to chemicals commonly used in offices such as 1,1,1-trichloroethane from correction liquids.

It should be pointed out that care has to be taken when using TVOC data, because there is no generally agreed definition of TVOC and two VOC mixtures giving rise to the same TVOC value may have different composition and health implications (Seifert, 1990). Using the definition of TVOC applied in Table 2.6 and Figure 2.1, the contribution of non hydrocarbon compounds is underestimated and concentrations of compounds eluting earlier than *n*-hexane from the gas chromatographic column are not included.

Measurements of a larger range of individual VOCs have been performed by Maroni et al. (1992) in 18 offices of the administrative headquarters of a large industrial company near Milan. Detected concentration ranges, median and mean concentrations are reported in Table 2.7. The offices are air conditioned, have tightly sealed windows and are supplied with 100% outdoor air (no air recycling). The measured concentrations are similar to those measured

TABLE 2.6

Compounds most frequently observed in 83 offices (10 buildings) and their concentration distribution ($\mu g\ m^{-3}$)

Compound	Median (50% ile)	90% ile	Maximum (100% ile)
n-hexane	2.4	21	1730
n-heptane	1.0	24	194
benzene	<1.0	6.8	16
toluene	11	36	280
1,3-xylene + 1,4-xylene	4.7	10	20
1,1,1-trichloroethane	<20	40	3670
tetrachloroethene	2.4	160	1250
formaldehyde	45	122	139
acetaldehyde	9	24	57
total VOC ≥ C6 (1)	220	870	3930

1 Sum of the areas of gas chromatographic peaks measured by a flame ionization detector at elution times equal to or greater than that of *n*-hexane and divided by the response factor of toluene.

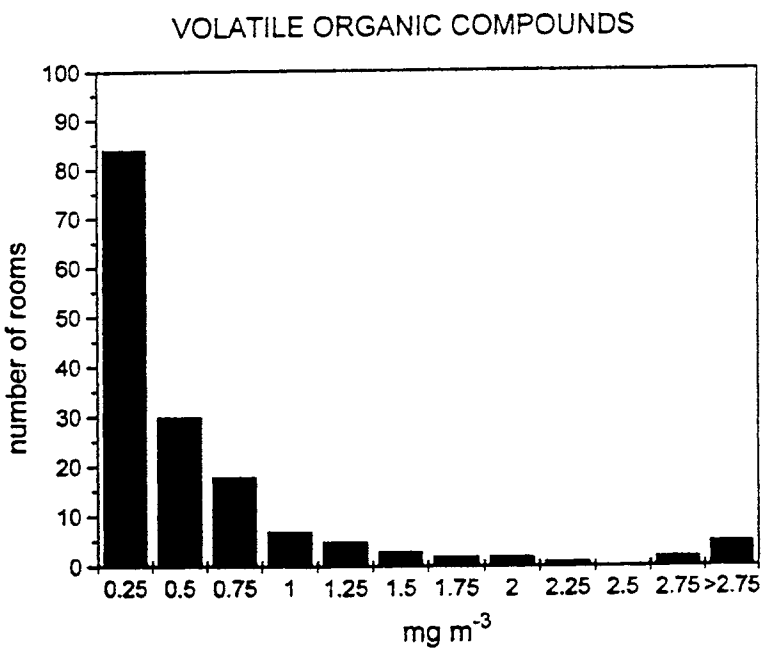

Fig. 2.1: Distribution of TVOC concentration values, determined in 10 buildings of the European Parliament (De Bortoli, 1991).

TABLE 2.7

Concentration ranges in offices provided with air quality control

Compound	No. of sampled offices	No. of quantifiable detections	Concentration ($\mu g/m^3$)		
			Range	Median	Mean[a]
Aliphatic hydrocarbons					
Hexane	18	17	<5–43	20	20
Heptane	18	15	<5–23[b]	12	11
Nonane	18	13	<5–32	10	11
Decane	18	13	<5–23[b]	10	11
Pentadecane	18	11	ND–30	10	-[c]
Isooctane	18	16	<5–41	11	14
5-methyl-decane	18	6	ND–19	ND	-[c]
3,4,4-trimethyldecane	18	7	ND–31	<5	-[c]
2,4-dimethylpentane	18	10	ND–30[b]	7	-[c]
2,5,6-trimethyloctane	18	4	ND–30[b]	ND	-[c]
2,2,4,6,6-pentamethylheptane	18	11	ND–54	8	-[c]
Cyclohexane	18	18	7–39	13	18
Methylcyclohexane	18	5	ND–33	ND	-[c]
Diethylcyclohexane	18	6	ND–37	ND	-[c]
2,4,4-trimethyl-2-pentene	18	8	ND–91	<5	-[c]
Aromatic hydrocarbons					
Benzene	18	6	<5–12	<5	5
Toluene	18	17	8–31[b]	16	16
Xylenes	18	15	<5–36[b]	17	16
Ethylbenzene	18	16	<5–32[b]	10	13
Propylbenzene	18	11	<5–37[b]	11	12
o-methylethylbenzene	18	11	<5–41[b]	8	12
1,3,5-trimethylbenzene	18	17	<5–29[b]	12	12
1,2,4-trimethylbenzene	18	17	<5–23	12	12
1,2,3-trimethylbenzene	18	16	<5–23[b]	11	12
2,5-dimethylethylbenzene	18	8	<5–22[b]	4	7
m-diethylbenzene	18	9	<5–21	5	8
o-methylpropylbenzene	18	10	ND–20[b]	4	-[c]
Naphthalene	18	3	ND–24	ND	-[c]

(continued)

TABLE 2.7 (*continuation*)

Compound	No. of sampled offices	No. of quantifiable detections	Concentration ($\mu g/m^3$)		
			Range	Median	Mean[a]
Chlorinated hydrocarbons					
p-dichlorobenzene	18	7	ND–21	<5	-[c]
Trichlorofluoromethane	18	3	ND–13	ND	-[c]
1,1,1-trichloroethane	18	7	ND–11	ND	-[c]
Terpenes					
Limonene	18	14	<5–39	18	17
Alcohols					
Ethanol	18	7	ND–32	ND	-[c]
Hexanol	18	6	ND–37	ND	-[c]
2-buthoxyethanol	18	15	<5–37	17	18
Aldehydes					
Formaldehyde	8	8	18–116	76	68
Acetaldehyde	8	8	35–53	40	42
Acroleine	8	8	1–5	3	3
Propanal	8	8	11–21	14	15
Butanal	8	8	2–6	4	4
Octanal	18	18	<5–26	10	10
Nonanal	18	18	<5–23	10	11
Benzaldehyde	25	25	<5–39	12	12
Total VOC $\geq C_6$	22	22	36–1920	578	565

ND = not detected.
[a] 2.5 $\mu g/m^3$ has been used for <5 interval while calculating the mean.
[b] Higher range values refer to a small surface printing office.
[c] The mean cannot be evaluated.

in the EP buildings although somewhat higher.

Important information about actual exposures to VOCs has been provided by the TEAM (Total Exposure Assessment Methodology) study (Wallace 1987). Weighted estimates of the average concentrations of 11 target compounds are summarized in Table 2.8.

TABLE 2.8

Typical exposures averaged over varying sampling periods

Contaminants	Weighted estimates of concentrations ($\mu g/m^3$) in personal air	
	Measurements in 2 cities over the course of 3 sampling periods in 3 years	Measurements in 1 city over the course of 3 sampling periods in 1 year
Total	200–338[a]	72–240[b]
1,1,1-Trichloroethane	45–94	16–96
m- and *p*-Dichlorobenzene	45–71	5.5–18
m- and *p*-Xylene	36–52	11–28
Tetracloroethylene	11–45	5.6–16
Benzene	NC[i]–28	–
Ethylbenzene	9.2–19	3.7–11
o-Xylene	12–16	4.4–13
Trichloroethylene	4.6–13	3.8–7.8
Chloroform	4.0–8.0	0.6–1.9
Styrene	2.1–8.9	1–3.6
Carbon Tetrachloride	ND[j]–9.3	0.8–1.3

a Sum of concentrations of the reported 11 compounds.
[b] Sum of concentrations of 19 compounds (reported compounds plus *n*-octane, *n*-decane, *n*-undecane, *n*-dodecane, α-pinene, 1,2-dichloroethane, *p*-dioxane, *o*-dichlorobenzene).
[i] Not calculated-high background contamination.
[j] Not detected in most samples.
Modified from Wallace, 1987.

The mean personal air exposures to the eleven prevalent target chemicals were always greater than the mean outdoor concentrations (with one exception at Los Angeles in February, where strong overnight inversions led to elevated outdoor concentrations). As a major reason for this, elevated indoor air levels at work and at home have been suggested. An additional reason for elevated personal air exposures may be the increased pollutant concentration around a "test person" performing professional or other activities during which VOCs are released, such as pumping gas, using a cleanser, or applying glues, paints, or other solvent containing products. High concentrations of pollutants in such a "personal activity cloud" are detected by personal but usually not by environmental monitoring (Rodes et al., 1991).

The value of personal samples as excellent indicators of exposure was confirmed by the TEAM study through measurements of exhaled breath, which proved to be a sensitive and noninvasive way of determining the

presence of the target chemicals in the blood. The breath levels correlated significantly with personal air exposures to nearly all chemicals but did not correlate with outdoor air levels.

During the TEAM study a number of specific sources of exposure were identified, including:

- smoking (benzene, xylenes, ethylbenzene, styrene in breath);
- passive smoking (same chemicals in indoor air);
- visiting dry cleaners (tetrachloroethene in breath);
- pumping gas or being exposed to autoexhaust (benzene in breath);
- various occupations, including: chemicals, plastics, wood processing, scientific laboratories, garage or repair work, metal work, printing etc. (mostly aromatic chemicals in daytime personal air);
- use of hot water in the home (chloroform in indoor air); and
- room air fresheners, toilet bowl deodorizers, or moth crystals (1,4-chlorobenzene in indoor air).

Many of these sources are activity related and therefore emphasize the potential importance of personal activity clouds for human exposure and, hence, of personal sampling for exposure assessment.

A further interesting result of the TEAM study is that the inhalation route provided more than 99% of the exposure for all chemicals except trihalomethanes. Drinking water provided nearly all of the exposure to the three brominated trihalomethanes, and a substantial fraction to most personal exposures to chloroform.

It should be noted that the target compounds of the TEAM study did not include polar VOCs such as ethyl or methylglycol, alcohols, esters or limonene which are often found in cleaning and conservation products (e.g. Colombo et al., 1991).

2.2.4 Health effects

Exposure to VOCs can result in both acute and chronic health effects. Table 2.9 summarizes some health effects for selected contaminants. Most of the available health effects data have been developed from animal studies or occupational studies. In general, the health effects data-base for VOCs, especially for low-level or intermittent exposures, is in its infancy.

Many of the VOCs are potent narcotics and result in depression of the central nervous system. VOCs can also result in irritation of the eyes and respiratory tract and sensitization reactions which involve the eyes, skin, and the respiratory tract. At higher concentrations, many of these chemicals have been shown to result in liver and kidney damage.

TABLE 2.9

Health effects and sources of selected volatile organic compounds

Compound	Health effects	Sources and uses
Benzene	carcinogen; respiratory tract irritant	plastic and rubber solvents; cigarette smoking; paints
Xylenes	narcotic; irritant; affect heart, liver, kidney and nervous system	adhesives, joint compound, wallpaper, caulking compounds, floor covering, floor lacquer, grease cleaners, shoe dye, tobacco smoke, kerosene heaters, varnish, solvent for resins, enamels; used in unleaded automobile fuels, pesticides, dyes, pharmaceuticals
Toluene	narcotic; may cause anaemia	solvents, solvent-based adhesives, water-based adhesives, edge-sealing, moulding tape, wallpaper, joint compound, calcium silicate sheet, vinyl floor covering, vinyl coated wallpaper, caulking compounds, paint, chipboard, kerosene heaters, tobacco smoke
Styrene	narcotic; affects central nervous system; possible human carcinogen	plastics, paints, synthetic rubber, and resins
Toluene diisocyanate (TDI)	sensitizer; probable human carcinogen	polyurethane foam aerosols
Trichloroethene	animal carcinogen; affects central nervous system	solvent for paints, varnishes, oil and wax, cleaning compounds, degreasing products, dry-cleaning
Ethyl benzene	severe irritation to eyes and respiratory tract; affects central nervous system	solvents, in styrene-related products
Dichloromethane	narcotic; affects central nervous system; probable human carcinogen	paint removers, aerosol finishers; acoustical office partitions
1,4-Dichlorobenzene	narcotic; eye and respiratory tract irritant; affects liver, kidney, and central nervous system	moth crystals, room deodorizers
Benzyl chloride	central nervous system irritant and depressant; affects liver and kidney; eye and respiratory tract irritant	vinyl tiles plasticized with butyl benzyl phtalate

(continued)

TABLE 2.9 (*continuation*)

Compound	Health effects	Sources and uses
2-butanone (MEK)	irritant; central nervous system depressant	floor/wall covering, calcium silicate sheet, fibreboard, caulking compounds, particleboard, tobacco smoke
Petroleum distillates	central nervous system; affect liver and kidney	cleaning products, solvents, paint thinners
4-phenylcyclohexene	eye and respiratory tract irritant; central nervous system effects	byproduct of styrene butadiene latex, used in most synthetic fibre carpets

For many indoor pollutants, there is insufficient data to determine the levels at which the specific effects listed would actually occur and the extent to which these levels are experienced in non-industrial indoor environments.
Source: Tucker (1988a), Dreisbach (1980), Turiel (1985).

Symptoms of VOC exposure (depending on the dose) could include fatigue, headache, drowsiness, dizziness, weakness, blurred vision, skin irritation, irritation of the eyes and respiratory tract, and cardiac arrhythmias (Rosenberg, 1990).

The term "solvent encephalopathy" is used to describe a group of symptoms (major symptoms — headache, irritability, difficulty concentrating, and fine-motor deficits) attributed to VOC exposures. A dose-effect relationship has not been described, but effects occur at levels below the threshold limit values for individual solvents, and there appears to be a relationship between duration of exposure and the time required to resolve symptoms after exposure stops. VOCs may be present in office environments at concentrations that have been associated with solvent encephalopathy (Hodgson, 1988).

Many of the VOCs which have been measured indoors are known human carcinogens (benzene) or animal carcinogens (carbon tetrachloride, chloroform, trichloroethene, tetrachloroethene, and 1,4-dichlorobenzene). VOCs such as 1,1,1-trichloroethane, styrene, and α-pinene are mutagens. Other VOCs such as octane, decane, and undecane are possible co-carcinogens. Although there are few risk assessments available for VOCs in indoor air, VOCs appear likely to pose a significant risk of cancer (U.S. EPA, 1989).

There is some evidence that VOCs can provoke some of the symptoms typical of the "Sick Building Syndrome" (SBS) (see Part IV for a definition and a discussion of this name). Mølhave observed increased mucous membrane

irritation and impaired memory in healthy subjects (who previously demonstrated symptoms of "sick building syndrome") who were exposed to 22 VOCs at total VOC concentrations of 5 mg/m^3 and 25 mg/m^3 (Mølhave et al., 1987).

Kjaergaard et al. (1987) also demonstrated a dose-dependent response in 63 randomly selected healthy subjects who were exposed to *n*-decane in the range of 0–100 ppm. Exposed subjects experienced mucous membrane irritation, decreased tear film stability, and sensation of increased odour intensity and reduced air quality. The authors concluded that these results support the hypothesis that VOCs can provoke some of the symptoms of the "sick building syndrome".

Some epidemiological studies have supported the hypothesis that VOCs are related to SBS symptoms. There are both cross-sectional and longitudinal studies available demonstrating a relationship between indoor VOC-concentrations and SBS-symptoms (Norbäck et al., 1990a; Norbäck et al., 1990b). In another longitudinal investigation in a suspected sick library building, a relationship was found between SBS-symptoms and the concentration of VOC (Berglund et al., 1990). In addition, a relationship between newly painted surfaces in dwellings and upper airway symptoms has also been demonstrated (Norbäck and Edling, 1991).

In an interesting cross-sectional study SBS symptoms of 147 office workers in five building areas were investigated using a linear-analog self-assessment scale questionnaire to define symptoms at a specific point in time (Hodgson et al., 1991). As a particular feature of this study, at the same time, the environment in the breathing zone was characterized by measuring, among other parameters, total volatile organic compounds. Analysis of the results showed that up to 25% of the variance in regression models could be explained by mucous membrane irritation and central nervous system symptoms. These two symptom groups were related to the concentrations of volatile organic compounds. This study suggests that the "sick building syndrome" may have specific environmental causes, including volatile organic compounds.

Individuals who appear to be affected by "Multiple Chemical Sensitivity" report severe reactions to a variety of VOCs and other organic compounds which are released by building materials and various consumer products including cosmetics, soaps, perfumes, tobacco, plastics, dyes, and other products. Many of the chemicals contained in these products are potent sensitizers.

These reactions can occur after exposure to a single sensitizing dose or sequence of doses, after which time a far lower dose can provoke symptoms. Reactions can also be provoked as a result of chronic exposure to low doses. Ashford and Miller (1991) summarize some of the studies that have been conducted and attempt to link multiple chemical sensitivity to exposure to VOCs and other organic compounds.

2.3 FORMALDEHYDE

2.3.1 Physico-chemical nature

Formaldehyde (HCHO) is the simplest and most common aldehyde found in the environment. It is perhaps the most important single indoor air pollutant because of its frequent occurrence and its strong irritation potential. Although formaldehyde is a volatile compound it is not detected by gas chromatographic methods applied to VOC analysis. At normal room temperature it is a colourless gas with a pungent odour. Formaldehyde is soluble in water and is used in a solution of about 30% or in polymerized form (paraformaldehyde).

Formaldehyde decomposes into methanol and carbon monoxide at temperatures above 150°C, although uncatalysed decomposition is slow below 300°C.

Under atmospheric conditions, formaldehyde is readily photooxidized in sunlight to carbon dioxide. It reacts relatively quickly with trace substances and pollutants in the air so that its half-life in urban air, under the influence of sunlight, is short. In the absence of nitrogen dioxide, the half-life of formaldehyde is approximately 50 min during the daytime; in the presence of nitrogen dioxide, this drops to about 35 min (Bufalini et al., 1972).

2.3.2 Occurrence and sources

The major anthropogenic sources of formaldehyde affecting human beings are in the indoor environment. They include cigarette smoke and other combustion sources, and urea-formaldehyde resins which are used in large quantities as glues in the manufacturing of wooden products such as particle board and plywood.

Emission from wooden products such as particle board or furniture made thereof results from the off-gassing of excess formaldehyde and of formaldehyde formed by hydrolytic cleavage of unreacted methylol groups in the resins. Because of this second contribution, formaldehyde can be released over long periods of time, even years, at a slowly decreasing rate.

Formaldehyde may also be used in urea formaldehyde foam insulation (UFFI). The foam can emit formaldehyde, even after completion of work, depending on factors such as processing and installation, age of foam, temperature, and humidity.

When considering the indoor presence of formaldehyde, it is necessary to differentiate between:

- Hospitals or other scientific facilities, where formaldehyde is used as a disinfectant or preservative; and

- All other indoor areas, especially living areas, schools, kindergartens, and mobile homes where there may be uncontrolled emissions of formaldehyde from:
  - the smoking of cigarettes and other tobacco products;
  - particle boards (mainly furniture);
  - urea formaldehyde foam insulation (UFFI);
  - urea-formaldehyde resin based lacquers and varnishes, e.g. for furniture and parquet;
  - gas cookers;
  - open fireplaces; and
  - other building materials made with adhesives containing formaldehyde, such as plastic surfaces.

2.3.3 Typical concentrations and exposures

Indoor levels of formaldehyde clearly differ from the concentrations in outdoor air. Indoor concentrations are influenced by temperature, humidity, ventilation rate, age of the building, product usage, presence of combustion sources, and the smoking habits of occupants.

The natural background concentration of formaldehyde in outdoor air is of the order of 1 $\mu g/m^3$. In urban air the annual average concentration is about 5–10 $\mu g/m^3$. Short term peak concentrations, about one order of magnitude higher, may occur in particular situations such as during peak traffic times or periods of smog. Elevated concentrations can also be found in the vicinity of industrial processes.

Concentrations measured in some larger studies ($n \geq 40$) in homes are reported in Table 2.10.

Formaldehyde concentrations found in buildings of the European Parliament are reported in Table 2.6.

In several countries over the past 10 years decreasing indoor concentrations of formaldehyde have been observed as a result of decreasing emission from materials used indoors. A competing factor is, however, the tendency to tighten buildings in order to save energy or improve insulation against external noise. The dependence of the indoor concentration level on both the emission rate of the sources and the ventilation rate makes it difficult to predict the effect of any measure to reduce the concentration levels acting on emission rates alone.

The total exposure of humans to formaldehyde is the result of contributions from various atmospheric compartments, from skin contact, and from ingestion. In a recent WHO publication (WHO, 1987) the contribution of ingestion has been estimated to amount to about 1–14 mg/day, most of it in a bound and

TABLE 2.10

Formaldehyde levels in homes[a]

Country	No. of homes	Formaldehyde concentration (mg/m^3)	Comments	Reference
Canada (1981)	378	0.042	homes without UFFI	UFFI (1981)
		(2.6% > 0.123)		
	1897	0.066	homes with UFFI	UFFI (1981)
		(10.4% > 1.23)		
Finland	432	0.20	average	Niemalä et al. (1985)
		0.11	25th percentile	
		0.33	75th percentile	
Germany	985	< 0.05	57.3% of samples	Prescher and Jander (1987)
		0.05–0.07	22.3% of samples	
		0.071–0.096	11.4% of samples	
		0.097–0.12	3% of samples	
		> 0.12	6% of samples	
USA	41	0.04	homes without UFFI	Ulsamer et al. (1982)
		(0.012–0.098)		
	636	0.15	homes with UFFI	
		(0.012–4.2)		
	244		UFFI homes	Breysse (1984)
		>1.23	2.8% of samples	
		0.61–1.22	1.9% of samples	
		0.12–0.60	24.1% of samples	
		<0.12	71.2% of samples	
	59		non-UFFI homes and apartments	
		>1.23	1.8% of samples	
		0.61–1.22	1.8% of samples	
		0.12–0.60	36.3% of samples	
		<0.12	60.1% of samples	

a Modified from: Meek et al. (1985). Where blanks appear, relevant information not available by authors.

TABLE 2.11

Contribution of various atmospheric compartments to average exposure to formaldehyde[a] (COST, 1990)

Source	Estimated concentration (mg/m^3)	Daily intake[b] (mg)
Ambient air (10% of time)	0.01	0.02
Indoor air		
Home (65% of time)		
– conventional	0.04–0.15	0.5–2
– prefabricated (chipboard)	0.08–0.80	1–10
Workplace (25% of time)		
– without occupational exposure[b]	0.04–0.16	0.2–0.8
– with 1 mg/m^3 occupational exposure	1	5
Environmental tobacco smoke (ETS)	0.02–0.20	0.1–1
Smoking (20 cigarettes/day)	–	1

a The contribution of food and water is ignored here.
[b] Assuming the normal formaldehyde concentration in conventional buildings.
[c] Assuming a respiratory volume of 20 m^3/day.

unavailable form (WHO, 1986b). Estimated average contributions of various atmospheric compartments to the total intake of formaldehyde are summarized in Table 2.11.

2.3.4 Health effects

Toxicological effects

Data on acute toxicity are available mainly from epidemiological studies of occupationally exposed populations and residents of buildings constructed of materials containing formaldehyde, and from controlled human exposure studies.

The symptoms displayed after short-term exposure to formaldehyde are: irritation of eyes, nose and throat together with exposure-dependent discomfort, lachrymation, sneezing, coughing, nausea and dyspnoea (Andersen and Mølhave, 1983). Symptoms are often more severe at the start of exposure and may diminish after some minutes or hours. Children have been reported to be more sensitive (Burdach and Wechselberg, 1980), their condition improving after removal of the sources. Table 2.12 shows that human responses start with an odour threshold at about 0.1 mg/m^3, progressing to eye and throat

TABLE 2.12

Effects of formaldehyde in humans after short-term exposure

Effects	Formaldehyde concentration (mg/m^3)	
	Estimated median	Reported range
Odour detection threshold (including repeated exposures)	0.1	0.06–1.2
Eye irritation	0.5	0.01–1.9
Throat irritation threshold	0.6	0.1–3.1
Biting sensation in nose, eye	3.1	2.5–3.7
Tolerable for 30 min (lachrymation)	5.6	5–6.2
Strong lachrymation lasting for 1 h	17.8	12–25
Danger to life, oedema, inflammation, pneumonia	37.5	37–60
Death	125	60–125

From WHO, 1987.

irritation at 0.5 mg/m^3; irritation and discomfort sharply increase between 1 and 20 mg/m^3, with danger to life at concentrations of 30 mg/m^3 and higher.

Numerous reports show that exposure to formaldehyde vapour causes direct, nonimmunological irritation of the skin (Report Consensus Workshop on Formaldehyde, 1984). A single application of 1% formalin in water on human skin will produce an irritant response in about 5% of the population (Maibach, 1983). Allergic contact dermatitis develops in man, but the threshold for induction is uncertain (Report Consensus Workshop on Formaldehyde, 1984).

A number of reports also show that formaldehyde gas exposure causes direct irritation of the respirable tract (reviewed in the Report Consensus Workshop on Formaldehyde, 1984). Because of absorption in the upper respiratory tract, higher concentrations of formaldehyde are required to stimulate bronchial receptors than those needed to cause sensory irritation. If formaldehyde is absorbed by particles, peripheral lung tissue receptors might be stimulated by the hydrolytic release of formaldehyde (Alarie, 1981). No precise thresholds exist for the irritant effects of inhaled formaldehyde; most people experience irritation of the throat within the range 0.12–3.7 mg/m^3 (Report Consensus Workshop on Formaldehyde, 1984).

Between 12 and 25 mg/m^3 symptoms become severe and it becomes difficult to breathe normally (Fassett, 1963). Effects in the pulmonary tissue and the lower airways are likely at concentrations of 6–40 mg/m^3, while pulmonary oedema, pneumonitis and pneumonia occur at concentrations of 60–120 mg/m^3

(Schachter et al., 1984). Some investigators report little or no adverse effect of formaldehyde on airway resistance in the range 1–3 mg/m^3 (Schachter et al., 1984), but above this range, if the subjects do moderate to heavy exercise, some decrease in lung function can occur.

A number of studies point to formaldehyde as a potential factor predisposing certain groups, particularly children, to respiratory tract infections (Helwig, 1977).

Sensitization arising from the release of formaldehyde into the circulation of chronic haemodialysis patients shows evidence of formaldehyde-dependent immunization (Report Consensus Workshop on Formaldehyde, 1984).

No sufficiently well controlled scientific studies have been carried out which definitely establish whether formaldehyde gas is able *per se* to cause respiratory tract allergy. Clinical reports describe tests for respiratory sensitivity to formaldehyde gas in which allergic reactions were elicited, but their interpretation is uncertain. There may be susceptible groups or genetic differences in the population. Occupational studies indicate that 1–2% of the population exposed to high concentrations may develop asthma (Frazier, 1980).

Neurochemical and morphological studies indicated changes in the nervous system of experimental animals. In short-term exposures of humans to concentrations of 0.3–2.5 mg/m^3 for 5 hours no changes in the performance of mathematical tests were observed (Andersen and Mølhave, 1983). Two studies of long-term formaldehyde exposure were carried out in adult populations. Olsen and Dossing (1982) observed no effects on the memory or concentration of subjects who underwent long-term exposure to 0.008–0.43 mg/m^3; similar results were obtained by Schenker et al. (1982).

Epidemiological studies in which neuropsychological symptoms due to occupational or environmental exposure to formaldehyde were evaluated have failed to overcome the problems commonly associated with such studies (Report Consensus Workshop on Formaldehyde, 1984).

Two occupational studies have been made on the irritant potential of formaldehyde in chronic disease. In a study at wood-processing plants, workers exposed to formaldehyde had a higher incidence of chronic upper respiratory disease (Efremov, 1970). Particle-borne formaldehyde was not measured in this study and the air concentrations of formaldehyde gas in all locations were below 0.4 mg/m^3. Workers in an acrylic-wool filter department where the working environment had phenol levels of 7–10 mg/m^3 and formaldehyde levels of 0.5–1.0 mg/m^3 had lower lung function values, but they may also have been exposed to particles and fibres that were not monitored (Schoenberg and Mitchell, 1975).

A number of studies are even more difficult to interpret. It is unlikely that chronic obstructive lung disease would occur in people exposed to less than 1.8

mg/m^3 and it is possible that some chronic disease might occur in those exposed to concentrations above 10 mg/m^3.

Several studies have addressed the possible links between formaldehyde and miscarriage, frequency of menstrual disorders, pregnancy complications and low-birth-weight babies, and reported increased effects. However, owing to the scarcity and limitation of the available data, it is not possible to draw definite conclusions (Report Consensus Workshop on Formaldehyde, 1984).

Mutagenic and carcinogenic effects

In the 1980s a number of occupational studies were carried out to assess the potential carcinogenicity of formaldehyde in humans: five historical cohort studies (Harrington and Shannon, 1975; Wong, 1983; Acheson et al., 1984; Levine et al., 1984; Harrington and Oakes, 1984), three proportional mortality studies (Marsh, 1982; Walrath and Fraumeni, 1983; Liebling et al., 1984) and six case control studies (Anderson et al., 1982; Fayerweather et al., 1983; Hernberg et al., 1983; Olsen et al., 1984; Brinton et al., 1984; Hayes et al., 1984). The subjects investigated were workers who used formaldehyde in the preservation of biological tissues (embalmers, anatomists and pathologists) and industrial workers involved in the production and use of formaldehyde. All these studies have their limitations: confounding factors such as smoking or exposures to other chemicals could not be taken into account in the design or analysis of most of the investigations. In addition, quantitative information about exposure is usually not available. None of the studies provides conclusive evidence about the carcinogenicity of formaldehyde in humans. However, owing to the low statistical power of the best studies conducted, the possibility cannot be excluded that formaldehyde is a human carcinogen. One study indicates a possible slight excess risk of nasal cancer (Brinton et al., 1984), and several studies found a slight excess in cancer of other organs such as brain or skin, however, it is not possible to clearly attribute the risk to formaldehyde.

No detectable differences in the frequency of chromosome aberrations and sister chromatid exchange were found in six pathology workers compared with unexposed controls (Thomas et al., 1984). A study of haemodialysis patients showed marked chromosome abnormalities. Such patients are at risk with regard to direct uptake of formaldehyde via equipment, but the study did not investigate control subjects and is therefore difficult to interpret (Goh and Cestero, 1979).

Sensory effects

Formaldehyde has a pungent odour. The odour detection threshold may be as low as 0.06 mg/m^3 (Berglund et al., 1985); the odour recognition threshold

is not known. It is not possible, on the basis of the scientific literature, to state a specific limit concentration of formaldehyde at which odour nuisance starts to appear. Indoor air usually contains other organic compounds which, in combination with formaldehyde or by themselves, may have odorous and irritating properties causing discomfort (Ahlström et al., 1984). It has been reported that some sensitive individuals can sense formaldehyde concentrations of 0.01 mg/m^3 and perhaps even lower concentrations by a "warm" feeling on the face (Ahlström et al., 1984).

2.4 PESTICIDES

2.4.1 Physico-chemical nature

Pesticides (including, among others, fungicides, insecticides, rodenticides, termiticides, and germicides) are used both by pest control agents in agriculture and by occupants of indoor spaces to eliminate a wide variety of organisms, ranging from rodents to insects, fungi, bacteria, and viruses. By definition, they are poisons but their range of toxicity varies with their target. They include several chemical classes, such as for example chlorinated hydrocarbons, carbamates, organophosphates, and dicoumarins. Their use, storage, and disposal in agriculture and public health are regulated, but cautionary information to home-owners in particular must be clarified and emphasized.

Many insecticides are lipophilic, i.e., they are soluble in fat. Insecticides sometimes are dissolved in petroleum distillates which are mixtures of low molecular weight aliphatic and aromatic hydrocarbons. Toluene or xylene are added to some formulations to stabilize the insecticide or to make it more emulsifiable. Alcohols, glycols, ethers, or chlorinated solvents are used as carriers (also called vehicles) for insecticides which are not strongly lipophilic. These carriers also make the insecticide more likely to be absorbed through the skin.

Due to the great variety of existing pesticides it is not possible here to give detailed information on the physico-chemical data of individual compounds. Exhaustive information on all existing pesticides can be found in specific books (Hayes and Laws, 1991) or in manuals (The Pesticide Manual, 1991). Some general characteristics are described for three important families.

Chlorinated hydrocarbon insecticides

DDT is the best known of the chlorinated hydrocarbon insecticides. All of them are aryl, carbocyclic, or heterocyclic compounds of molecular weights

ranging from 291 to 545. It is conventional to divide the chlorinated hydrocarbon into four groups: DDT (dichlorodiphenyl-trichloroethane) and its analogs, BHC (isomeric mixtures of benzene hexachloride), cyclodiene and related compounds, and toxaphene (chlorinated terpene mixtures).

Carbamate pesticides

Carbamates are *N*-substituted esters of carbamic acid. In general, simple esters or *N*-substituted derivatives of carbamic acid are unstable compounds, especially under alkaline conditions. When decomposition takes place, the parent alcohol, phenol, ammonia, amine, and carbon dioxide are formed. The salts and esters of substituted carbamic acid are more stable than carbamic acid. This enhanced stability is the basis for the synthesis of many derivatives that are biologically active pesticides.

Carbamate ester derivatives are crystalline solids of low vapour pressure with variable, but usually low, water solubility. In general, they are poorly soluble in non-polar organic solvents such as petroleum hydrocarbons but highly soluble in polar organic solvents such as methanol, ethanol, acetone, and dimethylformamide.

Organic phosphorus pesticides

The compounds are normally esters, amides, or thiol derivatives of phosphoric or phosphonic acid. Active ingredients of commercial products can be classified into three main groups: phosphates (without a sulphur atom), phosphothioates (with one sulphur atom), phosphodithioates (with two sulphur atoms). Since the –P=S moiety is intrinsically more stable, many insecticides are manufactured in this form which can then be converted into the biologically active –P=O group in tissues. Oxidation of phosphodithioates to phosphates is potentially dangerous, since the phosphates are more volatile and are directly toxic agents. This can occur by oxidation of stored products at elevated temperatures.

In order to be useful, these compounds must be reasonably stable at a neutral pH. Many are formulated as concentrates in oil, in water-miscible solvents such as ethylene glycol monomethyl ether, or are absorbed onto inert granules for application directly or after dispersion in water. However, nearly all are rapidly hydrolysed by alkali and many are also unstable at pH levels below 2.

The values for vapour pressure given in Table 2.13 refer to some active ingredients in household pesticides.

TABLE 2.13

Vapor pressures of some important ingredients used in household pesticides

Pesticide common name	Molecular weight	Vapour pressure (mm Hg)	Temperature (°C)
Chlorinated hydrocarbons			
DDT (dicofol, methoxychlor)	355	$1.5 \cdot 10^{-7}$	20
Chlordane	410	$1 \cdot 10^{-5}$	25
Lindane	291	$3.3 \cdot 10^{-5}$	20
Carbamates			
Carbaryl	201	$<5 \cdot 10^{-3}$	26
Propoxur	209	$6.5 \cdot 10^{-6}$	20
Organophosphates			
Diazinon	304	$4.1 \cdot 10^{-4}$	20
Dichlorvos	221	$1.2 \cdot 10^{-2}$	20
Malathion	330	$4 \cdot 10^{-5}$	30
Parathion	291	$3.8 \cdot 10^{-5}$	20
Naled	381	$2 \cdot 10^{-3}$	20

Modified from Hayes, 1982.

2.4.2 Occurrence and sources

It is estimated that 94% of pesticide usage is for agricultural purposes. In the home, pesticides are used to kill pests on lawns, trees, shrubs, flowers, and vegetables. In some regions, pesticides are almost universally used to control termite infestations either before or after construction. Pesticides are applied to living species to rid them of unwanted pests. People also use these products on themselves or on their pets to prevent the bites from mosquitos, chiggers, flies, ticks, fleas, and other pests. Pesticides, herbicides, and fumigants used in agriculture may become airborne and attached to airborne particulate matter, or, through soil runoff, contaminate rivers, lakes, and streams which may be a source of water supplies. A study on the role of house dust in, for example, DDT pollution (Davies, 1972) indicates that house dust can be a principal source of insecticides. The concentrations of DDT were higher than those found in soils in the area. Table 2.14 shows that there are many sources leading to indoor exposures of insecticides (Lewis et al., 1986). As indicated in Table 2.14, many pesticides are used directly in the indoor environment or in its close proximity (e.g., foundation treatment of termites). Chlordane has been

TABLE 2.14

Sources of pesticide exposure in or near houses

Compound	Type of pesticide	Household uses leading to potential human exposure
Chlorpyrifos	Insecticide	Control of mosquitoes, cockroaches and other household insect; turf and ornamental insects; fire ants, termites, and lice
Pentachlorophenol	Fungicide, Insecticide	Exterior wood preservative
Chlordane	Insecticide	Subterranean termite control
Ortho-Phenylphenol	Disinfectant fungicide	Household disinfectant; post-harvest application to fruits and vegetables
Propoxur	Insecticide	Control of cockroaches, flies, mosquitoes; lawn and turf insect
Resmethrin	Insecticide	Control of flying and crawling insects; fabric protection; pet sprays and shampoos; application on horses and in horse stables; greenhouse use
Dicofol	Insecticide	Control of mites on fruit, vegetable, and ornamental crops
Captan	Fungicide	Seed protectant; fungal control on fruits, vegetables, and berries
Carbaryl	Insecticide	Control of insects on lawns, ornamentals, shade trees, vegetables, and pets
Lindane	Insecticide	Seed treatment; insect control in soil, on vegetables, ornamentals and fruit and nut trees
Dichlorvos	Insecticide	Household and public health insect control; flea collars and no-pest strips
2,4-D esters	Herbicide	Post-emergent weed control
Malathion	Insecticide	Insect control on fruits, vegetables, ornamentals, and inside homes
Permethrin (*cis* and *trans*)	Insecticide	Control on flies, mosquitoes, ants, cockroaches, garden insects
Heptachlor	Insecticide	Subterranean termite control
Aldrin	Insecticide	Subterranean termite control
Dieldrin	Insecticide	Subterranean termite control
Ronnel	Insecticide	Fly and cockroach control
Diazinon	Insecticide, nematicide	Control of soil and household insects, grubs and nematodes in turf; seed treatment and fly control
Methoxychlor	Insecticide	Control of insects in garden, fruit, and shade trees
Atrazine	Herbicide	Weed control
a-Hexachloro-cyclohexane	Insecticide	Manufacture and use discontinued in U.S.; ubiquitous in air, residue from lindane

(continued)

TABLE 2.14 (*continuation*)

Compound	Type of pesticide	Household uses leading to potential human exposure
Bendiocarb	Insecticide	Household, ornamental, and turf insect control
Folpet	Fungicide	Fungus control on flowers, ornamentals, seeds, plant beds; paints and plastics
Chlorothalonil	Fungicide	Broad spectrum fungicide; wood preservative; paint additive
Dacthal	Herbicide	Selective pre-emergent weed control on turf, ornamentals, and vegetable crops
Oxychlordane	—	Oxidation product of chlordane
Heptachlor epoxide	—	Oxidation product of heptachlor
trans-Nonachlor	—	Component of chlordane

Source: Lewis et al. (1986).

detected in the air of some termiticide-treated homes as long as 14 years after application (Repace, 1982). It is also well recognized that many insecticides and herbicides find their way to man through the food chain.

A widely used product of concern is the pest strip. Generally, these strips contain DDVP (dichlorvos or Vapona) as the main ingredient. DDVP is of concern because it is classified as a possible human carcinogen, and it also causes liver and nerve damage in animals. Pesticides from these strips vaporize into the surrounding air, and prolonged exposure in an enclosed space is likely to increase health risks.

The application of pesticides to control subterranean termites may cause exposure to the occupants of the house. Prior to 1987, it was thought that when cyclodiene termiticides such as chlordane were applied correctly for subterranean termite control, the occupants of treated homes would not be exposed to the pesticide. However, studies in 1987 demonstrated that pesticides used for subterranean termite control can be found at low concentrations in the air of properly treated homes. As a result, EPA has taken a series of actions which have led to the withdrawal of cyclodiene termiticides from the market (U.S. EPA, 1991).

2.4.3 Typical concentrations and exposures

Information on concentrations of and exposures to pesticides in the indoor environment has mostly been made available in the USA.

In 1985, EPA extended its earlier work by developing a methodology for determining pesticide exposures in the general population (U.S. EPA, 1990). The methodology used in this study, which is known as the Non-Occupational Pesticides Exposures Study (NOPES), was designed as a means of developing estimates of exposure to some of the most commonly used household insecticides via air, drinking water, food, and dermal contact. In the two cities which were studied (Jacksonville, Florida, and Springfield/Chicopee, Massachusetts), the average number of pesticides in the home was 4.2 and 5.3 respectively.

Table 2.15 summarizes some of the indoor concentrations from the study. For the majority of the 34 target compounds which were studied, indoor air concentrations were substantially higher than outdoor air concentrations, and personal air concentrations were usually similar to indoor air concentrations. Another finding of this study was that seasonal variations existed for many of the compounds. This effect appears to be compound-specific and complex, and it probably reflects the interaction of many variables including temperature, patterns of pesticide usage, use of heating and cooling systems, and occupant activities.

The study also attempted to assess the relative contributions of air, food, water, and dermal exposure in the two cities tested. Based on limited data, it appears that exposure from water ingestion was negligible. Food appeared to be a dominant contributor for some compounds, while air dominated for others. Limited data were collected for dermal exposure, but the importance of this pathway needs further study.

2.4.4 Health effects

Most pesticides are inherently toxic and the potential hazards posed by these chemicals are magnified by improper use and storage. In the USA during the 1976 and 1977 nationwide survey, the EPA found that less than 50% of the people who participated in the survey read pesticide labels for application procedures. About 85% of the people used pesticide products without reservation and only 9% used these products with caution (U.S. EPA, 1987).

Table 2.16 summarizes some general statements of the health effects, products, and uses of 24 active ingredients in insecticides used in and around residences. The actual risks of any of these pesticides will depend on a variety of factors including the application method, protective measures and ventilation.

Pesticides currently in use include a wide variety of chemicals with great differences in their mode of action, uptake by the body, biotransformation and elimination from the body. Therefore, the resulting toxicity to humans varies greatly for different compounds and depends on the chemical structure of the molecules.

TABLE 2.15

Selected weighted summary statistics for indoor air concentrations of pesticides in Jacksonville and Springfield/Chicopee (ng/m^3)[a]

Pesticide		Jacksonville			Springfield/Chicopee		
		Summer	Spring	Winter	Summer	Spring	Winter
Gamma-BHC	mean	20.2	13.4	6.0	*	0.5	9.5
	max	245.0	1530.0	75.0		5.0	118.0
Chlorothalonil	mean	5.3	2.2	6.7	*	0.1	0.1
	max	264.0	51.0	523.0		35.0	9.2
Heptachlor	mean	163.4	153.9	72.2	*	31.3	3.6
	max	1600.0	2370.0	684.0		253.0	152.0
Dichlorvos	mean	134.5	86.2	24.5	*	4.3	1.5
	max	2280.0	2910.0	1090.0		324.0	158.0
Chlorpyrifos	mean	366.0	205.4	120.3	*	9.8	5.1
	max	2170.0	4350.0	1043.3		252.0	291.0
Aldrin	mean	31.3	6.8	6.9	*	0.0	0.3
	max	1840.0	320.0	106.0		0.0	3.9
Dieldrin	mean	14.7	8.3	7.2	*	1.0	4.2
	max	177.0	61.0	57.0		8.8	40.0
Dicofol	mean	0.0	11.0	0.0	*	0.0	0.0
	max	0.0	581.0	0.0		0.0	0.0
Chlordane	mean	324.0	245.6	220.3	*	199.4	34.8
	max	3020.0	4380.0	2050.0		1700.0	735.0
Ortho-phenyl phenol	mean	96.0	70.4	59.0	*	44.5	22.8
	max	1040.0	1240.0	1440.0		560.0	286.0
Propoxur	mean	528.5	222.3	162.5	*	26.7	17.0
	max	7920.0	2030.0	1370.0		505.0	669.0
Bendiocarb	mean	85.7	5.5	3.4	*	0.2	0.4
	max	1500.0	89.0	68.0		10.0	38.0
Diazinon	mean	420.7	109.2	85.7	*	48.4	2.5
	max	13700	2370.0	1080.0		1810.0	27.0
Carbaryl	mean	68.1	0.4	0.0	*	0.3	0.0
	max	3190.0	97.0	0.0		16.0	0.0
Malathion	mean	20.8	15.0	20.4	*	5.0	0.0
	max	1890.0	240.0	1660.0		275.0	0.0

a Table adapted from "Introduction to Indoor Air Quality", EPA/400/3-91/003 (July 1991) only pesticides with at least one "mean" value 5 ng/m^3 have been included.
* These pesticides were not sampled during the summer in Springfield/Chicopee.

TABLE 2.16

Summary of health effects, products, and uses of 24 active ingredients in household insecticides (adapted from EPA, 1991)

Pesticide common name	Uses	Comments
Organophosphates		
Acephate	Lawns, turf, ornamentals	Cholinesterase inhibitor; possible human carcinogen
Chlorpyrifos	Lawns, ornamentals, termines, ants and roaches, ticks and chiggers control	Cholinesterase inibitor; substitute for chlordane; applied in granular form outdoors, as aerosols and emulsifiable concentrates indoors, dietary exposure exceeds guidelines
Diazinon	Lawns; turf; ornamentals; indoor in no-pest strips, flea collars	Cholinesterase inibitor; one of the most widely used chemicals in consumer pesticides; applied as aerosols and in granular form
Dichlorvos (DDVP)	Indoors and garden	Cholinesterase inhibitor; possible human carcinogen; protective clothing must be worn during use; primarily used in "no-pest strips" by consumers to kill flying insects
Disulfoton	Fruits and vegetables, potted house plants, ornamentals	Cholinesterase inhibitor: 2% formulations require certified applicator
Malathion	Ornamental trees and shrubs, orchards, pet products, household insecticides	Low toxicity cholinesterase inhibitor; cancer studies are not adequate and will be redone; widely used in home products; one of GAO's top 10 home chemicals; banned by Florida in 1986 for community mosquito control
Naled	Pet flea collars, mosquito control, orchards and vegetables	Metabolizes to dichlorvos
Pyrethroids		
Allethrin	House and garden insecticides, pet products	Synthetic pyrethrin of low toxicity; widely used by consumers and professionals to control household pests; also used by professionals as a termiticide
Permethrin	Same as above	Synthetic pyrethrin; possible human carcinogen
Phenothrin	Same as above	Synthetic pyrethrin

(continued)

TABLE 2.16 (*continuation*)

Pesticide common name	Uses	Comments
Resmethrin	Same as above	Synthetic pyrethrin
Tetramethrin	Same as above	Synthetic pyrethrin; possible human carcinogen
Rotenone	Fruit and vegetable gardens, pet products, human lice and chigger dusts, fish poison	Human exposure has not been measured; widely used; viewed as comparatively safe but not fully tested; natural botanical derivative
Pyrethrins	House and garden products, pet products, anti-lice shampoos	Viewed as comparatively safe but not fully tested; natural botanical derivative
Carbamates		
Bendiocarb	Ant, roach, and flea control indoors; ornamentals; insecticide; shelfpaper; turf	Cholinesterase inhibitor; widely used by professionals for control of indoor pests; applied as a dust or wettable powder; consumer exposure occurs almost entirely from home uses; EPA recommends application of ready-to-use products by professionals only
Carbaryl	Fruit and vegetable gardens; turf; pet products; flea collars and dust; ornamentals; indoor use	Low toxicity cholinesterase inhibitor; commonly used insecticide for control of pests indoors and outdoors; applied to leaf surfaces as wettable powder and dust; should not be used on pregnant dogs because it may cause birth defects in dogs
Propoxur	Indoor use; ant and roach killers;pet products; mosquito foggers	Probable human carcinogen; cholinesterase inhibitor; toxic residue for weeks after application; infants crawling on treated surfaces may be exposed dermally; widely used
Chlorinated Hydrocarbons		
Chlordane	Termite control	Probable human carcinogen, cancelled in 1988; product may still be stored because shelf supplies were allowed to be depleted; most widely used termiticide before withdrawal; applied as a liquid poured or injected into soil around the building foundation

(*continued*)

TABLE 2.16 (*continuation*)

Pesticide common name	Uses	Comments
Dicofol	Vegetable gardens, indoor insect and mite control	Possible human carcinogen; home use is not large; DDT and related compounds may be present as contaminants
Lindane (HCH,BHC)	House and garden use; shelf paper; anti-lice shampoos; pet products; termite control	Possible/probable human carcinogen; use against termites (house) is restricted to unoccupied buildings; exposure from Kwell shampoo can be high if used too often or left on skin too long
Methoprene	Cockroach, mosquito, and flea control; control of mealy bugs and spider mites on house plants	Considered to be quite safe; chronic data gaps
Synergists		
MGK 264	House use; pet products	Inhalation and contact exposure could be significant; widely used
Piperonyl butoxide	House and garden use; pet products	Widely used; direct inhalation is an important exposure route

Several pesticides possess high or moderate acute toxicity to mammalian species, man included. Acute intoxication may result from accidental or inadvertent exposure when proper handling procedures are not followed, or from attempted suicides.

Besides acute effects from high-dose exposures, a large body of animal data supports the possibility of long-term health effects following prolonged exposure to low doses of pesticides. However, the relevance of animal data to man is limited and epidemiological confirmation on humans is available for only some of the effects (Maroni and Fait, 1993). Most of the published epidemiological studies on long-term health effects in man relates to the occurrence of cancer, while non-cancer health effects have been less frequently investigated. The major reported health effects in humans from prolonged exposure to selected pesticides are given in Table 2.17. Moreover, allergic sensitization dermatoses have been related to a number of pesticides and chronic health effects have been reported for other compounds, but further confirmation is needed.

A large number of epidemiological studies have investigated the possible association between exposure to pesticides and specific cancers. The types of cancer studied and the class of pesticides in possible connection with them are included in Table 2.17.

Poisonings

During 1987, poison control centres in the USA reported 57 430 cases of pesticide exposure and 98% of these were due to accidental exposures. Insecticides accounted for about 66% of the total cases, followed by rodenticides (17%), moth repellents (7.7%), herbicides (7.2%), and fungicides (2.3). About 60% of the cases involved children under 6 years of age (Blondell, 1989).

During the period 1980–1985, at least 46.5% of the accidental pesticide related deaths in the USA occurred in the home (40.9% of the locations were not specified). Organophosphate insecticides were responsible for about 32% of the deaths. Seventeen percent of the victims were under the age of 5; 29.6% were between 25 and 44 years of age; 23.9% were between 45 and 64 years of age; and 22% were over the age of 65 (Blondell, 1989).

About 400 cases of acute poisonings are annually reported to the Italian poison control centre (CAV) in Milan, 16% are accounted to occupational origin, 18% to attempted suicides and 65% to accidental poisonings. Within the occupational cases, insecticides accounted for about 50% of the total cases, followed by herbicides (31.8%) and fungicides (18.2%).

Inert ingredients may be more toxic than some active ingredients. However, inert ingredients are considered to be proprietary, and generally are not required to be identified by the product label. An exception are inerts which EPA classifies as being of "immediate toxicological concern" (a list of chemicals including carbon tetrachloride, formaldehyde, methylene chloride, and others). Pesticides with these chemicals must either be reformulated or have a label which states: "This product contains the toxic inert ingredient...".

2.5 POLYNUCLEAR AROMATIC HYDROCARBONS

2.5.1 Physico-chemical nature

Polynuclear (or polycyclic) aromatic hydrocarbons (PAHs) are a large group of organic compounds with two or more benzene rings. They have a relatively low solubility in water but are highly lipophilic.

PAHs in indoor air are partially in the vapour phase and partially adsorbed on particles, mostly on the respirable fraction (Van Vaeck and Van Cauwenberghe, 1985; Miguel and Friedlander, 1978). Following the WHO classification

TABLE 2.17

Health effects in humans from prolonged exposure to selected pesticides (Maroni and Fait, 1993)

Pesticide/classes	Evidence is well established	Evidence requires further confirmation
Phenoxyherbicides		
2,4,5-T, 2,4-D, MCPA and related compounds (TCP, TCDD)	Chloracne (TCDD)	Cancer: soft-tissue sarcoma; lymphatic and haematopoietic system; stomach; colon; prostate
		Teratogenesis
Other herbicides		
Triazines	Lung cancer	Ovarian cancer
Arsenicals	Liver disease	
Halogenated hydrocarbons		
Dichloropropene		Myelolymphoproliferate disorders
PCP		Aplastic anaemia
DBCP	Spermatogenesis suppresion	
Methylbromide		Mild neurotoxic effects
EDB	Sperm abnormalities	
Carbamates		
Carbaryl	Chromosome aberrations	
	Sperm abnormalities	
Organochlorine insecticides		
Chlordane/heptachlor		Myelolymphoproliferate disorders
		Brain cancer
Hexachlorobenzene	Porphyria	Liver cancer
DDT	Chloracne	Chromosome aberrations
		High cholesterol and triglyceride levels
		Tremors, muscular weakness
		Neurotoxic effects
Synthetic pyrethroids	Reversible paresthesia ("skin sensations")	
Organophosphorus esters	Delayed neuropathy*	Chromosome aberrations
		Central nervous system alterations
		Non-Hodgkin's lymphoma
Copper sulfate		Liver disease

*Some compounds only, after subacute or acute exposure only.

(cf. Table 2.1), naphthalene belongs to the VOC category whereas anthracene, phenanthrene, fluoranthene, and pyrene belong to the SVOC category. They are together with naphthalene the most abundant PAHs in air. Because of their distribution between the vapour and particle phase, PAHs are usually sampled from air using a combination of a particle filter and a sorbent such as polyurethane foam (PUF) or XAD-2.

There are several hundred PAHs; benzo(a)anthracene, quinoline, chrysene, benzofluoranthene isomers, benzo(a)pyrene, indeno(1,2,3-c,d)pyrene, 1-nitropyrene, and 2-nitrofluoranthene are of particular importance because of their carcinogenicity or mutagenicity. Benzo(a)pyrene has often been used as an indicator of the carcinogenic potential of an environmental mixture of PAHs.

2.5.2 Occurrence and sources

PAHs and their analogues and derivatives (nitro-PAHs, oxygenated PAHs) are ubiquitous in the atmosphere. They are generally produced by combustion of fossil fuels and by industrial processes. In homes and non-industrial buildings, the major sources are expected to be infiltration of outdoor air and indoor combustion sources such as cigarettes, unvented space-heaters, gas stoves, wood stoves, and fireplaces. Major sources of PAHs in outdoor air include combustion of gasoline and diesel fuels for transportation and combustion of oil, wood and coal for space heating and power production. Agricultural burning and forest fires can also be significant sources of PAHs for limited periods of time in certain areas. The industrial processes which are sources of PAHs in outdoor air include coke production, petroleum refining, and steel production.

PAH compounds are also present in foods (smoked, broiled, refined) and water; in fact the oral intake of PAH compounds may be much higher than the amount inhaled in the general population (WHO, 1987).

Some PAHs are characterized for specific source emissions such as cyclopenta(c,d)pyrene, benzo(g,h,i)perylene, and coronene for vehicle exhaust or quinoline and isoquinoline for tobacco smoke.

2.5.3 Typical concentrations and exposures

About 500 PAHs have been detected in the air (Herlan, 1977), but most measurements have been made on benzo(a)pyrene (BaP). The relation between the amount of BaP and some other PAHs is termed the "PAH profile". Although the PAH profiles of emissions can differ greatly, they are relatively similar in the ambient air of several towns or cities. The different PAH profiles of emissions appear mixed, producing a relatively uniform PAH profile in the

air, and what is most important is that these relations seem to be independent of the PAH concentration in the air.

The natural background level of BaP (excluding forest fires) may be almost zero. There is evidence that outdoor concentrations of BaP in large cities of the United Kingdom such as London have fallen to only a few percent of former values over the past 30 years, as a result of controls on smoke emissions and the virtual disappearance of the open coal fire for domestic heating purposes (Waller, 1982). This development is probably mainly due to modifications in heating systems and in the kind of heating fuel used. Coal burning in small domestic open fires or stoves produces amounts of BaP that are several orders of magnitude higher than those emitted by oil-fired central heating systems (Ahland et al., 1985).

Indoor concentrations of selected PAHs have been determined in 33 homes in California and Ohio (Wilson et al., 1991). The ranges and typical values are shown in Table 2.18. Each column in the table gives the average of the individual PAH concentrations in all rooms (kitchen, living room, and bed-

TABLE 2.18

Typical concentrations in homes (ng/m^3)[a]

Compound	Smoking	Non-smoking	
	Gas stove	Electric stove	Gas stove
Quinoline	240	3.1	2.8
Phenanthrene	87	19	31
Fluoranthene	8.6	2.99	3.7
Pyrene	5.2	2.13	2.8
Cyclopenta(c,d)pyrene	1.1	0.17	0.21
Benz(a)anthracene	1.4	0.20	0.46
Chrysene	2.6	0.37	0.67
Benzofluoranthenes	2.5	0.47	0.93
Benzo(a)pyrene	1.2	0.28	0.63
Indeno(1,2,3-c,d)pyrene	0.97	0.32	0.82
Benzo(g,h,i)perylene	1.4	0.53	0.88
Coronene	0.60	0.52	1.3
1-Nitropyrene	0.044	0.020	0.021
2-Nitrofluoranthene	0.052	0.012	0.022

a Concentrations in selected individual homes giving approximately median concentrations for 33 homes investigated; concentrations are averaged over three locations in the homes: living room, bedroom, and kitchen.

Taken from Wilson et al., 1991 (Munksgaard International Publishers Ltd., Copenhagen, Denmark).

room). Indoor concentrations were, with rare exceptions, higher than those outdoors and were associated with indoor combustion sources. In homes where smoking took place, its effects dominated the results with typical increases in PAH concentrations over non-smokers' homes by a factor of three or more. The other identified indoor residential sources were wood smoke, natural gas appliances and vehicle exhaust.

Under special conditions PAHs can increase to very high concentrations indoors. Benzo(a)pyrene levels of 6 $\mu g/m^3$ were found in houses without chimneys in southern China (National Centre for Preventive Medicine, 1984). In India the BaP exposure averaged about 4 $\mu g/m^3$ while cooking with biomass fuels (Smith et al., 1983).

2.5.4 Health effects

On the basis of experimental results, toxic effects other than carcinogenicity are not to be expected. In the past, chimney sweeps and tar-workers were dermally exposed to substantial amounts of PAHs and there is sufficient evidence to show that skin cancer in many of these workers was caused by PAHs (IARC, 1985). Epidemiological studies in coke-oven workers, coal-gas workers and employees in aluminium production plants provide sufficient evidence of the role of inhaled PAHs in the induction of lung cancer (Kawahata, 1938; Doll et al., 1972; Hammond et al., 1976; Redmond, 1976; IARC, 1984). In the extensive study by Redmond (1976), an excessively high rate of lung cancer mortality was found in coke-oven workers. Increases in lung cancer cases correlate closely with the time spent working on the top of ovens where an average BaP concentration of about 30 $\mu g/m^3$ has been detected (Björseth, 1979; Blome, 1981).

Because not all PAHs are carcinogenic, a suitable index for carcinogenic potential of the fraction of PAHs in the air is needed. If all PAH profiles were identical, the concentration of a single PAH would be a good index of the carcinogenic potential of the total PAH fraction. The PAH profiles of different emissions are far from this ideal, but, as stated, the variations of PAH profiles in workplaces are not so wide and the deviation from the mean is relatively low in ambient air. Therefore, as 5–15% of the total carcinogenic effect from PAH fractions of different exhaust condensates is due to BaP according to skin-painting studies, BaP may be provisionally regarded as a sufficiently informative indicator for the carcinogenic potential of the PAH fraction in ambient air. As mentioned, the PAH profiles detected in different emissions and workplaces sometimes differ widely from each other and from PAH profiles in the ambient air. Moreover, it cannot be excluded that PAH profiles in the ambient air vary under special conditions. Considerably more data are

necessary in order to develop a precise index for the carcinogenic potential of all PAH profiles which can occur under conditions relevant for lung cancer risk estimates. Furthermore, the carcinogenicity of PAH mixtures may be influenced by the synergistic effects of other components emitted together with PAH during incomplete combustion.

Benzo(a)pyrene is metabolized to approximately 20 primary and secondary oxidized metabolites and to a variety of conjugates. Several metabolites can induce mutations, transform cells and/or bind to cellular macromolecules.

PAHs can induce the synthesis of the enzymes responsible for their own metabolism. However, the additional effects of various exogenous and endogenous inducers and inhibitors of metabolism (genetic factors, age, sex and nutritional status) which have been shown to influence these enzyme systems, make it difficult to predict whether the carcinogenicity of PAH will be increased or decreased.

2.6 POLYCHLORINATED BIPHENYLS

2.6.1 Physico-chemical nature

The polychlorinated biphenyls (PCBs) are a class of chlorinated, aromatic compounds which have found widespread applications due to their outstanding physico-chemical characteristics, which are thermal stability, resistance to oxidation (non flammability), and to acids, bases, and other chemical agents as well as their excellent dielectric (electrically insulating) properties.

PCBs are prepared industrially by the catalytic chlorination of biphenyl with anhydrous chlorine. The resulting product is a complex mixture of chlorobiphenyls with a different number of chlorine atoms per molecule. This fact is the reason for the physical state of PCB mixtures produced; most individual chlorobiphenyls are solids at room temperature, whereas commercial mixtures are oily fluids or sticky resins due to the mutual depression of melting points of their components.

The solubility of PCBs in water is low and decreases with increasing chlorine content. Similar to the solubility data, the vapour pressures of PCB mixtures may be influenced by the components of lower chlorine content which are generally more volatile. Vaporization rates of several PCB mixtures are shown in Table 2.19 as a function of their weight percent of chlorine (Hutzinger et al., 1974).

No major components other than chlorobiphenyls have been reported to be present in PCB preparations. Impurities known to be present in commercial PCBs are chlorinated dibenzofurans (PCDFs) and chlorinated naphthalenes.

TABLE 2.19

Vaporization rates of PCB mixtures

Weight % of chlorine	Vaporization rate at 100°C ($\mu g/cm^2/h$)
21	1740
32	874
42	338
48	152
54	53
62	13

Adapted from Hutzinger et al. (1974) with permission.

PCDFs, which are of considerable toxicological significance, are formed by the pyrolysis of PCBs in the presence of O_2.

2.6.2 Occurrence and sources

PCBs have exclusively anthropic origins i.e. they do not occur in nature. A common source for indoor pollution by PCBs are defective capacitors of luminous discharge lamps. During the inspection of an office building one-third of the ballasts examined have been found to have leaks and it has been estimated that of all ballasts that have leaked or otherwise failed 10% involve a capacitor rupture (Petreas et al. 1990).

The valuable properties of PCBs as plasticizers has led to their use as sealants in building construction (so-called open PCB systems); examples are surface treatment for textiles, adhesive for waterproof wall coatings, paints, printing inks and sealant putties. Since 1973 the manufacture, sales, and use of PCBs have been restricted in the 24 member countries of the Organisation for Economic Co-operation and Development (OECD). Nevertheless there might have been widespread use in older buildings. Although PCBs have low volatility, there may be an appreciable loss to the atmosphere during the lifetime of a PCB-plasticized resin, whereby adsorption on dust particles or surfaces causes these compounds to settle down.

High concentrations of PCBs in surface wipe samples collected from ventilation ducts indicated HVAC systems to be possible sources of PCBs in air. In particular the lower chlorinated products present on surfaces or particles within segments of the HVAC systems may be selectively volatilized and carried into the air flowing past them, depending on temperature and pressure conditions within the system (Petreas et al., 1990).

Because of their insecticidal and fungistatic activity, PCBs have been used in the USA in pesticide formulations. The volatilization into the atmosphere from incineration of industrial and municipal waste causes outdoor air to be a secondary PCB source although, by analogy to DDT, it might be expected that PCBs entering the atmosphere in the vapour phase would be adsorbed rapidly onto particles, which would be deposited or washed out in rain.

2.6.3 Typical concentrations and exposures

Results from the U.S. Environmental Protection Agency indicate an outdoor air concentration range of PCBs between 1 and 50 ng/m^3 (Panel on Hazardous Trace Substances, 1972), and similar results have been reported from Japan (Tatsukawa and Watanabe, 1972).

Extensive studies of indoor air concentration levels have been performed in the Federal Republic of Germany over the last few years. Among these, relatively high PCB concentrations were encountered in concomitance with PCB containing sealants used in building construction over the last few decades.

In several rooms of a large school building (year of construction 1971) indoor concentrations of 1000 ng PCB/m^3 and more were registered (Burkhardt et al., 1990). Qualitative analysis of different materials, i.e. capacitor fluids, gave evidence about the origin of the PCB emission. Air samples were found to contain PCB mixtures corresponding to the commercial product Clophen A 50, having a higher chlorine content than that used for capacitor fluids. PCB concentrations ranging from 12.4 to 32.7 weight % were finally found in the sealing products which were used in the whole building. The presence of polychlorinated dioxins or furans, known to be present as contaminants in commercial PCB products, was investigated in one of the highly exposed rooms and found to be limited to 0.189 pg TCDD equivalents per m^3.

During investigations conducted in 100 German buildings, aimed at producing data about PCB indoor concentrations originating from PCB-containing sealing products, the emission rate was found to depend on the different technical PCB-products used and on atmospheric conditions, beside the PCB content in the product itself (Balfanz et al., 1991). Table 2.20 shows the maximum concentrations found in sealing materials and indoor air as a function of the PCB-products most commonly used in Germany.

Repeated measurements carried out at comparable temperatures during summer and winter in 10 rooms gave evidence about the temperature-dependence of PCB indoor concentrations. As shown in Table 2.21, winter values were always found to be lower than summer values.

TABLE 2.20

PCB indoor concentration ranges observed in German buildings as a function of the PCB product used in sealing materials; samples were collected under comparable conditions (Balfanz et al. 1991)

Clophen-type used in sealing material	Mean chlorine no. per molecule	PCB weight % in sealing material	PCB indoor concentration (ng/m^3)
A 40	4	max. 21	ca. 200–6000
A 50	5	max. 35	ca. 200–2500
A 60	6	max. 47	max. 550

TABLE 2.21

Seasonal fluctuations of PCB indoor concentrations observed in 10 rooms (Balfanz et al. 1991)

Room no.	PCB-concentrations (ng/m^3)	
	Summer	Winter
1	1460/1730	925
2	1685	1400/1055
3	1835	330/465/315
4	1560	1110/1180
5	1360	495/455
6	1140	495
7	486	60
8	1090	505
9	1120	510
10	690	250

2.6.4 Health effects

Several cases of chloracne, hyperpigmentation, gastrointestinal disturbances, elevated serum enzyme, metabolism abnormalities, and numbness of extremities have been reported among people highly exposed to PCBs (WHO, 1993).

There is suggestive evidence of association between the increased incidence of cancer and exposure to PCBs (Brown and Jones, 1981; WHO, 1993) and to PCBs containing significant amounts of polychlorinated dibenzo-*p*-furans

(PCDFs) (Urabe et al., 1979; Kuratsune, 1976).

PCBs are well absorbed by mammals through the gastrointestinal tract, lungs, and skin. They are mainly stored in adipose tissue, there is some placental transfer and they appear unchanged in human milk.

The most bioaccumulating PCB congeners have five to seven chlorine atoms per molecule. The more highly chlorinated congeners are generally less available to the organisms both because they are more tightly bound with soils and sediments and because they are usually present in lower quantities in the environment. Congeners with less chlorination are more readily metabolized and eliminated and so do not tend to bioaccumulate as highly.

Although there are 209 possible PCB configurations (congeners), perhaps half that number account for nearly all of the environmental contamination attributable to PCBs. Still fewer congeners are both prevalent and either demonstrably or potentially toxic (McFarland and Clarke, 1989).

A few PCB congeners that are sterically similar to coplanar 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) are directly toxic (McFarland and Clarke, 1989).

Other PCB congeners, as well as those that are directly toxic, may also be involved in toxicity indirectly by stimulating the production of bioactivating enzyme systems. For example, it has been demonstrated that PCBs have the ability to induce aryl hydrocarbon metabolizing mixed-function oxidases, the group of microsomal cytochrome P-450-dependent enzyme systems that catalyze oxidative biotransformations of aromatic ring-containing compounds. A result could be an increased capacity for bioactivation of otherwise nontoxic foreign compounds such as certain polynuclear aromatic hydrocarbons (PAHs) to cytotoxic or genotoxic metabolites (McFarland and Clarke, 1989). Although mixed-function oxidase (MFO) induction is not a proximate cause, it strongly correlates with certain kinds of toxicities.

Structural patterns can thus be used to discriminate among PCB congeners on the basis of toxic potential, if not entirely on toxicity *per se*. Congeners that demonstrate 3-methylcholanthrene-type (3-MC-type) and mixed-type MFO induction have the greatest toxic potential. These congeners most closely resemble 2,3,7,8-TCDD in their structures and in their toxic effects. The larger group of phenobarbital-type (PB-type) inducers have considerably less potential for contributing to toxic effects. Weak inducers and noninducing congeners have the least potential for toxicity (McFarland and Clarke, 1989).

Polychlorinated dibenzo-*p*-furans (PCDFs) are found mostly as contaminants of PCBs and can be formed by the incomplete combustion of PCBs. Since PCDFs are generally as toxic as the corresponding PCDDs, which are considered to be potent carcinogens, their presence as an impurity can be of great toxicological significance (Matanoski et al., 1986).

Levels of PCBs in the tissues of human subjects were investigated in several studies involving occupationally exposed subjects and the general population.

A study of worker populations from two plants that manufacture capacitors showed that PCB mixtures that were part of the dielectric fluid in the capacitors, contained significant levels of coplanar PCBs. PCDFs were also found as contaminants in capacitor fluid samples at very low levels. The investigations conducted on 200 workers considered to be highly exposed, have not demonstrated acute (i.e., chloracne) or chronic toxicity, although there was some evidence of induction of microsomal enzymes (Lawton et al., 1985b; Lawton et al., 1986). Two additional results were that the half-life of the PCBs in exposed workers was slowing with time and was 6–12 years at the time of the last measurement; higher chlorinated congeners appeared to be retained in the body preferentially and have extremely long half-lives (Brown, 1984; Lawton et al., 1985a; Brown et al., 1989; Kamrin and Fisher, 1991).

Extensive studies involving workers exposed to PCBs were performed in the Washington DC area (Emmet at al., 1988) and in northern Italy (Maroni et al., 1981). Concentrations of individual PCBs were determined in both serum and adipose tissue of currently and previously exposed transformer repair workers and comparison workers never occupationally exposed. The studies comprised investigations about urinary elimination of porphyrins giving evidence about porphyrinogenesis and enzyme induction in workers exposed to PCB (Maroni et al., 1984).

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Indoor Air Quality
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Chapter 3

Physical Pollutants[1]

3.1 PARTICULATE MATTER

3.1.1Physico-chemical nature

Airborne particulate matter (PM) represents a complex mixture of organic and inorganic substances. Mass and composition can be divided into two principal groups: coarse particles larger than 2.5 μm in aerodynamic diameter (D_{ae}), and fine particles smaller than 2.5 μm in aerodynamic diameter. The smaller particles contain the secondarily formed aerosols (gas to particle conversion), combustion particles and recondensed organic and metal vapours; the larger particles usually contain earth crustal materials and fugitive dust from roads and industries.

Because of the complexity of particulate matter and the importance of particle size in determining exposure, multiple terms are used to describe particulate matter. Some terms are derived from and defined by sampling methods, e.g. suspended particulate matter, total suspended particulates, black smoke. Other terms refer more to the site of deposition in the respiratory tract, e.g. inhalable, thoracic particles that deposit primarily in the lower respiratory tract below the larynx. Other terms, such as PM_{10} (particulate matter with an aerodynamic diameter of 10 μm), have both physiological and sampling components.

Chemical composition is less definable due to interactions among particles and other pollutants, although photochemical oxidation plays a negligible role

1 A part of the text of this chapter has been derived from World Health Organization (WHO), 1987 "Air Quality Guidelines for Europe", WHO Regional Publications, European Series No. 23, WHO Regional Office for Europe, Copenhagen; U.S. Environmental Protection Agency (EPA), 1987 "Preliminary Indoor Air Pollution Information Assessment", Appendix A, EPA-600/8-87/014, pp. 2–18, 19, Office of Health and Environmental Assessment Washington DC; U.S. Environmental Protection Agency (EPA), 1991 "Introduction to indoor air quality", EPA/400/3-91/003; World Health Organization (WHO), 1988 "Man-made Mineral Fibres", Environmental Health Criteria No. 77, Geneva, pp. 23; ECA (European Concerted Action "Indoor Air Quality and its Impact on Man", COST Project 613), 1994. Radon in indoor air, Report No. 15. Luxembourg: Office for Official Publications of the European Communities.

in transforming chemicals indoors. Some particles are highly reactive (e.g., acidic or basic) with other pollutants or with biological systems and indoor materials. Condensations from particle–particle, or gas–particle interactions do still occur. While the relationship between chemical composition and particle size has been relatively well characterized for ambient air, characterization of indoor particles is less well defined (U.S. EPA, 1982a).

In homes where wood is burned, respirable particulates which include polynuclear (or polycyclic) aromatic hydrocarbons (PAH) compounds, trace metals, nitrates, and sulfates have been measured.

PAHs are of particular concern because of their carcinogenic potential. PAH compounds include a large number of organic molecules which contain two or more benzene rings. These compounds are produced as the result of incomplete combustion. They are only very slightly soluble in water, but are very soluble in fat. Although fat soluble, these compounds are metabolized rapidly in the body and do not tend to bioaccumulate in the fatty tissues. It is thought that the carcinogenic effect of PAHs is due to diol-epoxides which are metabolites of PAH compounds in the body (WHO, 1987).

Once PAH compounds enter the air, they can be adsorbed on particles and inhaled into the lungs. PAH compounds are also present in foods (smoked, broiled, refined) and water. In fact, the oral intake of PAH compounds may be much higher than the inhaled amount in the general population (WHO, 1987). For more information on PAH refer to Chapter 2, Section 2.5.

3.1.2 Occurrence and sources

Combustion sources (combustion appliances and tobacco smoking) are probably the chief indoor generators of fine-mode particles which contain a host of organic and inorganic material about which little is known. Sprays and cooking aerosols may also contribute to the total fraction of fine-mode particles. Biological contaminants, including viruses, bacteria, fungal spores and fragments, pollens, fragments of house dust-mite faeces, and dried, reentrained animal secretions (e.g., urine, saliva) and animal dander, may also be found primarily in the fine-mode fraction. Coarse-mode fractions which may consist largely of material carried in from outdoors such as dusts, or larger biological fragments such as mould parts or insect fragments, and which may settle on floors and carpeting and be reentrained through human activity, have not been well-characterized.

It has been found that the indoor/outdoor (I/O) ratios are close to unity for inhalable particulate matter (IPM; $D_{ae} \leq 2.5$ μm) or respirable particulate matter (RPM; 2.5 μm $\leq D_{ae} \leq$ 10 μm) when no smokers are present. In contrast, I/O ratios are generally less than unity when total suspended particulate matter

TABLE 3.1

Emission rates for particulate matter and particulate-bound materials (mg/h); taken from U.S.Department of Energy (1985).

Source	Appliance type	Emission rate	
		Particles	Benzo(a)pyrene
Kerosene space heaters	Radiant	0.13–0.16	–[a]
	Convective	<0.03–0.034	–[a]
Gas space heaters		0.21–3.23	–[a]
Wood heaters		2.6	$1.4 \cdot 10^{-5}$
			$3.5 \cdot 10^{-3}$
Gas appliances	Range (1 burner)	1.9–30	–[a]
	Oven	0.118–0.126	–[a]

a Not detected.

is involved due to the filtering action of the dwelling's shell (Cohen and Cohen, 1980; Yocom et al., 1982; Yamanaka and Maruoka, 1984).

Particulate emissions from wood stoves and fireplaces vary widely, depending on the design and operation of the unit. In addition, these combustion devices were found to contribute significant quantities of PAHs (Moschandreas et al., 1981; Neulicht and Core, 1982; Hytonen et al., 1983). In general, airtight wood stoves and catalytic wood stoves contributed less pollution than the non-airtight units.

A summary table of emission rates for particles from indoor combustion sources has been published by the U.S. Department of Energy (1985) and is presented in Table 3.1. As can be seen, kerosene space heaters have the lowest emission rates among the sources shown and wood heaters have the highest emission rates.

In addition to the sources shown in Table 3.1, tobacco smoke can be a significant source of indoor particles. Particles in tobacco smoke are especially hazardous due to their chemical composition and because they are inhalable (0.1–1.0 μm) and remain airborne for hours after smoking stops. The synergistic effect between tobacco smoke, radon decay products and asbestos, respectively, has been demonstrated.

3.1.3 Typical concentrations and exposures

Early data concerned with human exposure to indoor particles were more related to pesticides adsorbed on particles than to the particles themselves

(Starr et al., 1974). Fugaš (1975) used indoor monitoring to measure lead. This work showed some of the relationships of I/O concentrations in European towns. Binder et al. (1976) found that particle uptake appeared to be caused more by exposure to indoor-, rather than outdoor pollutants.

At the same time, evidence began to emerge concerning the indoor pollutant load caused by cigarette smoking. Although the earliest work was more concerned with CO, particulate matter (PM) from tobacco smoking quickly became an important area of scientific interest. Dockery and Spengler (1977), Repace and Lowrey (1980), Repace (1981), Bock (1982), Girman et al., (1982), and many others demonstrated the widespread effect of smoking on particle concentration.

Measurement techniques routinely used in reported studies are unable to discriminate between the combustion particles and the other household particles generated from human activities and intrusion from the outside air. The particle mass collected is an heterogeneous mixture of material, with biological properties ranging from inert to highly toxic and carcinogenic. Thus, a mere measure of mass concentration in $\mu g/m^3$ has little direct relationship to health effects. Further evidence shows that much of the IPM in an indoor microenvironment is generated by human activity (e.g., particle resuspension when walking on carpets, shedding of hair and skin detritus, cooking, vacuuming, etc.) so that an average taken over a 24-hour period will smooth out the periods of high human exposure with the periods of quiescent air when no one is home.

Additional complications exist because of low resolution in time (24-hour averages), and seasonal effects which are known to introduce trends in the data due to variations in activity patterns and air exchange (air conditioning versus heating).

Spengler et al. (1985) showed that personal 24-hour IPM exposures are consistently higher than the time-weighted average concentration predicted by 24-hour indoor and outdoor values from stationary monitors. Table 3.2 gives the range of indoor and outdoor IPM concentrations and the comparative personal exposure of the people living in the homes under study. It is interesting to note that at most percentiles the personal exposure exceeds the indoor microenvironment concentration.

By conventional measures, the quality of ambient air has steadily improved over the years. However, although outdoor total suspended particles (TSP) concentrations may have decreased, human exposure to inhalable particles (IPM) has probably increased. As most people spend much of their time indoors, they will be proportionally more subjected to any elevated levels of pollutants from indoor sources. Modern studies tend to assume that central monitoring stations do not reflect or predict actual personal exposures. Continued research is needed on the relationship between measured concentrations and actual exposure.

TABLE 3.2

Quantile descriptors of personal, indoor, and outdoor IPM concentrations, by location

City	IPM sample		IPM Quantile, $\mu g/m^3$					Mean	SE
	Group	N	95%	75%	50%	25%	5%		
Kingston	personal	133	99	47	34	26	19	42	2.5
	indoor	138	110	47	31	20	10	42	3.5
	outdoor	40	28	22	16	12	6	17	2.7
Harriman	personal	93	122	54	35	24	15	47	4.8
	indoor	106	129	45	27	18	10	42	4.1
	outdoor	21	34	23	15	13	9	18	4.0
Total[a]	personal	249	113	48	34	26	17	44	2.8
	indoor	266	119	46	29	20	10	42	2.6
	outdoor	71	33	23	17	13	7	18	2.1

a Includes samples from 13 subjects living outside Kingston and Harriman, Tennessee town limits and from four field personnel.
Taken from: Spengler et al. (1985).

3.1.4 Health effects

The most extensive data on the health effects from exposure to particles are derived from epidemiological studies of outdoor air. In these studies, the exposure assessment was limited primarily to measurement of the mass of particulate matter (PM); only rarely was the mass characterized chemically. Additional supporting data from animal toxicological and human clinical studies are chemical-specific. Careful evaluation of the existing data base indicates that PM causes adverse health effects. However, relating this entire data base to the indoor situation is not scientifically possible, in as much as for the most part specific chemicals in PM have not been characterized indoors or outdoors.

However, there are chemical-specific elements of the PM data that can be useful in assessing the potential effects of exposure to indoor PM. Examples of useful data elements include: ETS (discussed in a separate chapter), soot, particle-bound PAHs, and other combustion particles.

The respiratory system is the major route of intake of particles, and its structure and function, along with the physical and aerodynamic properties of the particles, determine where and how particles are deposited, retained, or cleared. Breathing patterns, as they relate to route and ventilation level, greatly influence where the deposition of inhaled particles occurs.

The aerodynamic diameter (D_{ae}) has generally been characterized into two modes: the fine mode (< 2.5 μm) and the coarse mode (> 2.5 μm to ca. 15 μm, or greater). These distinctions relate to particle deposition, with fine-mode and small coarse-mode particles depositing principally in the thoracic (tracheo-bronchial and pulmonary) region and larger coarse-mode particles depositing primarily in the extrathoracic (nasopharyngeal) region during nose-breathing. Oronasal breathing associated with minute ventilations exceeding 0.35 l/min can significantly alter deposition patterns.

Irritant effects from inhaled particles may result in increased airway constriction, altered mucociliary transport, and changes in alveolar macrophage activity. These effects apply across a wide range of inhaled particles, acting alone, or in concert with common gaseous air pollutants, such as SO_2, NO_x, or ozone. Other toxic effects are more chemical-specific and, depending on the nature of the chemical, may include organs outside the respiratory tract.

Bronchioconstriction, arising from chemical and/or mechanical stimulation of irritant neural receptors in the bronchi, has been reported as a response to short-term exposure to high levels of various inert dusts as well as to acid and alkaline aerosols. Neurological receptors tend to concentrate near airway bifurcations where particle deposition is greatest so that stimulation may result in pulmonary reflexes such as bronchioconstriction and coughing. These reflex actions may be related to effects observed on various epidemiological studies such as aggravation of chronic respiratory disease states including asthma, bronchitis, and emphysema. Individuals with asthma or emphysema and other respiratory diseases may have increased particle deposition due to altered breathing patterns or airway structural changes, which may then contribute in a cascading effect to even more bronchioconstriction and particle deposition (U.S. EPA, 1986b).

It should be emphasized that the health effects postulated for PM are generally the result of epidemiologic studies that correlate health effects observations in a population and ambient air PM levels. The studies do not specifically involve measurement of indoor particles and, hence, do not assess effects specifically related to indoor air pollution. Furthermore, extrapolation of health effects from epidemiologic studies of ambient particle pollution should be approached with great caution because sampling methods for particles outdoors may not be directly comparable with the methods developed for indoor particle sampling.

Particles studied indoors have mainly been those in the fine-mode, primarily resulting from cigarette smoking, or as emission from combustion appliances. Coarse-mode particles resulting from reentrainment of fibres, dust, house dust mite faecal pellets, animal and human dander, mould spores and fragments, probably constitute the second most common form of indoor parti-

cle pollution. Methods for routine sampling of many of these types of particles have not been developed, and their health impact is, for the most part, scarcely unknown.

Health effects associated with exposure to soot

Virtually every combustion process in which carbonaceous fuels are burned results in the production of soot. Soot often consists of clusters of very small (ca. 30 nm) spheroids fused together to form particles with a 0.1–0.2 μm diameter. These soot particles contain a solvent extractable fraction and an elemental carbon fraction. The combustion of diesel fuel, coal, fuel oil, and wood has been shown to produce soot particles with extractable organic matter that is mutagenic in short-term bioassays and, in several of these sources, has been shown to be tumorigenic in animals. Chemical characterization of these organics shows that they contain carcinogenic PAHs, methylated PAHs, nitrated PAHs, and oxidized PAHs.

In addition occupational studies have shown increased risk of lung, larynx, and skin cancer after exposure to coal soot, and increased incidence of scrotum and bladder cancer after exposure to coal tar in gashouse workers, stokers, asphalt, coal tar and pitch workers, coke-oven workers, and chimney sweeps (Cole and Goldman, 1975). Nonoccupation-related lung cancer has been linked to the exposure of indoor unvented coal combustion emissions in a study conducted in a rural county, Xuan Wei, located in Yunnan province in China (Mumford et al., 1987).

Polycyclic aromatic hydrocarbons

Polycyclic aromatic hydrocarbons (PAHs) represent a diverse group of combustion products whose common characteristic is a nucleus of a different number of carbon rings in which interlinked rings have at least two carbon atoms in common. The number of rings varies from two to many. Configuration of ring structures determines physical and chemical properties and biological activities.

The PAHs are of specific human health concern because many of them are procarcinogenic, cocarcinogenic, or carcinogenic, and they affect the immune and cardiovascular systems. They are breathed into the lung as volatiles or they can adsorb to the surface of particles and be inhaled. For more information on PAH, the reader is referred to Chapter 2, Section 2.5.

Beyond the specific issues discussed above, one of the concerns for the potential adverse health effects of PM is related to the organics bound to the

particles. The particles and bound organics have properties that influence deposition, retention, and bioavailability. Hence, the specific chemical-physical characteristics of the particles and organics have major influence on the health effects. One of the most classical examples is that benzo(a)pyrene (BaP) alone has low carcinogenic potency; ferric oxide alone is not a carcinogen. When BaP is coprecipitated with ferric oxide and instilled into the lungs of animals, cancer results. Many particles adsorb organics as they are created in combustion processes. However, once within the air, they can adsorb other materials (i.e., radon, pesticides, other organics from home products). These absorbed materials will have different pulmonary deposition sites and deposition efficiencies when adsorbed on a particle, as opposed to existing in vapour phase. Retention and pharmacokinetics would also be affected. Thus, even an inert particle could contribute to health risks from indoor air. Assessment of the risks of individual pollutants does not take into account such complex interactions. Risk assessment on complex mixtures must be done if a clear picture of the health effects of indoor pollutants is to emerge.

3.2 ASBESTOS

3.2.1 Physico-chemical nature

Asbestos is a generic term that applies to a group of impure hydrated silicate minerals which occur in various fibrous forms, which are incombustible and separable into filaments. Asbestos fibres belong to the mineral groups of amphibole (such as amosite, crocidolite, tremolite, anthophyllite, and actinolite) and serpentine (chrysotile). Chrysolite, which is very commonly found if a product is asbestos-containing, is a magnesium silicate whose fibres are strong and flexible, and its longer fibres can be spun into thread for weaving. It is the most widely used form of asbestos. Amphibole asbestos includes various silicates of magnesium, iron, calcium, and sodium. Its fibres are generally brittler and cannot be spun, but it is more resistant to heat than chrysotile asbestos.

Chrysotile fibres consist of aggregates of long, thin, flexible fibrils that resemble scrolls or cylinders of uniform chemical composition. Although chrysolite is a reasonably well defined mineral, the five amphibole asbestiform fibres have such variable chemical compositions and physical properties that positive identification is sometimes troublesome.

The macroscopic asbestos fibres are actually bundles of thinner fibres made up of fibrils which, in the case of chrysolite, have a diameter of 20–25 nm. Each macroscopic fibre is highly anisotropic and tends to decompose into its thinner

constituents under industrial handling or from weathering, giving rise to a fibrous, partially respirable aerosol.

3.2.2 Sources and occurrence

Natural sources are important, because asbestos minerals are widely spread throughout the earth's crust and are not restricted to the few mineable deposits. In particular, chrysolite is present in most serpentine rock formations. Emissions are due to natural weathering and can be enhanced by man's activities such as quarrying or street building. Very little is known, however, about the amounts emitted from natural sources.

Man-made emissions originate from activities in the following categories:

(a) mining and milling;
(b) manufacture of products;
(c) construction activities;
(d) transport and use of asbestos-containing products; and
(e) disposal.

There has been steep rise in the production and use of asbestos in the last 100 years. Asbestos consumption has levelled off in recent years (WHO, 1986a) to about 4 million tonnes (1983) and, because of the relative decline in crocidolite and amosite usage (WHO, 1986a), the figure represents essentially chrysolite production; in 1982 only about 5% of the total asbestos produced was in the form of amphibole asbestos (crocidolite and amosite) (Organization for Economic Cooperation and Development, OECD, 1984).

In many industrialized countries most of the asbestos used was in the building sector (70–90% in several western European countries) (OECD, 1984) and the demand for asbestos in these countries had already reached a constant level before the health effects of asbestos were widely debated. Because of its specific technical properties, asbestos has found an extremely large variety of applications (in about 3000 different products). Currently, legislative restrictions and the success in finding substitutes for asbestos in fibre-cement, brake-linings, insulation and many other applications are leading to declining asbestos consumption in the above-mentioned countries.

Asbestos emissions occur during processing. When air filtration is used, the dust emissions from processing can be kept below 100 g per tonne (OECD, 1984). Rain acidity (from carbon dioxide and air pollutants) is known to corrode asbestos-cement sheets, constituting a further source of emission (Meyer, 1984). Brake-linings in cars are another source in the urban environment. Only a few measurements have been made of the contribution made by these sources to fibre emissions in urban air (Fischer and Meyer, 1983; Marfels et al., 1984).

Indoor asbestos fibre concentrations can be considerably higher than outdoor concentrations (Nicholson et al., 1975). Indoor asbestos dust originates from insulation material sprayed on steelwork or ceilings (such material may become highly friable after some years), asbestos plasters, low-weight insulation plates, etc. (Nicholson et al., 1975; Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario, 1984). Sometimes such materials have been used in direct or close contact with air-conditioning equipment. Even though some of these materials, such as spray asbestos, are no longer used, they are still found in many public buildings. Until the mid-1970s electric storage-heaters and some other electrical household equipment contained asbestos. One of the main forms of use of asbestos is as asbestos-cement; in this case the release of fibres to the general environment is minimized, since the fibres are essentially "locked" in the cement matrix. Asbestos cement products, therefore, do not usually pose problems for indoor air quality.

Factors such as renovation and repair, maintenance, external vibrations and vandalism can considerably increase the emission of asbestos dust from existing indoor sources (NRC, 1984; Sawyer, 1979). Increased emission is also possible as a result of changes in temperature and reduction of humidity.

Unlike levels of asbestos fibres in ambient air, fibre concentrations in the air of indoor environments can always be related to their source, thus offering the possibility of modifying or removing the source of emission. Because people in temperate climates live indoors most of the time, the most relevant source of inhalation exposure will often be asbestos-containing material in buildings.

Asbestos fibres normally constitute only a relatively small fraction of the total fibrous aerosol in ambient air (Lohrer, 1983; Marfels et al., 1984). The biologically more important so-called "critical" fibres are those equal to or longer than 5mm and having diameters up to 3μm (Sawyer, 1979; NRC, 1984; WHO, 1986a) with a ratio equal to or greater than 3:1 between the diameters.

Asbestos fibres of respirable size form part of a range of fibrous aerosols in the lower atmosphere. Other fibres include man-made mineral fibres, fibrous silica and aluminium oxide, fibrous gypsum and, in some geographical areas, fibrous zeolite, attapulgite, sepiolite and wollastonite.

3.2.3 Typical concentration and exposure

Although asbestos fibres can be readily detected and monitored in occupational situations by using phase-contrast optical microscopy, their assessment in the environment calls for an integrated method capable of microchemical analysis of single fibres, measurement of fibre length and diameter, and counts of fibre numbers in given air samples. Electron microscopy is the only method

which can accurately detect and identify asbestos fibres among the very wide range of other fibrous and nonfibrous particles. In order to positively identify asbestos fibres an X-ray diffraction analyser is attached to an electron microscope. Such instrumentation is costly and highly-trained personnel are required to obtain reliable results. Although integrated electron microscopy methods have been developed during the last 15 years, they are still not in widespread use. Therefore, the number of asbestos measurements remains limited. However, if one compares various sets of such data and restricts quantification to orders of magnitude, the following pattern of concentration emerges[1]. All concentrations are expressed as fibres per m^3 air, although concentrations in terms of fibres per litre and fibres per ml are often reported. Some studies have been carried out where results have been expressed as ng or μg per m^3 air. Because only results such as fibre number concentrations are considered to be directly relevant to an index of exposure with regard to possible health implications (Commins, 1985; WHO, 1986a) and factors in attempts to convert mass to number concentrations are very variable (Commins, 1985), mass concentrations are not considered here.

Rural areas (remote from asbestos emission sources):

- below 100 F/m^3 (Marfels et al., 1984; NRC, 1984)

Urban areas:

- general levels may vary from below 100 to more than 1000 F/m^3 (Marfels et al., 1984; Commins, 1985)

Near various emission sources the following figures have been measured as yearly averages (OECD, 1984; Marfels et al., 1984)

- downwind from an asbestos-cement plant at 300 m: 2200 F/m^3; at 700 m: 800 F/m^3; at 1000 m: 600 F/m^3 (Marfels et al., 1984);
- at a street crossing with heavy traffic, 900 F/m^3 (Sawyer, 1979);
- on an express-way, up to 3300 F/m^3 (Report of the Royal Commission on Matters of Health and Safety arising from the use of Asbestos in Ontario, 1984).

Indoor air:

- in buildings without specific asbestos sources, concentrations are generally below 1000 F/m^3 (Commins, 1985);
- in buildings with friable asbestos, concentrations vary irregularly; usually less than 1000 F^*/m^3 are found, but in some cases exposure reaches 10000 F^*/m^3 (Report of the Royal Commission on Matters of Health and Safety arising from the use of Asbestos in Ontario, 1984), where F^* = fibres counted with an optical microscope.

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1 If not otherwise stated, concentrations in the following text are given as numbers of critical asbestos fibres per m^3, i.e.all fibres of length $L > 5$ μm, diameter $D < 3$ μm and aspect ratio L:D > 3:1 measured by electron microscopy methods.)

Workplace air:

- occupational levels are orders of magnitude higher than those found in the environment, with values from 10^5 to more than 10^8 F^*/m^3 (Nicholson, 1981; Commins, 1985) but are now being reduced to below 2×10^8 F^*/m^3 in most countries and to (0.2–0.5) x 10^8 F^*/m^3 in some.

Various subgroups of the population are exposed to different fibre concentrations for varying lengths of time. It is usually assumed that the risks of any two persons are roughly comparable — other factors being equal — if their accumulated fibre burdens are the same. The fibre burden is the accumulated number of critical fibres; it can be calculated as [fibre concentration (F/m^3)] ·[number of years exposure] · [air volume inhaled each year at place of exposure (m^3/year)]. In this context it is important to note that not all fibres inhaled are deposited and available for retention in the body. However, it is assumed that the fraction of fibres exhaled is more or less the same during all inhalation exposures. Assuming a breathing rate of 10 m^3 for an 8-hour working day and 200 working days per year, a worker inhales 2000 m^3 per year during working hours, while total inhalation for the general population is 7300 m^3 per year (NRC, 1984).

A calculation of indoor concentration has been made in terms of average fibre concentration in USA. The median values range from 400 F/m^3 (NRC, 1984) to 500 F/m^3 (Commins, 1985). If these estimates were correct, they would result in a lifetime fibre burden of up to 2×10^8 F. In this case outdoor exposure would be of minor importance to most of the US population.

There can also be more irregular exposure of the general public, with peak concentrations at specific sites.

Drinking water and food

Drinking water and food may contain asbestos fibres from natural sources (e.g. rock) and man-made sources (e.g. asbestos-cement pipes). The total fibre content (fibres of all lengths) in drinking water can vary from 10^4 F/litre to more than 10^8 F/litre (Umweltbundesamt, 1980; Commins, 1983; Meyer, 1984; Toft et al., 1984).

In several studies, rats and other laboratory animals were exposed to asbestos in diet or drinking-water. Only a single — usually very high — dose was applied in each investigation. The results are inconclusive, if not negative (Toft et al., 1984; WHO, 1986a).

Inhalation is by far the most important route of exposure of humans to critical fibres, the amount of uptake by ingestion being questionable and, at the least, significantly lower.

3.2.4 Health effects

The most serious health effects from asbestos exposure are lung cancer and mesothelioma (cancer of tissue of mesothelial origin), and these have been established as the most important causes of death from asbestos exposure. While the relationship of occupational exposure to asbestos with these cancers has been established from occupational/epidemiologic studies, the occurrence of asbestosis has been documented more extensively than that of asbestos-related malignancies.

Asbestosis is a slowly developing fibrosis of the lung caused by the inhalation of high concentrations of asbestos dust and/or long exposure (Weicksel and Ulrich, 1984). Its severity depends both on the length of time since onset of exposure and on the intensity of the latter (Doll and Peto, 1985). Although nonmalignant in itself, advanced asbestosis is often associated with lung cancer, especially among smokers. Hypoxia with cor pulmonale does occur in severe cases, while mild forms may not necessarily be associated with marked disablement (NRC, 1984).

Mesothelioma is a malignant tumor of the pleura or peritoneum (Report of the Royal Commission on Matters of Health and Safety arising from the use of Asbestos in Ontario, 1984; Doll and Peto, 1985). It is a rare type of cancer (less than 0.04% of all deaths in the general population of the USA) (NRC, 1984). A higher incidence of mesothelioma has nearly always been related to the inhalation of mineral fibres, and in the majority of cases to occupational asbestos exposure (Report of the Royal Commission on Matters of Health and Safety arising from the use of Asbestos in Ontario, 1984). Causing few symptoms initially, mesothelioma is incurable when diagnosed (NRC, 1984).

The time elapsing between first exposure to asbestos and the clinical manifestation of tumours ranges from 20 to 50 years for mesothelioma in the populations of workers studied (Doll and Peto, 1985). Dose–response relationships are derived from retrospective epidemiological studies of exposure data relating to situations several decades ago. Clearly these exposure data can only be approximations (Nicholson, 1981; Report of the Royal Commission on Matters of Health and Safety arising from the use of Asbestos in Ontario, 1984). Increased incidence rates seen in nonoccupationally exposed people living in the same household as asbestos workers or in the vicinity of strong asbestos emission sources have been attributed to this exposure (Hain, 1984).

Lung cancer (bronchial carcinoma) is the most frequent kind of cancer in the male population (accounting for about 10% of all male deaths in many industrialized countries) and is obviously related to external factors such as smoking, ionizing radiation (e.g. radon) and occupational exposures to certain substances, including asbestos, in the latter case even without co-existing

asbestosis (Report of the Royal Commission on Matters of Health and Safety arising from the use of Asbestos in Ontario, 1984). Many studies have shown that smokers have a higher risk of developing lung cancer than nonsmokers when exposed to asbestos (Doll and Peto, 1985).

Clarifying the relationship between asbestos exposure and lung cancer is a complicated task. Both the synergism of smoking and asbestos inhalation and the high level of lung cancer in the general population (background exposure) must be taken into account. The synergistic effect can be described by a multiplicative model (Friedberg and Ulmer, 1983) which is used below in connection with the extrapolation of lung cancer risk. The risk of lung cancer seems to rise consistently from mining and milling operations, through branches such as asbestos-cement production, to asbestos textile and insulation work, according to the increasing portion of more dangerously shaped fibres in these processes.

Recent evaluations (Report of the Royal Commission on Matters of Health and Safety arising from the use of Asbestos in Ontario, 1984; NRC, 1984; Toft et al., 1984; Doll and Peto, 1985) of epidemiological occupational studies provide little evidence for the induction of other cancers, including gastrointestinal cancers, although recently it has been suggested that laryngeal cancer may be related to heavy occupational exposure to asbestos (Doll and Peto, 1985). Clearly the risk, if any, for the general population from "other" cancers is very small (Aurand and Kierski, 1981; Report of the Royal Commission on Matters of Health and Safety arising from the use of Asbestos in Ontario, 1984; Toft et al., 1984).

Extrapolations to evaluate the risk of asbestos cancers from occupational circumstances have been made, although numerical estimates in a specific exposure circumstance have a large (approximately tenfold) uncertainty. Because of this uncertainty, calculations of unit risk values for asbestos at the low concentrations measured in the environment must be viewed with caution.

3.3 MAN-MADE MINERAL FIBRES

3.3.1 Physico-chemical nature

The man-made mineral fibres (MMMF) discussed in this document will be restricted largely to a subset known as man-made vitreous fibres (MMVF), which are fibres manufactured from glass, natural rock, or other minerals. They are classified according to their source material. Slag wool, rock wool, and glass wool or filaments are produced from slag, natural rock, and glass, respectively (Ottery et al., 1984). While naturally occurring fibres are crystalline

TABLE 3.3

Some synonyms and trade names of MMMF products

Term/name	Category	Remarks
TEL	GW	
Fibreglass insulation	GW	Fiberglas®R is a trade name
Boron silicate glass fibre	GW	Most glass wools are of borosilicate type
Saint Gobain	GW	Major insulation producer(TEL process)
GF/D Whatman filter	GF	Filters made from glass fibres
GF/C Microfilter	GF	Filters made from glass fibres
Rock wool	RW	Rockwool®R is a trade name
Pyrex glass fibres	GF	Pyrex® is a glass with high chemical resistance
Basalt wool	RW	Mineral wool
Man-made mineral insulation fibres		Slag or rock wool (USA) Glass, slag, or rock wool (Europe)
Insulation wool		
Refractory fibres	CF	
Fibrous ceramic aluminium silicate glass	CF	
Fiberfrax®	CF	
Ceramic wool	CF	
Owens-Corning Beta®	GF	
Calcium silicate	CF	
Calcium-alumino-silicate	CF	
Refractory ceramic fibre	CF	
Alumina and zirconia fibre	CF	
Zirconia	CF	
Fireline ceramic	CF	
Nextel® ceramic fibre	CF	

GW = glass wool; GF = glass fibre other than wool; RW = rock wool; CF = ceramic fibre. Taken from WHO (1988).

in structure, most man-made mineral fibres are amorphous silicates. The amorphous networks of MMMF are composed of oxides of silicon, boron, and aluminium, oxides of the alkaline earth and alkali metals, oxides of bivalent iron and manganese, or amphoteric oxides (e.g., Al_2O_3, Fe_2O_3). Common trade names are given in Table 3.3. The terminology currently used is not consistent or well defined. Categories and materials are named with reference to use,

structure, raw material, or process. The distinctions are not always clear, and categories many overlap.

In North America, slag wool and rock wool are often referred to as "mineral wool"; in Europe and Asia, the term "mineral wool" also includes glass wool. A "wool" is an entangled mass of fibres in contrast to the more ordered fibres in continuous filament (textile) glass.

MMMF products usually contain a binder and an oil for dust suppression. Textile fibres may contain a sizing agent for lubrification. Unlike some natural fibres, MMMF do not split longitudinally into fibrils of smaller diameter, though they may break transversely into shorter fragments. Consequently, the diameters of fibres to which workers may be exposed will depend on the diameter as manufactured, but the length of such fibres will vary according to the extent to which they have been broken subsequently.

3.3.2 Occurrence and sources

MMMF are used widely as asbestos substitutes. The global production of man-made mineral fibres has been estimated to be 4.5 million tonnes in 1973 and 6 million tonnes in 1985. Fibrous glass accounts for approximately 80%, mainly used in acoustic or thermal insulation. Textile grades (5–10% of fibrous glass production) are used principally for the reinforcement of resinous materials and in textiles, such as draperies. Less than 1% of the production of glass fibre is in the form of fine fibres used in speciality applications, such as high efficiency filter paper and insulation for aircraft (WHO, 1988). Mineral wool (rock wool/slag wool), which accounts for approximately 10–15% of MMMF production in the USA, is used mainly in acoustic and thermal insulation. In Europe, glass wool and rock wool are produced in approximately equal volumes and are also used for thermal and acoustic insulation. Refractory fibres (1–2% of all MMMF) are used for high temperature application (WHO, 1988).

Data on emission of MMMF from manufacturing facilities are limited. Fibre levels in emissions from fibrous glass and mineral wool plants in Germany, determined by scanning electron microscopy (SEM), were of the order of 10^4 F/m^3. On the basis of these data, it was estimated that the total fibrous dust emission from MMMF plants in this country were about 1.8 tonnes/year. Emissions of respirable fibres (defined by the investigators as fibres with lengths exceeding 10 μm and diameters of less than 1 μm) were estimated to be 80 kg/year.

It seems likely that the main source of emissions of MMMF (mainly glass fibres) in indoor air is insulation in public buildings or homes. Although quantitative data are not available, emissions are probably highest shortly after installation or following disturbance of the insulation (NRC, 1984).

3.3.3 Typical concentrations and exposures

As a general rule, levels of MMMF in the occupational environment have been determined by phase contrast optical microscopy. Average concentrations during the current manufacture of fibrous glass insulation range from 1 to 5×10^4 F/m^3.

In general, airborne fibre concentrations during the installation of products containing MMMF are comparable to, or less than, levels found in production (1 fibre/ml). Exceptions occur during blowing or spraying operations conducted in confined spaces, such as during the insulation of aircraft or attics, when mean levels of fibrous glass and mineral wool have ranged up to 2×10^6 F/m^3 and 4×10^6 F/m^3 respectively.

Some data are available on the levels of MMMF in ambient and indoor air. Largely varying concentrations of MMMF (from 2 to 10000 F/m^3) have been found in ambient air. In indoor air mean values between 50 and 500 F/m^3 with maximum values of up to several thousand F/m^3 during installation of MMF insulation have been reported.

On the basis of analysis using phase contrast optical microscopy (PCOM) (Balzer et al., 1971; Esmen et al., 1980) and calculation of the "glass" content of collected particulate (Cholak and Schafer, 1971), it has generally been concluded that the contribution of fibrous-glass-lined air transmission systems to the fibre content of indoor air is insignificant.

3.3.4 Health effects

The effects of MMMF on the skin were recognized as early as the turn of the century. Fibrous glass and rock wool fibres (mainly those greater than 4.5–5 μm in diameter) cause mechanical irritation of the skin characterized by a fine, punctate, itching erythema, which often disappears with continued exposure.

Data concerning the incidence or prevalence of dermatitis in workers exposed to MMMF are scarce. There are also few reliable data concerning the tolerance of workers to MMMF induced dermatitis. The irritant dermatitis induced by MMMF may be complicated by an urticarial and eczematous reaction that sometimes mimics an allergic response, both clinically and histologically. In addition, allergic reactions to resins used in MMMF production occasionally occur.

Until recently, reports of eye irritation in populations exposed occupationally to MMMF were restricted to a few isolated cases in the early literature.

The epidemiological studies of non-malignant respiratory disease in populations exposed occupationally to MMMF are mainly of two types: cross-sectional investigations of the prevalence of signs and symptoms of respiratory

disease and historical prospective studies of mortality due to non-malignant respiratory disease.

The results of the few studies published are summarized in WHO (1988).

While carcinogenic effects have been observed under conditions of high occupational exposure, no such effects have been found at medium and lower level of exposure; therefore a remarkable risk is not expected under non-occupational circumstances.

3.4 RADON

3.4.1 Physico-chemical nature

Radon (Rn) is a radioactive noble gas which exists in several isotopic forms. Only two of these isotopes occur in significant concentration in the general environment: radon-222 (usually referred to as 'radon'), a member of the radioactive decay chain of uranium-238, and radon-220 (often referred to as 'thoron'), from the decay chain of thorium-232 (see Fig. 3.1a). Radon is the first and only gaseous and inert element of the radioactive chains, so that it can easily leave the place of production (soil, rock and building material) and enter the indoor air. The contribution made by thoron to the human exposures in indoor environments is usually small compared with that made to radon, due to the much shorter half-life (55 s vs. 3.8 days), and it will only occasionally be referred to here.

As shown in the decay scheme in Figs. 3.1a and 3.1b, the gas radon decays to produce a series of decay or daughter products. From a health perspective the daughter products of most significance are the four short-lived ones — polonium-218 to polonium-214 inclusive — which are referred to in various ways: radon daughters, radon progeny, radon decay products. These elements, unlike radon, shortly after their formation attach themselves to aerosol particles; only a small fraction of them remains in unattached form, depending on aerosol size and concentration and on ventilation (Nazaroff and Nero, 1988). When radon and its short-lived decay products are inhaled the radiation dose to lung tissue is dominated by the alpha particles emitted by the deposited decay products, which cause, especially those ones attached to small size aerosols or in unattached form, damage to sensitive lung cells, thereby increasing the probability of cancer developing. The dose to the lung due to the beta and gamma radiation emitted by the decay products is comparatively negligible for two reasons: (a) the energy of these radiations are much smaller than the energy of alpha particles; (b) these types of radiation interact with matter less strongly than alpha radiation and therefore they release only a small part of their energy to the lung. It has to be underlined that the contribution to lung

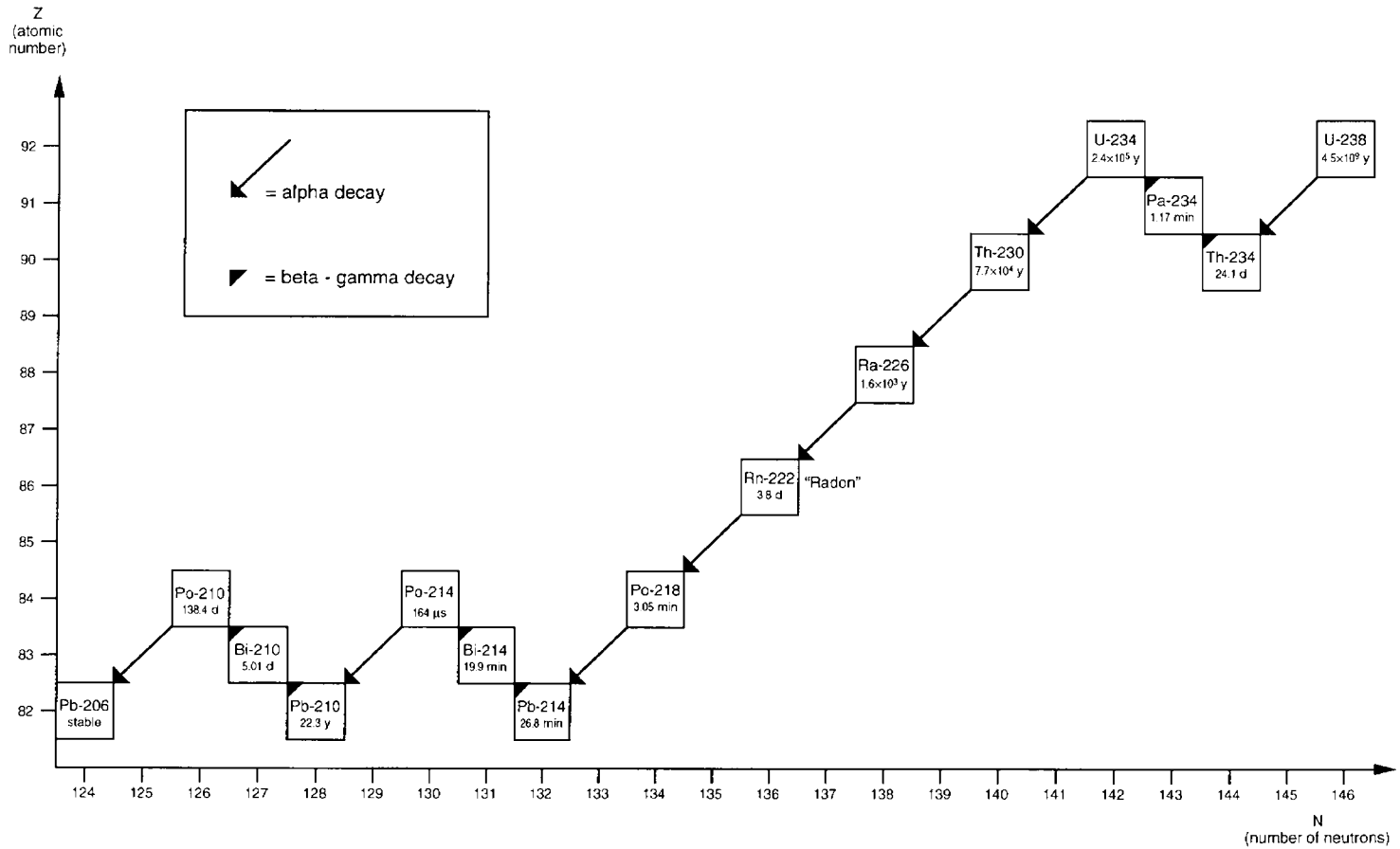

Fig. 3.1a. Radioactive decay chains of (*this page*) uranium-238 and (*next page*) thorium-232. Under the symbol of each isotope the half-life time is reported.

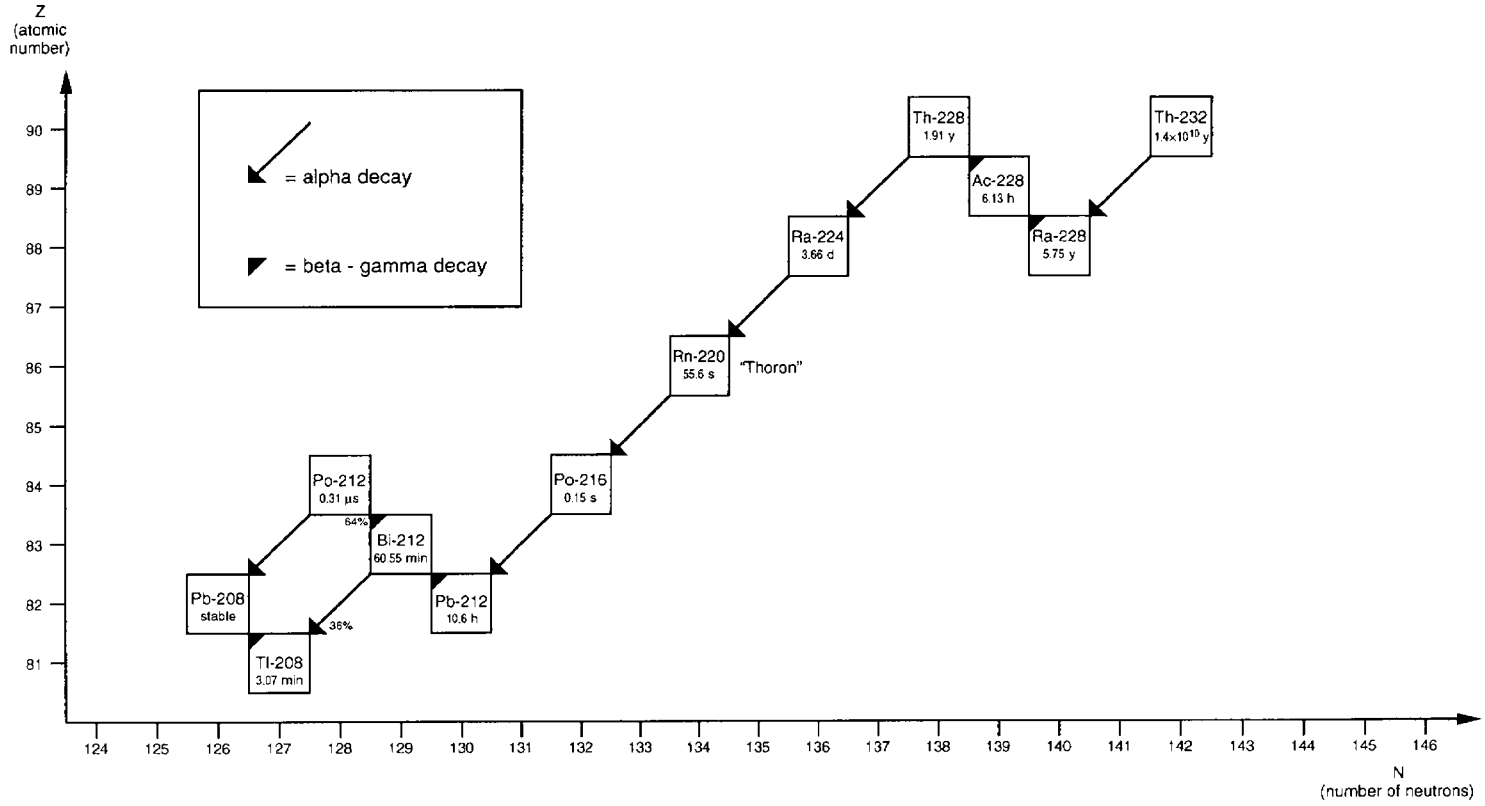

Fig. 3.1a. Continued.Caption on previous page.

Radionuclide	Historical name	Half Life	Energies (MeV) and intensities of the principal radiation emitted		
			Alpha	Beta	Gamma
^{222}Rn	Emanation Radon	3.82 d	5.49 (100%)	–	0.51 (0.07%)
↓ α					
^{218}Po	Radium A	3.05 min	**6.00** (~100%)	–	–
↓ α					
^{214}Pb	Radium B	26.8 min	–	0.65 (50%) 0.71 (40%) 0.98 (6%)	0.295 (19%) 0.352 (36%)
↓ β,γ					
^{214}Bi	Radium C	19.7 min	5.45 (0.012%) 5.51 (0.008%)	1.0 (23%) 1.51 (40%) 3.26 (19%)	0.609 (47%) 1.12 (17%) 1.76 (17%)
↓ β,γ					
^{214}Po	Radium C'	164 μs	**7.69** (100%)	–	–
↓ α					

Fig. 3.1b. Decay scheme of radon-222 and its short-lived decay products. The energy of the most important emitted radiations are indicated in bold. Insignificant branch decays from Po-218 and Po-214 are neglected.

dose arising from the radon gas itself is small in comparison as very little radon is absorbed by lung tissue because it is an inert gas and, for the same reason, unlike its decay products it cannot be adsorbed onto lung airway surfaces. The principal role of radon is to carry itself and therefore its short-lived decay products from soil and building material to the indoor air and finally to the lung. The dose to the lung due to all the decay products following polonium-214 is comparatively negligible because of the very long decay time of lead-210 (22.3 years). For the deposited decay products, which are short-lived, mostly absorbed and therefore decay in the lung tissue, the concept of Potential Alpha Energy (PAE) is used (see also the following section). PAE is the total alpha energy emitted by the decay of each decay product along the decay chain down to lead-210. For example an atom of deposited polonium-218 has a PAE of 13.7 MeV (i.e. 6.00 + 7.68) while even beta emitters such as

lead-214 and bismuth-214 each have a PEA of 7.68 MeV as they ultimately give rise to the alpha decay of polonium-214.

3.4.1.1 Special quantities and units for radon and radon decay products

Absorbed dose

It is defined by $D = d\varepsilon/dm$, where $d\varepsilon$ is the mean energy imparted to a mass dm of matter by ionising radiation. For radiation protection purposes, a tissue — or organ — average absorbed dose D_T is defined by $D_T = \varepsilon_T / m_T$, where ε_T is the total energy imparted to a tissue — or organ — of mass m_T.

The SI unit of absorbed dose is the Gray (Gy), equal to one Joule per kilogram (J/kg).

1 Gy = 100 rad (old unit).

Equivalent dose

The equivalent dose is the product of the absorbed dose and the weighting factor for a specific type of radiation. The weighting factor w_R accounts for the different ability of the different type of radiation to cause biological damage. For beta particles, gamma rays and X rays w_R is taken as unity, for alpha particles as 20, for neutrons as from 5 to 20, depending on energy (ICRP, 1991).

The SI unit of equivalent dose is the Sievert (Sv), equal to one Joule per kilogram.

1 Sv = 100 rem (old unit).

Effective dose

This is the sum of the products obtained by multiplying the equivalents doses to various organs and tissues by the appropriate risk weighting factor w_T for each. This quantity is considered to be proportional to the total probability of stochastic effects.

Its SI unit is the Sievert (Sv), as for equivalent dose.

1 Sv = 100 rem (old unit).

The most recent estimation of the risk of stochastic effects for general population, expressed as probability of fatal cancer per unit of effective dose, is 5×10^{-2} Sv^{-1} (ICRP, 1991).

Activity

The activity of a radioactive source is defined by $A = dN/dt$, where dN is the number of decays in time dt.

The SI unit of activity is the Becquerel (Bq), equal to one nuclear transformation per second.

1 Curie (Ci) (old unit) = 3.7×10^{10} Bq.

Activity concentration

It is the activity per unit of volume or mass, whose SI unit is the Becquerel per cubic meter (Bq/m^3) or the Becquerel per kilogram (Bq/kg).

1 pCi/l (old unit) = 37 Bq/m^3.

Radon progeny concentration

The concentrations of the four short-lived radon daughters are usually expressed in terms of Potential Alpha Energy Concentration (PAEC) or Equilibrium Equivalent Radon Concentration (EERC or EER). These two quantities combine the four concentrations in a single value, using weighting factors that take into account the relative importance for the health effects to the lung, which are due to the alpha particles emitted by the radon daughters along the decay chain down to lead-210.

The *PAEC* of any mixture of radon daughters is the sum of the potential alpha energy (i.e. the total energy of all alpha particles emitted by a radon daughter atom in its chain decay to the Pb-210) of all the short-lived radon daughter atoms present per unit of air volume. Its SI unit is the Joules per cubic meter (J/m^3), but the old Working Level (WL) unit is still widely used, representing a PAEC of 1.3×10^5 MeV per litre.

1 WL = 2.08×10^{-5} J/m^3.

The *EERC* is the fictitious activity concentration of radon in equilibrium with its daughters (i.e. all daughters having the same concentration as radon; actually there is always a certain degree of disequilibrium) giving the same PAEC of the actual non-equilibrium mixture of radon daughters. Its SI unit is the Becquerel per cubic meter (Bq/m^3).

A PAEC of 1 WL corresponds to $\approx$ 3700 Bq/m^3 (= 100 pCi/l) of EERC.

Equilibrium factor

The ratio of EERC to actual radon concentration is called 'equilibrium factor'. It is usually represented by the symbol 'F'. Due to the actual non-equilibrium conditions its value in indoor air is usually in the range 0.2–0.7.

Cumulated exposure to radon progeny

Its practical measurement unit is the Working Level Month (WLM). It corresponds to an exposure to $\cong$ 3700 Bq/m^3 of radon progeny concentration in equilibrium with radon gas (i.e., all progeny having the same concentration as radon; actually there is always a certain degree of disequilibrium), during the working period of one month (170 hours).

This unit is used historically for occupational exposure in mines, while for indoor exposure of the general public in dwellings it is preferable to express the cumulative exposure in terms of mean radon gas concentration during a

one-year period, because this is the quantity that is usually measured in dwellings. The following conversion formula can be used:

$$1 \text{ WLM} \cong 72/(F \times IOF) \text{ Bq y/m}^3$$

where F is the equilibrium factor between radon and its progeny, and IOF is the indoor occupancy factor, i.e. the fraction of time spent indoors. Using F = 0.5 and IOF = 0.8, a cumulated exposure of 1 WLM corresponds to an exposure to $\cong$ 180 Bq/m^3 of radon gas concentration during the period of 1 year, or 90 Bq/m^3 for a period of 2 years, and so on. If F = 0.4 is assumed, 1 WLM $\cong$ 220 Bq y/m^3.

3.4.2 Occurrence and sources

The main source of indoor radon is its immediate parent radium-226 in the ground of the site and in the building materials (Nero, 1988, 1989). The outdoor air also contributes to the radon concentration indoors, via the ventilation air. Tap-water and the domestic gas supply are usually radon sources of minor importance, with a few exceptions.

In most situations it appears that elevated indoor radon levels originate from radon in the underlying rocks and soils (e.g. Castrén et al., 1985). This radon may enter living spaces in dwellings by diffusion or pressure driven flow if suitable pathways between the soil and living spaces are present (see Fig. 3.2). It should be noted, however, that in a minority of cases elevated indoor radon levels may arise due to the use of building materials containing high levels of radium-226. Examples of such materials, used in some buildings, are by-product gypsum, alum shale and volcanic tuffs.

The United Nation Scientific Committee on the Effects of Atomic Radiations (UNSCEAR) has made a very simple model to try to estimate the relative contribution of these sources: for a 'typical' house, with a radon concentration of 50 Bq/m^3 at ground floor, the contributions of soil, building materials and outdoor air are, respectively, ~60%, ~20% and ~20%, while for the upper floors in high-rise buildings, where the radon concentration is estimated to be 'typically' 20 Bq/m^3, these values become ~0%, ~50% and ~50% (UNSCEAR, 1993).

Soil

For those who live close to the ground, e.g., in detached houses or on the ground floor of apartment buildings without cellars, the most important radon source is radium in the ground. The radium concentration in soil usually lies in the range 10–50 Bq/kg, but it can reach values of hundreds Bq/kg, with an estimated average of 40 Bq/kg (UNSCEAR, 1993). Typical radon concentra-

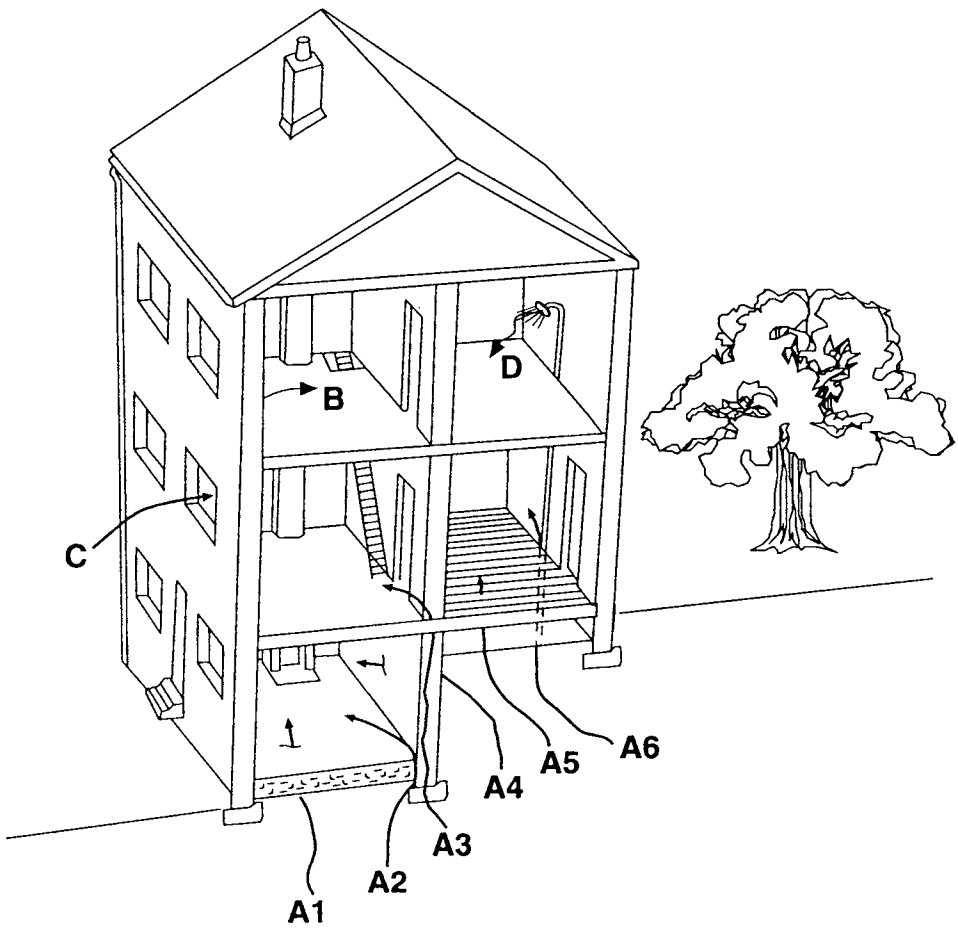

Fig. 3.2. Typical radon sources and entry routes. (A) Entry points of radon from soil (A1: Cracks in solid floors; A2: Construction joints; A3: Cracks and cavities in walls; A4: Cracks in walls below ground level; A5: Gaps in suspended floors; A6: Gaps around service pipes). (B) Radon exhalation from building materials. (C) Entry of radon with outdoor air. (D) Radon released from water.

tions in soil gas range from 10 000 Bq/m^3 to 50 000 Bq/m^3. The potential for radon entry from the ground depends mainly on the activity level of radium-226 in the subsoil and its permeability with regard to air flow. Example of terrains with a high radon potential are alum shales, some granites and volcanic rocks, due to high concentrations of radium-226 and the presence of eskers (gravel, sand and rounded stone deposited from subglacial streams during the ice ages), all these being characterised by high permeability. The ground could also be contaminated with waste tailings from uranium or phosphate mining operations with enhanced activity levels (e.g. Tyson et al.,

1993). The ingress of radon from the soil is predominantly one of pressure-driven flow, with diffusion playing a minor role (de Meijer et al., 1992). The magnitude of the inflow varies with several parameters, the most important being the air pressure difference between soil air and indoor air, the tightness of the surfaces in contact with the soil on the site, and the radon exhalation rate of the underlying soil. If there is no airtight layer between the basement and the ground, the underpressure indoors causes radon to be drawn in from the ground under the building. Underpressure occurs in most houses if either the adjustment of inlet and outlet of air in forced ventilation systems or the outdoor air supply for vented combustion appliances is inappropriate. The underpressure may be considerable for all types of ventilation systems when the inlet air is restricted too much. The tightness of the structures in contact with the ground varies with different building regulations and techniques, and is very dependent on cracks, openings and joints. Structures are hardly ever so airtight that radon inflow is completely prevented. For example, to get a radon daughter concentration of less than 100 Bq/m^3 EER (see Section 3.4.1.1) in a house with a volume of 500 m^3 and a ventilation rate of 0.5 air changes per hour, not more than 1 m^3 per hour must be allowed to leak into the house if the radon gas concentration in soil air is about 50 000 Bq/m^3. These values are quite typical.

Building materials

Building materials are generally the second main source of radon indoors, while in the seventies they were considered the principal one (UNSCEAR, 1977). Radon exhalation from building materials depends not only on the radium concentration, but also on factors such as the fraction of radon produced which is released from the material, the porosity of the material and the surface preparation and finish of the walls. In general, no action needs to be taken concerning traditional building material. Typical values for radium and thorium content in building materials are 50 Bq/kg or less (NEA/OECD, 1979). Building materials containing by-product gypsum (UNSCEAR, 1982) and concrete containing alum shale (Swedjemark and Mjönes, 1984) may have much higher radium concentration. The activity concentrations in brick and concrete may also be high if the raw materials were taken from locations with high levels of natural radioactivity. Examples of such natural materials, used in some buildings, are volcanic tuffs and pozzolana (Sciocchetti et al., 1983; Campos Venuti et al., 1984; Battaglia et al., 1990), where radium and thorium content can reach some hundreds of Bq/kg. Other measurements of radioactivity content and exhalation of building materials are reported in Ingersoll (1983) and NEA/OECD (1979).

Building materials are the main sources of radon-220 (also called 'thoron')

in indoor air. Due to its short half life (55 s), thoron originating in soil in effect is usually prevented from entering buildings and therefore makes negligible contribution to indoor thoron levels. For this reason and due to the greater difficulties of measurement, thoron or thoron progeny concentration measurements are very much fewer than those for radon. Although the indoor thoron concentrations are usually low (e.g. Cliff et al., 1992; UNSCEAR, 1993), in some cases the doses due to this isotope and its daughters are significant and comparable to those due to radon-222 (Sciocchetti et al., 1983, 1992; Guo et al., 1992; Bochicchio et al., 1993b; Doi and Kobayashi 1994).

Outdoor air

Outdoor air usually acts as a diluting factor, due to its normally low radon concentration, but in some cases, as in high rise apartments built with materials having very low radium content, it can act as a real source. The radon concentration in outdoor air is mainly related to atmospheric pressure, and (in case of non-perturbative weather) it shows a typical oscillating time pattern, with higher values during night.

Until a few years ago the average level of radon gas concentration in the atmosphere at ground level was usually assumed to be of the order of a few Bq/m^3, e.g. in the range of 4–15 Bq/m^3 in the USA (Gesell, 1983), but more recent measurements seem to indicate higher values, reaching some tens of Bq/m^3 (Hopper et al., 1991; Robé et al., 1992; Bochicchio et al., 1993b; Deyuan, 1993; Grasty, 1994; Price et al., 1994). Quite high radon concentrations in the outdoor air have been reported near substantial radon sources, such as mine tailings (e.g. Tyson et al., 1993), or in the case of particular weather conditions, such as thermal inversion or very low precipitation (Grasty, 1994).

Ambient air over oceans has very low values (~0.1 Bq/m^3) of radon concentration, due to the minimum presence of radium in the sea water and the high solubility of radon in water at low temperatures. Therefore radon concentration in outdoor air of islands and coastal regions is generally lower than in continental countries, e.g., United Kingdom and Japan have an average outdoor air value of ~4 Bq/m^3.

Taking into account recent measurements, the mean value of outdoor radon concentration adopted by UNSCEAR in its last report has been changed from 5 to 10 Bq/m^3 for continental areas and somewhat less in coastal regions (UNSCEAR, 1993).

Tap-water

In wells drilled in rock the radon concentration of the water may be high. When such water is used in the household, radon will be partially released into the indoor air, causing an increase in the average radon concentration. In a

few regions, such as Finland and Maine (USA), the tap-water from wells drilled in rock has been shown to contribute significantly to radon concentrations indoors. Radon concentrations in tap-water from deep wells can range from 100 kBq/m^3 to 100 MBq/m^3 (UNSCEAR, 1988). The indoor radon concentration in these regions may already be high due to high rates of radon entry from the ground. The world average radon concentration in all types of water supplies is assumed to be 10 kBq/m^3 (UNSCEAR, 1993).

Domestic gas

In some regions, natural gas used for cooking and heating contains elevated concentrations of radon, which is released on combustion. Normally this source is not significant, and it can be monitored at transmission and distribution points. Typically the radon level in natural gas is about 1000 Bq/m^3. Natural gas as supplied usually contains gas from a number of wells and fields and thus can vary over time, depending on the proportions supplied by different sources (UNSCEAR, 1993).

3.4.3 Typical concentrations and exposures

During the 1980s in many countries surveys of indoor radon levels have been carried out (e.g. Hildingson, 1982; McGregor et al., 1980; Put and de Meijer, 1984; Schmier and Wicke, 1985; Sørensen et al., 1985; Swedjemark and Mjönes, 1984; see McLaughlin, 1987 for other references). These surveys range in type from small localised short term screening surveys to national surveys in which year-long average indoor radon concentrations have been determined in randomly chosen population weighted representative samples of national housing stock, which is the recommended methodology (UNSCEAR, 1993). National surveys which approximate in character to this latter description have been carried out or are in progress in many European countries: UK (Wrixon et al., 1988), Ireland (McLaughlin and Wasiolek, 1988), Italy (Bochicchio et al., 1993), Finland (Castrén, 1993), Sweden (Swedjemark et al., 1993); and non-European countries, e.g. the USA (Marcinowski, 1992) and Australia (Langroo et al., 1991). The preferred method of measurement in such long-term surveys is to use passive alpha track detectors which record the alpha activity from radon and its decay products.

Table 3.4 gives a summary of the principal results from a number of national and regional surveys carried out in recent years in EU Member States, other European countries, North America, Japan and Australia (see also a similar table in UNSCEAR, 1993). Unless otherwise stated, the measuring technique in most surveys was based on the use of some form of passive alpha track detector. The summary results presented here are not always

directly comparable for a number of reasons. In some surveys the dwellings were chosen in a random and representative fashion, while in others they were chosen from a specially selected group of dwellings. For a number of countries the surveys are still in preliminary stage and the number of houses surveyed per million inhabitants is rather small. In other countries such as Sweden and the UK the up-to-date total radon dwelling data that has been acquired is much more extensive than the data of the representative surveys presented in Table 3.4.

National surveys to date have shown that the average indoor radon concentration is in the 10 to 140 Bq/m^3 range. Regional average values above this range have been found in some countries. A good example of this is the UK which has a national average value of 21 Bq/m^3, while Cornwall in south-west England has an average value of about 170 Bq/m^3.

As far as the maximum indoor radon concentration likely to be present in any country is concerned it is impossible to estimate its value. Concentrations greater than 100 000 Bq/m3 have already been detected in individual dwellings in some countries. In most situations it appears that elevated indoor radon levels originate from radon in the underlying rocks and soils.

While national average indoor radon levels are in the 10 to 140 Bq/m^3 range a small percentage are considerably above this range. In keeping with the current Recommendation of the Commission of the European Communities (CEC, 1990), if 400 Bq/m^3 is taken as an 'action level' for an existing dwelling (see also Chapter 34, Section 34.1) then in many surveys the percentage of dwellings in excess of this level ranges from about 0.5 to 3%. Indoor radon levels in most surveys appear to be approximately log-normally distributed; a number of surveys have shown, however, that a log-normal approximation approach may significantly underestimate the percentage of dwellings at the highest radon levels (e.g., Goble and Socolow, 1990; Bochicchio et al., 1993; Castrén, 1993).

The observed radon daughter concentrations are usually not in equilibrium (i.e. the radon daughters have a lower concentration than radon), owing to various removal mechanisms which act on them. Radon and radon daughter concentration values are connected by the 'equilibrium factor F' (see Section 3.4.1.1). Average values reported for the equilibrium factor normally are in the range of 0.3–0.5 for residential buildings in different areas (UNSCEAR, 1988).

All previous radon concentration values refer to dwellings, because people usually spend most of their time there. However in recent years, in some countries, there has been an increase in the number of measurements being carried out in normal workplaces such as schools, offices, etc. (e.g. Strand and Kolstad, 1991; Gooding and Dixon, 1992; Poffiijn et al., 1992; SSI, 1993). As discussed in Section 34.1 on recommended and regulatory radon levels, the

TABLE 3.4

Summary of radon surveys in dwellings

Country/region (population size in millions)	No. of houses sampled	Period and duration of exposure	Sample characteristics	Radon conc. (Bq/m^3)		Geom. std. dev.	Percent over 200 Bq/m^3	Percent over 400 Bq/m^3	References
				Average	Geom. mean				
Australia (17.3)	3413	1989–90; 1year	stratified random	11	8	2.1	<0.1	<0.1	Langroo et al. (1991)
Belgium (10.0)	300	1984–90; 3 months to 1 year	popul. based selected acquaintances	48	37	1.9	1.7	0.3	Poffijn (1993)
Canada (study pop. = 7.8)	13457	summer 1977–80; grab sampling	restricted to 19 major urban areas, excluding apartments	33	15	3.6	2.3	0.6	McGregor et al. (1980); Lètourneau et al. (1984)
Czechoslovakia (15.6)	1200	1982; RnD grab sampling	–	140	–	–	–	–	UNSCEAR (1993)
Denmark (5.2)	496	1985–86; 6 mo. (1/2 houses in winter, 1/2 in summer)	random	47 (dwelling aver.); 53 (population aver.)	29	2.2	2.2	<0.4	Ulbak et al. (1988)
Finland (5.0)	3074	1990–91; 1 year	random	123	84	2.1	12.3	3.6	Castrén (1993)
France (56.9)	1548 (ongoing)	1982–91; ~3 months (using open alpha track detectors)	biased (not stratified)	85	52	2.3	7.1	2.3	Rannou et al. (1992)
Germany (77.4)	1500	1991–93; 1 year	random	50	40	–	1.5–2.5	0.5–1.0	Przyborowsky (1994)
Greece (10.2)	73	1988; 6 months	–	52	–	–	–	–	UNSCEAR (1993)

Country/region (population size in millions)	No. of houses sampled	Period and duration of exposure	Sample characteristics	Radon conc. (Bq/m^3)		Geom. std. dev.	Percent over 200 Bq/m^3	Percent over 400 Bq/m^3	References
				Average	Geom. mean				
Hungary (10.6)	122	1985–87; ~2.5 years	preliminary survey	55	42 (median)	–	–	–	Sztanyik & Nikl (1993)
Ireland (3.5)	1259	1985–89; 6 months	random	60	34	2.5	3.8	1.6	McLaughlin & Wasiolek (1988)
Italy (56.8)	4800	1989–93; 1 year	stratified random	77	–	–	5	1	Bichicchio et al. (1993; 1994)
Japan (123.9)	6300	1985–91; 1 year	random in a selected group (high school teachers)	29	23 (median)	1.6	<0.4	–	Kobayashi et al. (1991)
Luxembourg (0.4)	2500	1991	–	–	65	–	–	–	UNSCEAR (1993)
Netherlands (0.4)	~1000	1982–84; 1 year	–	29	24 (median)	1.6	–	–	Put et al. (1984; 1985); UNSCEAR (1993)
Norway (4.2)	7525	1987–89; 6 months (spread over all seasons)	random	50 (uncorr.); 60 (corr. for exposure period)	26 (uncorr.); 32 (corr.)	–	3.7 (uncorr.); 5 (corr.)	1.6 (corr.)	Strand et al. (1992); Strand (1993)
Portugal (10.3)	4200	1989–90; 1–3 months	volunteers in a selected group (high school students)	81	37	–	8.6	2.6	Faisca et al. (1992)

(continued)

TABLE 3.4 (*continuation*)

Country/region (population size in millions)	No. of houses sampled	Period and duration of exposure	Sample characteristics	Radon conc. (Bq/m^3)		Geom. std. dev.	Percent over 200 Bq/m^3	Percent over 400 Bq/m^3	References
				Average	Geom. mean				
Spain (39.0)	1555–2000	winter of 1988–89; grab sampling	random	86	41–43	2.6–3.7	–	4	Quindos et al. (1991b); UNSCEAR (1993)
Sweden (8.4)	1360	1991–92; 3 months in heating season	random	108	56	–	14–26	4.8–11	Swedjemark et al. (1993)
Switzerland (6.6)	1540 (ongoing)	1982–90; ~3 month (mainly in winter)	biased (not stratified)	80 (corr. for sample bias); 70 (corr. for expo. period)	–	–	5.0	–	Surbeck et al. (1991)
UK (57.0)	2093	1986–87; 1 year	random	20.5 (corr. for the pop. housing stock)	15	2.2	0.5	0.2	Wrixon et al. (1988)
USA (249.0)	5694	1989–90; 1 year	stratified random	46	25 (median)	3.1	~3.5	~0.6	EPA (1992c); Marcinowski et al. (1994)

Source: European Collaborative Action document no. 15 "Radon in Indoor Air", 1994.

International Commission on Radiological Protection (ICRP) has recommended that the action level for intervention in the workplace where the occupancy of members of the public is low — e.g. in offices, libraries and theatres — should be in the range 500 to 1500 Bq/m^3 (ICRP, 1993).

3.4.4 Health effects

Radon is one of a very small number of substances which has been established to be a human carcinogen on the basis of human studies. As such it is a Group 1 and Group A carcinogen, according to the classification used by the World Health Organisation (WHO/IARC, 1988) and by the US Environmental Protection Agency (EPA, 1987a), respectively. The principal adverse health effect arising from the inhalation of radon and mainly its decay products is lung cancer. Recent suggestions (Henshaw et al., 1990; 1992) that exposure to elevated levels of indoors radon may be implicated in the occurrence of other cancers such as childhood leukaemia have not yet been scientifically verified.

When radon and its short-lived decay products are inhaled the radiation dose to lung tissue is dominated by the alpha particles emitted by the deposited decay products. From a health perspective the daughter products of most significance are the four short-lived ones from polonium-218 to polonium-214 inclusive. The contribution to lung dose arising from the radon gas itself is small in comparison, as very little radon is absorbed: radon gas acts mainly as a 'carrier' of radon daughters. It has to be emphasised that although the dose is mostly due to inhaled radon progeny (and its characteristics) rather than to radon, some studies (James, 1987; Vanmarke et al., 1989; Postendorfer and Reineking, 1992) show that, in domestic ambient air, changes in ventilation rate produce opposite variations in the equilibrium factor F (i.e., the ratio between radon progeny and radon gas concentration) and in the unattached fraction, so that the absorbed dose (defined in Section 3.4.1.1) to the lung remains relatively constant at a given radon concentration.

Currently there are different approaches used to estimate the lung cancer risk arising from exposure to radon decay products in indoor air. These are: (a) the dosimetric approach, in which the radiation doses to lung tissues is estimated and from this estimated dose the associated risk is evaluated using currently accepted dose/risk factors for ionising radiations; (b) the mines epidemiology approach, in which risk estimates for underground miners are modified and applied to the general population; (c) the residential epidemiology approach, in which case-control studies of the general population are used to estimate risk factors. The risk factors obtained using these three approaches seem to be reasonably well in agreement (see Chapter 16).

Applying these risk/exposure factors to the typical average indoor radon

concentrations in North American and European countries, a not insignificant fraction (typically of the order of 10%) of total lung cancers can be attributed to radon and its decay products exposure. However, it should be strongly emphasised that the majority total lung cancers are due to smoking. For example, in a country of 50 millions with a lung cancer lifetime risk of 3% (which is the value assumed by ICRP for its 'reference' population), we can estimate that 3 persons per 1000, that is about 2000 each year, may die because of lung cancer due to radon exposure. It has to be underlined that these figures are not precise, and there is an associated uncertainty that will be discussed later in Chapter 16.

A synergistic effect seems to occur, in a greater or lesser degree, between radon and cigarette smoking, so that smokers exposed to radon have probably a quite higher risk (6–10 times) than non-smokers (ICRP, 1991; Pershagen et al., 1994). However the numerical estimates of this synergism are still very uncertain.

Indoor Air Quality
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Chapter 4

Environmental Tobacco Smoke[1]

4.1 INTRODUCTION

Smoking of tobacco is practised worldwide by hundreds of millions of people. In 1982, 6.7 million tonnes of tobacco were produced; annual *per capita* consumption in the USA ranged up to more than 3500 cigarettes. In developing countries, cigarette smoking is increasing, and many cigarettes and other products have very high tar (up to 55 mg per cigarette) and nicotine yields. In many developed countries, sizeable decreases in total consumption, sales and smoking rates have occurred. Generally, between one third and one half of men smoke, with some countries having notably higher rates. In most developed and some developing countries, about one third of women smoke, although in some countries fewer do.

Sales-weighted average tar and nicotine contents (as measured by standard laboratory methods) have declined significantly since the 1950s in some parts of the world.

Environmental tobacco smoke (ETS) is a term which describes the contaminants released into air when tobacco products burn or when smokers exhale. ETS is composed of exhaled mainstream smoke (MS) from the smoker, sidestream smoke (SS) emitted from the smouldering tobacco between puffs, contaminants emitted into the air during the puff, and contaminants that diffuse through the cigarette paper and mouth end between puffs (NRC, 1986, U.S. Department of Health and Human Services, DHHS, 1986; Guerin et al., 1992). The hazards of inhaling both mainstream and sidestream smoke are well documented (EPA, 1992). The inhalation of ETS is known as "involuntary smoking" or "passive smoking".

"Passive smoking" is a universal phenomenon where smoking is common.

1 A part of the text of this chapter has been derived from U.S. Environmental Protection Agency (EPA), 1991 "Introduction to indoor air quality", EPA/400/3-91/003; National Research Council (NRC), 1986 "Environmental tobacco smoke: measuring exposures and assessing health effects", National Academy Press: Washington, DC; U.S. Environmental Protection Agency (EPA), 1992 "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders", EPA/600/6-90/006F, December 1992, Office of Health and Environmental Assessment Office of Research and Development, U.S. EPA, Washington, DC.

The uptake of smoke constituents by smokers and by passive smokers has been studied in only a few countries, although extensive analysis of smoke shows cigarette smoking to be a major source of exposure to tobacco-specific *N*-nitrosamines, polynuclear aromatic compounds, aromatic amines and some other carcinogens (WHO, 1987).

Emissions contain both vapour phase and particle phase contaminants. SS is the major component of ETS, contributing nearly all of the vapour phase constituents and over half of the particulate matter.

ETS is a complex mix of several thousand compounds. This mix contains many known or suspected human carcinogens and toxic agents. The information necessary to evaluate human exposures to each of the compounds of human health interest in ETS does not exist.

Recognizing that it is impractical to characterize all individual compounds that make up ETS and then to assess exposures to those compounds, this chapter focuses on the characterization of the complex ETS-contaminants mix and exposure to it by nonsmokers. Particular attention is focused upon environmental and biological marker compounds that serve as proxies of the complex ETS mix and the compounds of human health interest, and on the health effects that have been recently described (EPA, 1992). Further information on health risk related to passive smoking will be found in Chapter 17.

4.2 PHYSICO-CHEMICAL NATURE

The physical and chemical characterization of MS air contaminant emissions from cigarettes, cigars, or pipes can be derived from laboratory-based studies using standardized testing protocols (Federal Trade Commission, 1990; Guerin et al., 1992). The data available are primarily for tobacco combustion in cigarettes and provide a substantial database on the nature of MS. These protocols employ smoking machines, set puff volumes and frequencies, and standardized air contaminant collection protocols (small chambers, Cambridge filters, chamber air flow rates, etc.).

Existing standardized protocols reflect conditions representative of human smoking practices of over 30 years ago for nonfiltered cigarettes and may not reflect current human smoking parameters for today's filtered low-tar cigarettes (NRC, 1986; U.S. DHHS, 1986; Guerin et al., 1992). It has been suggested that current standardized protocols, particularly for filter cigarettes, may underestimate MS deliveries (Guerin et al., 1992). MS air contaminant emission rates determined in these studies using standardized protocols can be affected by a number of factors, such as puff volume, air dilution rate, paper porosity, filter ventilation air flow around the cigarette, and moisture content of the tobacco.

Actual smoking habits of individuals can also dramatically alter the MS deliveries. Variability in any of the factors can affect the nature and quantity of the MS emissions.

Results of laboratory evaluations have indicated substantial similarities and some differences between MS and SS emissions from cigarettes (NRC, 1986; U.S. DHHS, 1986; Guerin et al., 1992). Differences in SS and MS emissions are due to differences in the temperature of combustion of the tobacco, pH, and degree of dilution with air, which is accompanied by a corresponding rapid decrease in temperature. SS is generated at a lower temperature (approximately 600°C between puffs vs. 800–900°C for MS during puffs) and at a higher pH (6.7–7.5 vs. 6.0–6.7) than MS. Being slightly more alkaline, SS contains more ammonia, is depleted of acids, contains greater quantities of organic bases, and contains less hydrogen cyanide than MS. Differences in MS and SS are also ascribable to differences in the oxygen concentration (16% in MS vs. 2% in SS). SS contaminants are generated in a more reducing environment than those in MS, which will affect the distribution of some compounds: nitrosamines, for example, are present in greater concentrations in SS than in MS.

SS is rapidly diluted in air, which results in a SS particle size distribution smaller than that for MS and in the potential for changes in phase distribution for several constituents. Nicotine, for example, while predominantly in the particle phase in MS, is found predominantly in the gas phase in ETS (Eudy et al., 1985). The shift to gas phase is due to the rapid dilution in SS. SS particle size is typically in the range of 0.01–1.0 μm, while MS particle size is 0.1–1.0 μm. The SS size distribution shifts to small sizes with increasing dilution (Ingebrethsen and Sears, 1985; NRC, 1986: U.S. DHHS, 1986; Guerin et al., 1992). The differences in size distribution for MS and SS particles, as well as the different breathing patterns of smokers and nonsmokers, have implications for deposition of the produced particle contaminants in various regions of the respiratory tract. Estimates of from 47% to more than 90% deposition for MS and of 10% deposition for SS have been reported (U.S. DHHS, 1986).

Despite quantitative differences and potential differences in phase distributions, the air contaminants emitted in MS and SS are qualitatively very similar in their chemical composition because they are produced by the same process. Over 4000 compounds have been identified in laboratory-based studies of MS (Dube and Green, 1982; Roberts, 1988). In a 1986 IARC monograph evaluating the carcinogenic risk of tobacco smoke to humans (International Agency for Research on Cancer, IARC, 1986), 42 individual MS components were identified as carcinogenic in bioassays with laboratory animals, with many of these either known or suspected human carcinogens. Many additional com-

pounds in MS have been identified as toxic compounds. Although SS emissions have not been chemically characterized as completely as MS emissions, many of the compounds found in MS emissions, including carcinogenic agents, are found in SS emissions (Dube and Green, 1982; NRC, 1986; U.S. DHHS, 1986; Roberts, 1988; Guerin et al., 1992) and at emission rates considerably higher than for MS.

Part of the data available from studies of MS and SS emissions is shown in Table 4.1 (NRC, 1986). These data are for nonfilter cigarettes and represent a summary of data from several sources. For the compounds shown in Table 4.1, all of the five known human carcinogens, nine probable human carcinogens, and three animal carcinogens are emitted at higher levels in SS than in MS, several by an order of magnitude or more. For example, *N*-nitrosodimethylamine, a potent animal carcinogen, is emitted in quantities 20 to 100 times higher in SS than in MS. Table 4.1 similarly shows that several toxic compounds found in MS are also found in SS (carbon monoxide, ammonia, nitrogen oxides, nicotine, acrolein, acetone, etc.). Again, for many of these compounds, SS emissions are higher than MS emissions, in some cases by an order of magnitude or higher.

The SS/MS emission ratios shown in Table 4.1 can be highly variable and potentially misleading because, as noted earlier, a number of factors can have a substantial impact on MS emissions. A filtered cigarette, for example, can substantially reduce MS of total mass well below that shown in Table 4.1, thus resulting in a much higher SS/MS ratio. A number of recent studies (Browne et al., 1980; Adams et al., 1987; Guerin, 1987; Higgins, 1987; Chortyk and Schlotzhauer, 1989; Guerin et al., 1992) indicate that, quantitatively, SS emissions show little variability as a function of a number of variables (puff volume, filter vs. nonfilter cigarette, and filter ventilation). The lack of substantial variability in SS emissions is related to the fact that sidestream emissions are primarily related to the weight of tobacco and paper consumed during the smouldering period, with little influence exerted by cigarette design (Guerin et al., 1992).

More recent summary data on SS emission rates from filtered test cigarettes and commercial cigarettes for many compounds of human health interest are presented by Guerin et al. (1992) and shown, with modifications, in Table 4.2. Much of the data in Table 4.2 is extracted from detailed data presented in an R.J. Reynolds report (1988). Table 4.2, like Table 4.1, shows that appreciable quantities of important air contaminants are emitted into the air from SS emissions resulting from tobacco combustion. The table demonstrates that SS emissions are reasonably similar across different brands of cigarettes, varying by only a factor of about 2. So, while MS emissions can vary considerably (Table 4.1), SS emissions are relatively constant (Table 4.2).

TABLE 4.1

Distribution of constituents in fresh, undiluted mainstream smoke and diluted sidestream smoke from nonfilter cigarettes[1] (extracted from NRC, 1986)

Constituent	Amount in MS	Range in SS/MS
Vapor phase[2]		
Carbon monoxide	10–23 mg	2.5–4.7
Carbon dioxide	20–40 mg	8–11
Carbonyl sulfide	12–42 µg	0.03–0.13
Benzene[3]	12–48 µg	5–10
Toluene	100–200 µg	5.6–8.3
Formaldehyde[4]	70–100 µg	0.1–.50
Acrolein	60–100 µg	8–15
Acetone	100–250 µg	2–5
Pyridine	16–40 µg	6.5–20
3-Methylpyridine	12–36 µg	3–13
3-Vinylpyridine	11–30 µg	20–40
Hydrogen cyanide	400–500 µg	0.1–0.25
Hydrazine[4]	32 ng	3
Ammonia	50–130 µg	3.7–5.1
Methylamine	11.5–28.7 µg	4.2–6.4
Dimethylamine	7.8–10 µg	3.7–5.1
Nitrogen oxides	100–600 µg	4–10
N-Nitrosodimethylamine[4]	10–40 ng	20–100
N-Nitrosodiethylamine[4]	ND–25 ng	<40
N–Nitrosopyrrolidine[4]	6–30 ng	6–30
Formic acid	210–490 µg	1.4–1.6
Acetic acid	330–810 µg	1.9–3.6
Methyl chloride	150–600 µg	1.7–3.3
1,3-Butadiene[4,6]	69.2 µg	3–6
Particle phase[2]:		
Particulate matter[7]	15–40	1.3–1.9
Nicotine	1–2.5	2.6–3.3
Anatabine	2–20	–0.5
Phenol	60–140	1.6–3.0
Catechol	100–360	0.6–0.9
Hydroquinone	110–300	0.7–0.9
Aniline[4]	360	30
2-Toluidine	160	19

(continued)

TABLE 4.1 (*continuation*)

Constituent	Amount in MS	Range in SS/MS
2-Naphtylamine[3]	1.7	30
4-Aminobiphenyl[3]	4.6	31
Benz(a)anthracene[5]	20–70	2–4
Benzo(a)pyrene[4]	20–40	2.5–3.5
Cholesterol	22	0.9
γ–butyrolactone[5]	10–22	3.6–5.0
Quinoline	0.5–2	3–11
Harman[8]	1.7–3.1	0.7–1.7
N-Nitrosonornicotine[5]	200–3000	0.5–3
NNK[9]	100–1000	1–4
N–Nitrosodiethanolamine[4]	20–70	1.2
Cadmium[4]	110	7.2
Nickel[3]	20–80	13–30
Zinc	60	6.7
^{210}Polonium[3]	0.04–0.1	1.0–4.0
Benzoic acid	14–28	0.67–0.95
Lactic acid	63–174	0.5–0.7
Glycolic acid	37–126	0.6–0.95
Succinic acid	110–140	0.43–0.62
PCDDs and PCDFs[10]	1	2

[1] Data come from the NRC report (1986), except where noted, with compiled data from Elliot and Rowe (1975), Schmeltz et al. (1979), Hoffman et al. (1983), Klus and Kuhn (1982), Sakuma et al. (1983, 1984a,b) and Hiller et al. (1982). Full references are given in NRC (1986). Diluted SS is collected with airflow of 25 ml/s, which is passed over the burning cone; as presented in the NRC report on passive smoking (NRC, 1986).
[2] Separation into vapor and particle phases reflects conditions prevailing in MS and does not necessarily imply same separation in SS.
[3] Known human carcinogen, according to U.S. EPA or IARC.
[4] Probable human carcinogen, according to U.S. EPA or IARC.
[5] Animal carcinogen (Vainio et al., 1985).
[6] Data from Brunnemann et al., 1990; PCDDs = polychlorinated dibenzo–p–dioxins.
[7] Contains di- and polycyclic aromatic hydrocarbons, some of which are known animal carcinogens.
[8] 1-Methyl-9H-pyrido[3,4–b]–indole.
[9] NNK = 4-(*N*-methyl-*N*-nitrosamino)-1-(3-pyridyl)-1-butanone.
[10] Data from Löfroth and Zebühr (1992). Amount is given as International Toxic Equivalent Factor (I-TEF).

TABLE 4.2

Example of sidestream cigarette smoke deliveries[1]

Constituent	Kentucky reference[2] (mg per cig.)	Commercial (mg per cig.)
Condensate		36–67
Total particulate matter	16.9	16–36
Nicotine	5.6	5.7–11.2
Carbon monoxide	54	41–67
Carbon dioxide	474	
Nitrogen oxides	0.9	
Ammonia	9.1	
Formaldehyde	0.7	
Acetaldehyde	4.2	
Acrolein	1.3	1.4
Propionaldehyde	0.9	
Benzene	0.3	0.4
Toluene	0.8	1.3
Pyrrole	0.4	
Pyridine	0.3	
Limonene	0.3	<0.1–0.4
Neophytadiene		0.1–0.2
Isoprene	2.5	6.1
nC_{27}–nC_{33}	0.2–0.8	
Acetonitrile	1.0	0.8[3]
Acrylonitrile	0.2	
Hydrogen cyanide	53	17[3]
Phenol		44–371
o-Cresol		24–98
m+*p*-Cresol		59–299
Catechol		46–189
Hydroquinone		26–256
Naphthalene		53–177
Phenanthrene		2.4
Anthracene		0.7
Fluoranthene		0.7
Pyrene		0.5
Benz(a)anthracene	0.2	0.2
Benzo(a)pyrene	0.1	0.1
NNN[4]	0.2	1.7
NNK[4]	0.4	0.4
NAT[4]	0.1	
NAB[4]	<0.1	
DMNA[4]	0.3	0.7–1.0
EMNA[4]		<0.1
DENA[4]		<0.1–0.1
NPYR[4]	0.2	0.2–0.4

(continued)

TABLE 4.2 (*continuation*)

Constituent	Kentucky reference[2] (mg per cig.)	Commercial (mg per cig.)
2-Naphtylamine		<0.1–1[5]
4-Aminobiphenyl		<0.1–0.2[5]

1 Table reprinted from Guerin et al. (1992) who compiled data from Browne et al. (1990); Brunnemann et al. (1977,1978); Chortyk and Schlotzhauer (1989); Grimmer et al. (1987); Guerin (1991); Higgins et al. (1987); Johnson et al. (1973); O'Neill et al. (1987); R.J. Reynolds (1988); Rickert et al. (1984); Sakuma et al. (1983, 1984a,b; and Norman et al. (1983). Full references are given in Guerin et al. (1992).
2 Filter IR4F unless otherwise specified.
3 Nonfilter IRI.
4 *N*-nitrosonornicotine (NNN), 4-Methylnitrosoamino-1-(3-pyridinyl)-1-butanone (NNK), *N*-nitrosoanatabine (NAT), *N*-nitrosoanabasine (NAB), dimethylnitrosamine (DMNA), ethylmethylnitrosamine (EMNA), diethylnitrosamine (DENA), *N*-nitrosopyrrolidine (NPYR).
5 Calculated from NRC, 1986, SS/MS ratio.

Detailed chemical characterizations of ETS emissions under conditions more typical of actual smoking conditions (e.g., using smokers rather than smoking machines) are limited. As a result, the impact on ETS of factors such as the rapid dilution of SS emissions, adsorption and remission of contaminants, and exhaled MS is not well understood. Several studies conducted in chambers or controlled environments and using smokers (e.g., NRC, 1986; U.S. DHHS, 1986; Benner et al., 1989; Duc and Huynh, 1989; Reynolds, 1988; Leaderer and Hammond, 1991; Guerin et al., 1992) have characterized some of the ETS components (total mass, carbon monoxide, nicotine and other selected compounds, including known carcinogenic and toxic substances). These studies indicate that many of the contaminants of interest in SS are measurable in ETS and that several SS contaminants (e.g., total mass, carbon monoxide, nicotine) are easily measurable in ETS. It is not known how the MS and SS air contaminant emission data for specific compounds, generated by the standardized testing protocols utilized, compare to data gathered under conditions more representative of actual smoking in occupied spaces.

4.3 TYPICAL CONCENTRATIONS AND EXPOSURES

The concentrations of the known and unidentified contaminants in the ETS complex mix in an enclosed space are the result of a complex interaction of several important variables, including: (1) the generation rate of the contaminant(s) from the tobacco (including both SS and exhaled MS emissions); (2)

location in the space that smoking occurs; (3) the rate of tobacco consumption; (4) the ventilation rate; (5) the concentration of the contaminant(s) in the ventilation or infiltration air; (6) air mixing in the space; (7) removal of contaminants by surfaces or chemical reactions; (8) re-emission of contaminants by surfaces; and (9) the effectiveness of any cleaners that may be present. Additional parameters influencing the concentration level relate to the location at which contaminant measurements are made, the time of sample collection, the duration of sampling.

Variations in any one of the above factors can have a marked impact on the resultant indoor ETS constituent concentrations. Any one of these parameters can vary by an order of magnitude or more. For example, infiltration rates in residence can range from 0.1 to over 2.0 air changes per hour, and house volumes can range from 100 to over 700 m^3. Smoking rates and mixing within and between rooms can also show considerable variability.

Numerous field studies in "natural" environments have been conducted to assess the contribution of smoking occupancy to indoor air quality. These studies, summarized in a number of reviews (e.g., NRC, 1986; U.S. DHHS, 1986; Guerin et al., 1992), have measured several ETS-related contaminants of human health concern (e.g., particle mass, carbon monoxide, benzene, nicotine, polycyclic aromatic hydrocarbons, *N*-nitrosamines), in a number of enclosed environments (e.g., residential, office, transportation) and under a variety of smoking and ventilation rates. These studies demonstrate that: (1) many of the contaminants of health interest found in SS are also found in ETS; (2) ETS contaminants are found above background level in a wide range of indoor environments in which smoking occurs; and (3) the concentrations of ETS contaminants indoors can be highly variable. These findings are supported by the data given in Table 4.3 which relate to tobacco generated nitrosamines.

Acetaldehyde is the most prominent carbonyl compound in both, MS and SS cigarette smoke accounting for 57 ± 15% of the total amount in carbonyl compounds. Concentrations of carbonyl compounds in MS smoke have been shown to increase exponentially with the puff number. As a result under standard smoking conditions the last two of an average of 10 puffs/cigarette contain about the same amount of carbonyl compounds as the preceding eight (Schlitt and Knöppel, 1989). Cigarettes with and without filter release comparable amounts of carbonyl compounds to the environment. This amount is under standard smoking conditions (35 mL/puff, 1 puff/min) from 4 to 500 times higher than that delivered to MS smoke, i.e. to the smoker, depending on the compound and cigarette type. In particular a "passive" smoker at 50 cm distance of a cigarette may inhale more than 10 times the amount "actively" taken up by the smoker (Schlitt and Knöppel, 1989).

TABLE 4.3

Tobacco-specific *N*-nitrosamines in indoor air (ng/m^3)[1]

Site	Approx. no. cigarettes	Collection time (h)	Flow rate (l/min)	Tobacco-specific *N*-nitrosamines		
				NNN[2]	NAT[2]	NNK[2]
Bar I	25–35	3	3.2	22.8	9.2	23.8
Bar II	10–15	3	3.2	8.3	6.2	9.6
Bar III	10–15	3	3.2	4.3	3.7	11.3
Restaurant[3]	25–30	6	2.15	1.8	1.5	1.4
Restaurant[3]	40–50	8	2.1	ND	ND	3.3
Car[4]	13	3.3	2.15	5.7	9.5	29.3
Train I	50–60	5.5	3.3	ND	ND	4.9
Train II	50–60	6	3.3	ND	ND	5.2
Office	25	6.5	3.3	ND	ND	26.1
Smoker's home	30	3.5	3.3	ND	ND	1.9

1 Data corrected for recovery.
2 NNN = NNN-*N*-nitrosonornicotine; NAT = NAT-*N*-nitrosoanataline; NNK = NNK-4-methylnitrosoamino-1-(3-pyridinyl)-1-butanone.
3 Smoking section.
4 Windows partially open.
ND = not detected (in some cases due to chromatographic interference).
Source: Brunnemann et al., 1992.

N-nitrosamines are important constituents of SS because they are considered to be carcinogenic and they are emitted in much larger quantities in SS than in MS (Table 4.1). Tobacco combustion is the only identified air source of *N*-nitrosamines in the nonoccupational indoor environment. Guerin et al. (1992) reviewed the available data on indoor levels of *N*-nitrosamines related to smoking occupancy. They concluded that levels associated with smoking can range from less than detectable to as high as 100 ng/m^3 for nitrosodimethylamine (NDMA) under conditions of heavy smoking. A more typical range of concentrations of NDMA were < 10–40 ng/m^3. In a recent paper, Brunnemann et al. (1992) demonstrated that exposure to tobacco specific *N*-nitrosamines can occur in a variety of indoor spaces under a range of smoking conditions (Table 4.3).

The potential for high exposures of nonsmokers to carcinogenic components found enriched in SS can be demonstrated in the case of 4-aminobiphenyl (4-ABP). Tables 4.1 and 4.2 show 4-ABP emissions in SS to be approximately 30 times higher than in MS (100–200 μg/cigarette). Despite the fact that SS

emissions of 4-ABP are diluted rapidly in the indoor environment, presumably resulting in considerably less exposure than to smokers, 4-ABP haemoglobin adduct levels in nonsmokers have been found to be 10–20% of those in smokers (EPA, 1992).

There are important circumstances where concentrations of ETS-related contaminants in indoor spaces may considerably underestimate potential levels of exposure. These circumstances occur when the SS emissions or exhaled MS emissions are in direct proximity to a nonsmoker (e.g. an infant held by a smoking mother or father, or when a nonsmoker is directly downwind of the plume of a smouldering cigarette). While there are no measurements to assess the impact on the nonsmoker's exposure under these conditions, the effect may become important and exposure will be much higher than it would be predicted from existing environmental measurements of more diluted SS and exhaled MS emissions.

The data discussed above represent concentrations measured in selected indoor environments and indicate that exposure will occur for individuals in those spaces. Estimating the actual level of exposure (concentration $\times$ time) requires knowledge of the actual time spent in those environments.

Nicotine and its metabolite, cotinine, in the saliva, blood, and urine are widely used as biomarkers of active smoking and exposure to ETS and are valuable in determining total or integrated short-term dose to ETS across all environments (NCR, 1986; U.S. DHHS, 1986). Nicotine and cotinine are specific to tobacco and are accurately measured by gas chromatography, radio immunoassay, or high-pressure liquid chromatography in concentrations down to 1 ng/ml. Nicotine has a half-life typically of about 2 hours in the blood and is metabolized to cotinine and excreted in the urine. The short half-life of nicotine makes it a better indicator of very recent exposures rather than a measure of integrated exposure.

Cotinine in saliva, blood, and urine is the most widely accepted biomarker for integrated exposure to MS or ETS (NRC, 1986; U.S. DHHS, 1986). Cotinine is the major metabolite of nicotine, is specific to tobacco, and has a longer half-life for elimination from the body. The elimination half-life in smokers is approximately 20 hours (range of 10–37 hours), while it is typically longer in non-smokers with ETS exposure, particularly in children (Elliot and Rowe, 1975; Collier et al., 1990; Goldstein et al., 1987; Etzel et al., 1985; Greenberg et al., 1984). The longer half-life of cotinine makes it a good indicator of integrated ETS exposure over the previous day or two. Laboratory studies of non-smokers exposed to acute high levels of ETS over varying times have shown significant uptake of nicotine by the non-smokers and increases in their levels (Russell and Feyerabend, 1975; Hoffmann et al., 1984; NCR, 1986; U.S. DHHS, 1986).

4.4 HEALTH EFFECTS

Tobacco smoke affects not only people who smoke but also people who are exposed to the combustion products of other people's tobacco. The effects produced are not necessarily the same, as the constituents of smoke vary according to its source. Smokers are exposed to MS and SS to a greater extent than are nonsmokers. It follows that it is unlikely that any effects will be produced in passive smokers that are not produced to a greater extent in smokers and that types of effects that are not seen in smokers will not be seen in passive smokers. Examination of smoke from the different sources shows that MS and SS contain chemicals that are both carcinogenic and mutagenic. The amounts absorbed by passive smokers are, however, small, and effects are unlikely to be detectable unless exposure is substantial and very large numbers of people are observed. The observations on nonsmokers that have been made so far would be compatible with either an increased risk from "passive" smoking or an absence of risk. Knowledge of the nature of MS and SS, of the materials absorbed during "passive" smoking, and of the quantitative relationships between dose and effect that are commonly observed from exposure to carcinogens, however, leads to the conclusion that passive smoking gives rise to risk of cancer (WHO, 1987).

Certain biochemical markers of smoke intake, e.g. cotinine (metabolite of nicotine) in plasma, urine or saliva, are sufficiently sensitive and specific to identify passive smokers. Passive smokers who have been examined in western Europe and North America generally have levels between about 0.1–1% of these markers as compared to active smokers. The precise quantitative relationship between the measured levels of these markers and the intake of carcinogenic compounds in tobacco smoke is not known.

Acute, noxious effects (NRC, 1986)

The most common acute effects associated with exposure to ETS are eye, nose, and throat irritation, and objectionable smell of tobacco smoke. Annoyance with noxious tobacco odour largely governs the reactions of visitors, while occupants of smoky rooms are more likely to complain about irritating effects to the eye, nose, or throat. Particle filtration appears to lead to little or no decline in odour and irritation, suggesting that the effects are produced by gas-phase constituents. During exposure to ETS, eye blink rate is correlated with sensory irritation, such as burning eyes and nasal irritation. For some persons, eye tearing can be so intense as to be incapacitating. There is some evidence that nonsmokers are more sensitive to the noxious qualities of cigarette smoke than are smokers.

Smoke contains immunogens, that is, substances that can activate the immune system. Approximately half of atopic (allergy prone) individuals react to various extracts of tobacco leaf or smoke presented in skin tests. However, the components of the extract that are responsible for this reaction have not been isolated. There is little correlation between positive reactions to skin tests and self-reported complaints of tobacco smoke sensitivity.

Respiratory symptoms and lung function (NRC, 1986)

Passive smoking substantially increases respiratory illness in children. Children who live in households where there are smokers are more likely to have respiratory infections (including bronchitis and pneumonia) than children in nonsmoking households. Additional effects in children include increases in coughing, wheezing, sputum production, slower lung function growth, and low-birth-weight babies in mothers that are nonsmokers but are exposed to ETS. The prevalence of these effects has been found to increase with the number of the smokers in the home.

An important issue currently unresolved is whether a child who is affected by exposure to ETS from parental smoking may be at an increased risk for the development of chronic airflow obstruction in adult life.

The respiratory tract is protected from injury by many interrelated defense mechanisms including mechanical defences, fluids that line the airways, airway reflexes, and antimicrobial, inflammatory and immune defences. If one or more of these is impaired, respiratory damage and disease can result.

The tracheobronchial region includes the trachea and bronchi. The conducting airways are lined with ciliated epithelium and coated with a thin layer of mucus. Heavy smokers typically have to cough up mucus in the morning when they arise because of a build-up of mucus during the night as a result of cilia not being able to remove mucus efficiently. If the cilia becomes damaged they cannot propel the mucus. It remains in the airways until it is caught up and expectorated or swallowed

The bronchioles do not contain as many ciliated cells as the larger airways; they have almost no mucus producing cells and a very thin layer of airways secretions that line airways. The bronchioles appear to be particularly susceptible to injury from contaminants, particularly cigarette smoke (and ozone).

Cancer (NRC, 1986)

Considering the evidence as a whole, exposure to ETS increases the incidence of lung cancer in nonsmokers. Estimates of the magnitude of the increased risk vary.

Since carcinogenic agents contained in ETS are inhaled by nonsmokers, in the absence of a threshold for carcinogenic effects, an increased risk of lung cancer due to ETS exposure is biologically plausible.

The NAS Report (NRC, 1986) estimates that the risk of lung cancer is about 30% higher for nonsmoking spouses of smokers than for nonsmoking spouses of nonsmokers, and that as many as 20% of lung cancers in nonsmokers may stem from exposure to tobacco smoke.

There have been few studies of risk for cancers other than lung cancer in nonsmokers exposed to ETS. Some of the sites considered have been brain, haematopoietic, and all sites combined. The results of these studies have been inconsistent. For a more complete assessment of the carcinogenic risk of ETS, see Chapter 17.

Cardiovascular disease (NRC, 1986)

Since active smoking has an adverse effect on cardiovascular disease morbidity and mortality, ETS exposure has also become suspect. Reports have noted an excess risk of cardiovascular disease in ETS-exposed nonsmokers; however, methodologic problems in the designs and analysis of these studies preclude a firm conclusion about results (see also Chapter 12).

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Chapter 5

Biological Agents[1]

5.1 INTRODUCTION[2]

The four major categories of biological particle which affect indoor air quality (ECA, 1993) are mites and their faeces, dander from pets and other furred animals, fungi, including moulds and yeasts, and bacteria, including actinomycetes.

Various reports have linked the four categories of biological particle in houses, non-industrial workplaces and public buildings with one or more of the following allergic manifestations among the occupants:

1. Rhinitis, with "hay fever" symptoms such as nasal congestion, runny nose, sneezing, conjunctivitis and lachrymation;
2. Asthma, with symptoms which include wheeze, tightness of the chest and shortness of breath;
3. Humidifier fever, with symptoms including fever, chills, muscle ache and malaise, but no obvious respiratory effects;
4. Extrinsic allergic alveolitis (hypersensitivity pneumonitis), with acute pneumonia-like bouts of fever, cough, tightness of the chest and lung infiltration, or chronic development of cough, shortness of breath and infiltration of lungs; and
5. Atopic allergic dermatitis.

However, allergens produced by house dust mites and in the dander of furred domestic animals are generally the most important cause of disease episodes in atopic individuals.

In a U.K. study, some 80% of asthmatic children were allergic to house dust mite (Price et al., 1990), and a Swedish study showed that 57% of asthmatic children were allergic to at least one type of furred animal (Kjellman and Pettersson, 1983). Various investigations have shown that the numbers of

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1 A part of the text of this chapter has been derived from ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man"), 1993 Biological Particles in Indoor Environments, Report No. 12 (EUR 14988 EN), Luxembourg: Office for Official Publications of the European Communities.

2 Text excerpted from ECA (1993).

house dust mites are higher in damp houses (e.g. Burr et al., 1980), so that the incidence of mite-induced asthma is likely to be higher in damp housing. The major mammalian sources of allergens are the dander (skin scales) of cats and dogs, but dried urine and saliva of these animals are also potential sources of allergens (Knysak, 1989). Among atopic children, there appears to be a greater incidence of allergy to cats than to other pets. Mice, rats and a number of other rodents which are popular as pets may also contribute to problems of allergy, e.g. hamsters, desert-rats (gerbils) and guinea pigs (Knysak, 1989).

Mould growth in any building is in itself undesirable and controllable, but in addition to being a potential health hazard it is an indicator that conditions of moisture availability may also be favourable for yeasts and bacteria, which can also impact on health. Although most investigations have shown that far fewer respiratory patients with suspected allergy react to moulds than to house dust mites and animal dander (e.g. Beaumont, 1985), mould allergy among atopic children is frequent and may be severe.

Fungi can be the cause of individual cases of rhinitis, asthma, extrinsic allergic alveolitis and atopic allergic dermatitis, and have been invoked as a cause of humidifier fever. They may have a wider role in respiratory health via non-allergic mechanisms. Such mechanisms may involve mycotoxins in inhaled mould spores. Wall lipopolysaccharide fractions of Gram-negative bacteria are also toxic, and can provoke symptoms very similar to those of humidifier fever (Rylander, 1986).

Like fungi, bacteria have also been implicated in allergic diseases, and in buildings both types of organism generally live as saprotrophs (Flannigan et al., 1991). As such, they are able to lead an independent existence under damp conditions by utilizing for growth and multiplication the organic matter in wall coverings, soil (e.g. plant pots), dust, humidifiers, air ducts and soft furnishings, such as upholstered furniture and carpets. A very small number of these organisms which can lead a saprotrophic existence in buildings are also opportunistic pathogens of man, e.g. the mould *Aspergillus fumigatus*. In addition, members of the bacterial genus *Legionella*, which can proliferate in water supplies and humidifiers, include *L. pneumophila*, the infectious agent of the acute respiratory disease Legionnaires' Disease and the non-pneumonic disease Pontiac Fever. Outbreaks of Legionnaires' Disease have been associated with hotels, hospitals and other large public buildings in particular.

Biological particles which are not dealt with in this chapter include pollen and insect-derived material, although both may affect respiratory health. In the former case, respiratory problems are primarily associated with exposure to the higher concentrations of pollen outdoors, and the number of cases of respiratory problems involving insect particles is small in relation to cases involving the four main categories dealt with. However, it must be acknow-

ledged that airborne particles from cockroaches (particularly *Blatella germanica* and *Periplaneta americana*) may be of significance in the health of individuals in socioeconomically disadvantaged groups, and particles from the cat flea or the common clothes moth may also affect health (Mathews, 1989).

Some viral illnesses such as the common cold and measles may be transmitted via indoor air, e.g. measles in schools (Riley et al., 1978), and rates of viral infection in buildings with heating, ventilating and air conditioning (HVAC) systems with recirculating air may be higher than in naturally ventilated buildings (Brundage et al., 1988). However, it is generally held that person-to-person transmission is the principal cause of outbreaks of most viral diseases, e.g. mumps. Therefore, although they are of great epidemiological significance, such viruses and other pathogenic organisms from human and animal sources will not be considered in this chapter.

5.2 HOUSE DUST MITES[1]

5.2.1 Occurrence

The natural food source of house dust mites appears to be skin scales, primarily from man, and/or fungi growing on skin scales. However, many other food sources may be used (Platts-Mills and de Weck, 1988). House dust mites require particular conditions of temperature and humidity in order to grow. For *Dermatophagoides pteronyssinus*, the optimum conditions are 25°C and a relative humidity of 70–80%. The humidity seems to be the most critical factor in the survival and development of house dust mite populations (Mosbech, 1985).

House dust mites have been identified in most parts of the world, and mite-allergen levels in house dust have traditionally been assessed by counting isolated mite bodies under the light microscope. The earliest studies on mite counts reported seasonal and geographic variation in the numbers of mites in house dust attributable to differences in humidity. For example, in temperate climates the lowest numbers are found in winter and the highest numbers in summer. In tropical countries, high numbers of mites can generally be found throughout the year. At high altitudes, the numbers of mites are lower. Environmental conditions also influence the species distribution. *D. pteronyssinus* usually dominates in continuously humid conditions, whereas *D. farinae* tends to predominate in areas where there is a prolonged period (more than 3 months) of dry weather. In most cases, it is unusual for other mite species to account for more than 10% of the mite population (Platts-Mills and Chapman, 1987).

The highest numbers of mites in houses are usually found in mattresses,

1 Text excerpted from ECA (1993).

bedding, pillows, children's stuffed toys, upholstered furniture, and carpeting. Overall, the number of mites may vary from 10 to 1000 mites/g house dust (Pollart et al., 1988).

5.2.2 Typical concentrations

As mite numbers in dust vary considerably, it is to be expected that there will be differences in the amounts of mite allergen between different locations, seasons and geographic areas. However, mite allergen levels in house dust do not correlate well with mite numbers in house dust. For example, in temperate climates, levels of Der p I allergen in dust may stay high until January, even though living mite numbers fall in September. It is important to realize that dead or degraded mite bodies still have their allergenic properties (Platts-Mills and Chapman, 1987; Pollart et al,. 1988). As is the case for the mite numbers, there is a wide range in the levels of house dust mite allergens in house dust, from < 10 ng to > 300 000 ng/g dust.

Tables 5.1 and 5.2 and Figure 5.1 indicate recently reported values of major house dust mite allergens (Der p I, Der f I and Der m I) obtained with immunochemical assays. The observed values from Europe and USA range from 20 to 200 000 ng major allergens/g dust from house dust samples in private homes. The detection limits for these assays vary from 10 to 20 ng/g dust.

5.2.3 Health effects

Since the importance of house dust mites in human health lies in their allergenic properties, it can expected that dust extracts will give relevant positive reactions in skin tests on asthmatics. The role of mites in the family Pyroglyphidae as the most important source of house dust allergens is established. The most important mite species are *D. pteronyssinus*, *D. farinae*, *D. microceras* and *Euroglyphus maynei* (Mosbech 1985). However, a variety of non-pyroglyphid (storage) mites in the genera *Acarus*, *Lepidoglyphus*, *Tyrophagus* and *Glycyphagus* can live in ordinary house dust as well, especially in damp houses (Mosbech, 1985; Revsbech and Dueholm, 1990).

In the last decade, considerable progress has been made in the identification, purification and characterization of allergens produced by *Dermatophagoides* spp. Their major allergens (Der p I, Der p II, Der f I, Der f II and Der m II) are well characterized and purified (Lind, 1981; Platts-Mills and Chapman, 1987). Der p I, Der f I and Der m I have been shown to be faecal allergens, and very high concentrations of Der p I are present in mite faeces. Faecal pellets of the mites are about 25 μm in size, but can break into smaller particles which can be inhaled (Platts-Mills and Chapman, 1987).

TABLE 5.1

Overview of five studies on mite allergen levels (after Schwartz et al., 1987)

Study no.	Samples (n)	Allergens	Ranges (ng/g dust)	Location	Home of allergics
1	48	Der p I	$<20–1{\cdot}10^5$	Austria	Yes
		Der f I	$<20–5{\cdot}10^4$		
		Der m I	$<45–8{\cdot}10^3$		
2	24	Der p I	$<20–8{\cdot}10^3$	Belgium	Yes
		Der f I	$<10–5{\cdot}10^2$		
		Der m I	$<20–1{\cdot}10^4$		
3	42	Der p I	$<60–1{\cdot}10^4$	Maryland, USA	No
		Der f I	$<45–1.5{\cdot}10^4$		
		Der m I	<80		
4	18	Der p I	<20–50	Sweden	Yes
		Der f I	<20–50		
		Der m I	<45–50		
5	22	Der p I	$<30–2{\cdot}10^5$	Denmark	No
		Der f I	$<10–6{\cdot}10^4$		
		Der m I	$<60–2{\cdot}10^5$		

TABLE 5.2

Der p I concentrations in house dust (μg/g dust), The Netherlands (after Verhoeff, 1994)

Sample type	Geometric mean	Range	n
Living room, uncarpeted	0.37	0.10–31.08	112
Living room, carpeted	3.98	0.09–150.52	400
Bedroom, uncarpeted	0.64	0.09 –14.10	144
Bedroom, carpeted	3.56	0.10–103.56	370
Mattress	5.07	0.10–280.88	512

Allergy to mite allergens is fairly common in the atopic population. The reported prevalence among asthmatics varies from 45 to 85% (Platts-Mills and de Weck, 1988). Sensitivity to mites is therefore a severe risk factor for asthma. For individuals with manifest allergy to house dust mite, exposure to 500

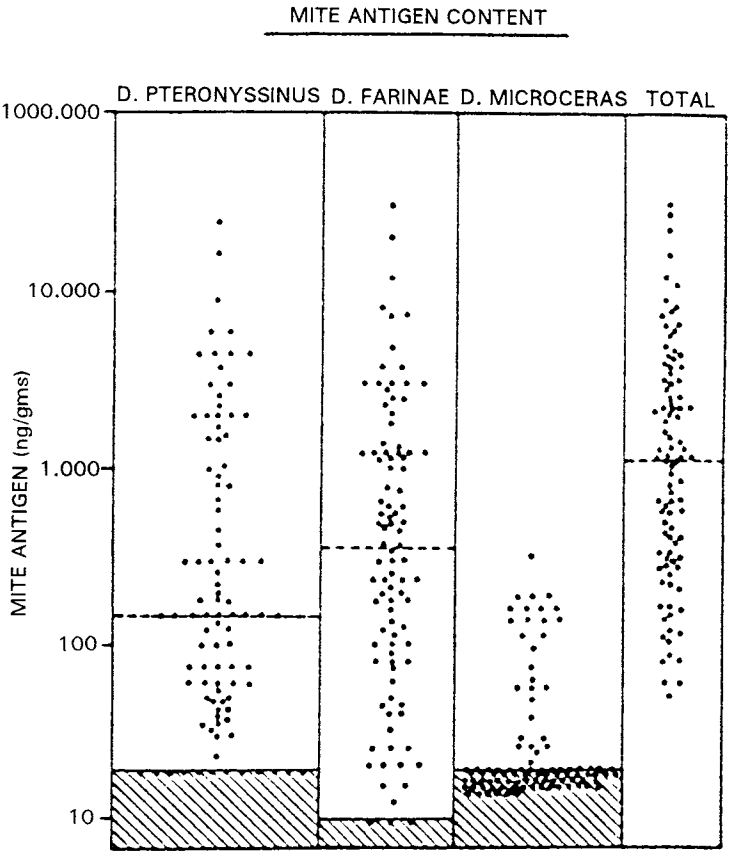

Fig. 5.1: Mite antigen content (ng/g) as determined for 97 homes by ELISA for three mite species (*Dermatophagoides pteronyssinus*, *D. farinae* and *D. microceras*) and total content (determined as the sum of the three species). Dotted lines indicate medians. Hatched areas below solid lines indicate lower limits of sensitivity of the assays (20 ng/g for *D. pteronyssinus* and *D. microceras*, 10 ng/g for *D. farinae*). From Wood et al. (1988).

mites/g dust, corresponding to 10 fg Der p I/g dust, is considered to be a level that provokes acute asthma attacks (Platts-Mills and de Weck, 1988).

5.3 DANDER FROM FURRED ANIMALS (PETS)[1]

5.3.1 Occurrence

Dogs and cats are likely to be the most common animal species with which humans have close domestic contact, but the keeping of other pets such as

1 Text excerpted from ECA (1993).

guinea pigs, rabbits, hamsters and birds is also widespread in Western society. Most pets produce allergens which can be inhaled (Lowenstein et al., 1986). In addition, rats, mice and insect vermin, such as cockroaches, may be sources of allergens in indoor environments (Reed and Swanson, 1986).

The presence of pet-derived allergenic material is independent of environmental factors such as altitude, humidity, temperature or the quality of the building. The major factor of importance is the thoroughness of cleaning in the building, i.e. the removal of allergenic material produced in the house or brought in from outside.

The allergens produced by pets are mostly associated with dander, hair, saliva and/or urine (Lowenstein et al., 1986). The most important source of cat allergens is dander. The major allergen of the cat (Fel d I) has been isolated, characterized and standardized. This allergen is released into indoor air and house dust in dander and the shed hair to which the allergens are attached (Wentz et al., 1990). The allergen becomes airborne as particles 1–10 μm in diameter, presumably after it dries and flakes off the fur (Reed and Swanson, 1986). The recent indication by Luczynska et al. (1990) that airborne Fel d I is associated with small particles which remain airborne for long periods might explain the distinctive rapid onset of asthma or rhinitis in patients allergic to cats on entering a house with a cat. Lopes da Mata et al. (1990) suggested that cats are the most important pets causing allergy in the home, owing to their widespread occurrence and the allergenic potency of the allergens they produce.

5.3.2 Typical concentrations

Fel d I can be detected in indoor air, house dust and also mattress dust. In air samples taken with cascade impactors, impingers or high volume samplers in houses where at least one cat was present, the allergen concentrations ranged from 250 to 1140 ng/m^3 air. In houses without cats, the concentration of Fel d I in house dust was from below the limit of detection to 100 000 ng/g dust, but in houses where cats were present the concentrations ranged from zero to around 300 000 ng/g (e.g. Lowenstein et al., 1986; Wood et al., 1988; Luczynka et al., 1990). In houses where cats are present, dust samples usually contain more than 8 μg Fel d I/g dust. Figures 5.2 and 5.3 indicate levels of cat allergen (Fel d I) observed in samples in Europe and USA to be in the range 30–100 000 ng Fel d I/g dust.

The concentrations of dog allergens found in house dust vary widely. In house dust sampled in houses without dogs, concentrations of 110–82 500 IU/g have been found, and in houses where dogs are present 1100–585 000 IU/g have been reported (Wood et al., 1988). Dog allergens have also been demon-

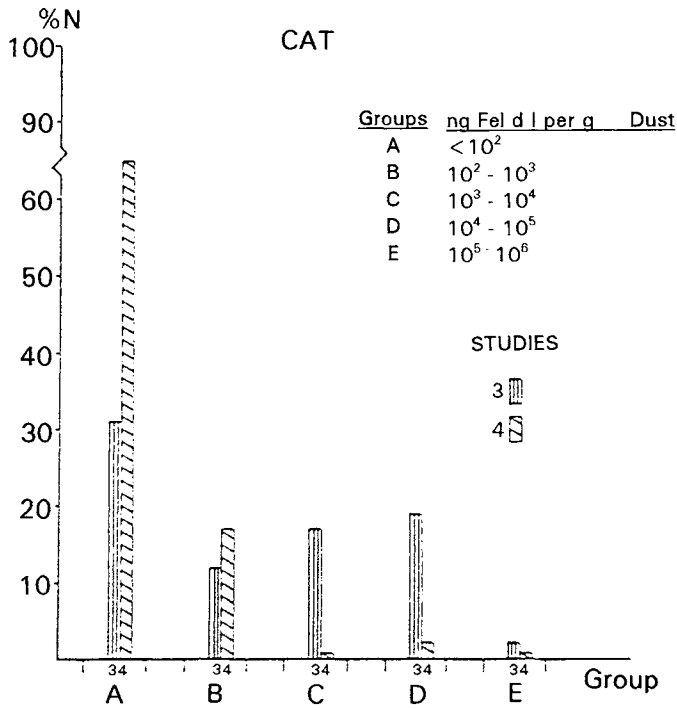

Fig. 5.2: The content of Fel d I in dust samples collected at two different locations: one from homes of allergics (3) and one from homes of non-allergics (4). From Schwartz et al. (1987) (S. Karger AG, Basel)..

strated in mattress dust (Lind et al. 1987b). In houses where dogs are present, dust samples contain generally more than 10 μg Can f I/g dust.

5.3.3 Health effects

Prevalence studies of hypersensitivity to cat allergens in unselected populations have shown a frequency of positive skin reactions of around 15%. Hader et al. (1990) found in a cross-sectional study with 704 unselected schoolchildren (7–16 years) that 10.5% had a positive skin test against cat dander. Sears et al. (1989) followed a birth cohort of children in New Zealand for 13 years. Of 714 children skin-tested, 13.3% were sensitive to cat dander. Lelong et al. (1990) reported for a group of allergic children a frequency of sensitization to cats of 30%. Among atopic individuals who are not sensitive to house dust mite allergens, a prevalence of 80% is reported (Pauli et al., 1979; Ammann and Wütrich, 1985).

Adverse reactions to dog dander are considered to be species-specific (Blands et al., 1977), although evidence of breed-specific allergens has been

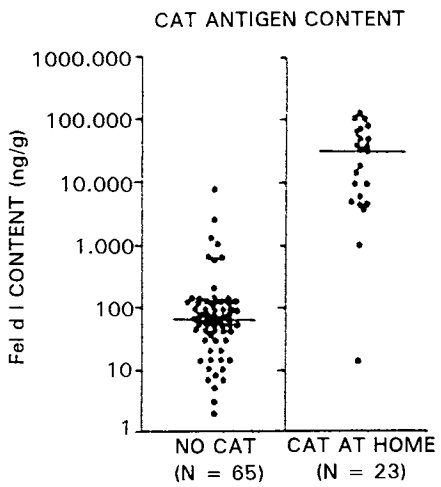

Fig. 5.3: Cat antigen concentration (ng/g) as determined for 90 homes by ELISA specific for Fel d I without a cat on left, with a cat on right. Lower limit of sensitivity is 0.5 ng/g. Difference between median antigen concentration (solid lines) of homes with and without a cat are statistically significant (median 63 versus 18 000 ng/g; $p < 0.001$). From Wood et al. (1988).

presented (Lindgren et al., 1988). Furthermore, cross-reactivity between allergens of cats and dogs has been found. Isolation and standardization of the major allergen(s) produced by dogs is still a problem, although recently the first dog-hair and dander specific allergen, Can f I, was isolated by Schou et al. (1991). These authors suggested that Can f I could be used as a marker allergen for future studies of the environmental exposure to dog-hair and dander allergens, and for the study of the relationship between exposure and sensitization.

The prevalence of dog allergy in unselected populations is reported to be 4–15%. Lelong et al. (1990) showed among a group of allergic children a frequency of sensitization of 17% to dogs. Vanto and Koivikko (1981) found in a study among 203 asthmatic children a relatively high prevalence (56%) of positive skin prick tests to dog dander.

5.4 FUNGI[1]

5.4.1 Occurrence

Among the approximately 100 000 known species of fungi, those of prime interest in indoor environments are moulds belonging to the Deuteromycetes

1 Text excerpted from ECA (1993).

or Fungi Imperfecti, but a few species in the Mucorales, Ascomycetes and wood-rotting Basidiomycetes, and some yeasts are also of relevance.

The majority of fungi are saprophytic, utilizing dead organic material for food. Providing temperature and moisture requirements are met, many species can utilize a wide range of organic materials. This ranges from plant or animal remains to materials such as cellulose, paint or stored products (Gravesen, 1979). Although some fungi will grow at 2–5°C and others at temperatures as high as 55–60°C (thermophiles), the majority of the fungi in indoor environments grow at 10–35°C. The water content of the substrate is the most critical factor in the development of fungi. The minimum water activity (a_W, the ratio of the vapour pressure above a substrate to the vapour pressure above pure water under the same conditions of temperature and pressure) needed for the growth of fungi on building related substrates varies from 0.75 to more than 0.98 for different mould species (Grant et al., 1989).

Unless scrupulously clean, indoor environments offer a wide variety of substrates for growth. Damp, non-living organic material can be quickly colonized (Burge, 1985). Condensation is the principal source of the moisture needed for growth of fungi on the internal surfaces of domestic dwellings. Besides superficial condensation, interstitial condensation within porous building materials such as concrete, brick and plaster, may provide a reservoir allowing fungal growth to continue in circumstances under which the surface would otherwise dry out. Interior dampness problems are also often related to construction faults, such as inadequate insulation or "cold-bridging", in combination with inadequate ventilation and/or the pattern of usage of buildings. Condensation and mould problems may also be encountered in "tight" buildings built to conserve energy, particularly when measures to prevent excessive generation of moisture are not taken, e.g. in cooking or laundering.

The prevalence of fungi shows wide seasonal differences (Gravesen, 1972; Wilken-Jensen and Gravesen, 1984). For most genera, the highest numbers in outdoor air are found during summer and autumn. During these seasons the outdoor air is the main source of fungi in indoor air. Recent studies indicate that the outdoor air spora influences the spora indoors, but the indoor air spora is not simply a reflection of the fungi in outdoor air (Fradkin et al., 1987; Verhoeff et al., 1992) (Table 5.3). Indoor sources of spores may also be present, e.g. growths on damp walls or in damp filters or drainage pans in HVAC systems.

5.4.2 Typical concentrations

A large number of species can be found in indoor air. The most common are likely to belong to the genera *Cladosporium*, *Penicillium*, *Alternaria*, *Aspergillus*, *Eurotium* and *Wallemia*. However, spores and other viable propagules

TABLE 5.3

Comparison (paired *t*-test) between the concentrations in CFU/m^3 (geometric mean and geometric standard deviation) of seventeen mould species in the indoor air of 18 Dutch homes and parallel outdoor samples (after Verhoeff et al., 1992)

Species	Indoors		Outdoors		T	P
	GM	GSD	GM	GSD		
Total number of CFU/m^3	669	2.5	941	2.4	–1.27	0.22
Aspergillus (total)	24	6.6	8	6.0	1.70	0.11
A. penicillioides	6	6.6	2	4.5	1.80	0.09
A. versicolor	4	5.4	2	2.3	1.69	0.11
Aureobasidium pullulans	1	2.2	3	4.4	–2.19	0.04
Botrytis cinerea	2	2.7	6	4.0	–2.54	0.02
Cladosporium (total)	245	2.8	501	3.0	–3.08	0.01
C. cladosporioides	65	5.5	72	8.4	–0.18	0.86
C. herbarum	59	7.4	224	3.8	–3.49	<0.01
C. macrocarpum	2	2.3	1	1.6	1.86	0.08
C. sphaerospermum	3	5.3	3	3.9	–0.03	0.98
Eurotium amstelodami	2	3.1	2	3.5	–0.45	0.66
E. herbariorum	4	4.3	6	4.2	–0.88	0.39
Penicillium (total)	154	3.6	65	4.4	2.04	0.06
P. aurantiogriseum	2	2.8	3	5.6	–0.72	0.46
P. brevicompactum	15	7.6	6	4.8	1.85	0.08
P. glabrum	6	12.1	2	4.0	1.34	0.20
P. jensenii	2	4.5	2	2.4	0.87	0.40
P. olsonii	8	8.4	3	3.3	1.88	0.08
Ramularia deusta	1	2.0	1	2.1	–0.35	0.73
Wallemia sebi	5	5.3	4	5.3	0.55	0.59

may only comprise 1–2% of the total number of fungal particles. The number of propagules or colony forming units (CFU)/m^3 varies widely, from less than 10 to more than 20 000, and may reach even as high as 400 000 in exceptional cases. Table 5.4 presents data from a study where airborne fungi were sampled in 27 homes in Toronto, Canada, during July–August, when the mean number indoors was 742 CFU/m^3 and outdoors 1131 CFU/m^3 (Fradkin et al., 1987).

The data in Tables 5.5 and 5.6 are from indoor air samples in 15 dwellings in England and 47 in Scotland obtained during three winters, from early November to early April, when the range was from less than 12 to 449 800 CFU/m^3 (Hunter et al., 1988)

TABLE 5.4

Fungal species in the air of indoor and outdoor environment during summer (after Fradkin et al., 1987)

Mould	Number of homes with indoor/outdoor ratio			CFU/m^3 (mean of 27 samples)	
	<1	1–2	>2	Outdoor	Indoor
Total (respirable fraction)	16	8	3	1131	742
Cladosporium	11	14	2	696	456
C. cladosporioides	16	8	3	334	211
C. sphaerospermum	19	5	3	232	111
C. herbarum	9	6	10	96	96
C. macrocarpum	10		10	34	30
Alternaria	19	4	2	124	52
A. tenuis	16	2	6	102	41
A. alternata	12	1	8	23	11
Epicoccum purpurascens	13	1	9	40	26
Candida spp.	8	1	9	45	19
Penicillium	13	1	12	79	108
P. chrysogenum	15	1	4	34	22
Other penicillia			14	<7	30
Aspergillus	5	1	14	45	22
A. fumigatus		1	9	23	11
Other species	15	6	5	102	59

Fungi in house dust also appear to be an important source of airborne spores in indoor environments (Burge, 1985). In house dust, species in the following genera are commonly isolated: *Cladosporium*, *Penicillium*, *Alternaria*, *Aspergillus*, *Eurotium*, *Mucor* and *Wallemia*. The number of CFU/g dust ranges from < 10^2 to 10^6 (see Table 5.7).

5.4.3 Health effects

In addition to fungi being implicated in cases of atopic allergic dermatitis, their causative role in individual cases of respiratory allergy is well-known. However, their overall significance in respiratory health is still debated. Allergic reactions to single or clustered spores and hyphal elements of fungi in air which have been reported include rhinitis, asthma and extrinsic allergic alveolitis (Tobin et al., 1987), although the last is most often associated with

TABLE 5.5

Range of numbers of fungal propagules (CFU/m^3) in the air spora of different rooms in dwellings in the London area and Central Scotland (after Hunter et al., 1988)

Mould growth	n	Range	Median	Interquartile range	Modal class*
Living room					
Entirely absent	177	<12–23070	236	118–463	<12–200 (44)
Present in other rooms only	144	<12–48000	360	165–759	<12–200 (31)
Present in the room itself	61	424–21790	2673	1165–4944	1001–2000 (21)
Bedroom					
Entirely absent	158	<12–8950	177	95–353	<12–200 (55)
Present in other rooms only	103	12–6300	247	118–522	<12–200 (46)
Present in the room itself	163	35–449800	1165	406–5615	<12–200 (15)
Kitchen					
Entirely absent	9	12–1170	212	115–507	<12–200 (44)
Present in other rooms only	7	12–1890	180	59–230	<12–200 (57)
Present in the room itself	41	71–20320	2296	1107–5862	1001–2000 (23)
Bathroom					
Present in other rooms only	12	212–19990	829	598–1036	1001–2000 (33)
Present in the room itself	13	71–19070	671	388–5615	201–400 (23)

*Percentage of counts in modal class.

high levels of actinomycete or fungal spores (10^6–10^{10} spores/m^3 air) released from mouldy agricultural materials and causing occupational diseases such as farmer's lung. For patients with respiratory allergy, prevalence of allergy to fungi ranges from 2 to 30% (Gravesen 1979). A multi-centre study in Denmark revealed that the prevalence of fungal allergy in an atopic population was 8% in adults and 23% in children, 44% of the children having asthma and 23% rhinitis (Osterballe et al., 1981).

Recent epidemiological studies have observed a strong association between reported dampness and mould in houses and respiratory symptoms in children (Strachan and Elton, 1986; Martin et al., 1987; Strachan, 1988; Brunekreef et al., 1989; Platt et al., 1989; Dales et al., 1991b) and adults (Dales et al., 1991a) occupying the houses. Although Strachan (1988) and Strachan et al. (1990) have cautioned against reporting bias among participants in surveys and highlighted the need for further valid and objective data, the epidemiological

TABLE 5.6

Viable fungi isolated most frequently from air of 47 dwellings in Central Scotland (after Hunter et al., 1988)

Type	Percentage frequency in:	
	Dwellings	Samples
Penicillium spp.	95.7	90.2
Yeasts	93.6	82.1
Cladosporium spp.	88.7	73.7
Aspergillus spp.	74.5	52.0
Ulocladium spp.	61.7	18.2
Geomyces pannorum	56.7	20.3
Sistotrema brinkmannii	51.1	55.5
Aureobasidium pullulans	46.8	13.8
Acremonium strictum	42.6	4.2
Mucor spp.	38.3	8.4
Phoma herbarum	36.2	6.1
Oidiodendron griseum	23.4	4.4
Scopulariopsis brevicaulis	21.4	3.6
Alternaria alternata	17.0	2.6
Cunninghamella elegans	14.9	2.8
Stachybotrys atra	12.8	4.5
Fusarium spp.	10.6	1.0
Botrytis cinerea	8.5	1.3
Papulospora spp.	8.5	0.9

evidence appears to be robust.

The literature on the relationship between inhaled fungal spores and induction of respiratory allergy is also still inadequate and controversial in many respects. In the last ten years there has been considerable progress in the identification, purification and characterization of at least some allergens produced by fungi, especially by *Alternaria* (Alt a I) and *Cladosporium* (Cla h I). Nevertheless, much needs to be done in standardizing allergens used in testing; there is still a need for highly purified and standardized extracts (Burge, 1985).

Although, traditionally, allergologists have assumed that mould-induced asthma is entirely due to an allergic reaction, it is highly likely that toxigenic properties of fungi are involved as well as allergenic properties. In the case of pathogenic species of genera such as *Aspergillus*, both properties probably

TABLE 5.7

Frequency of viable airborne and dust–bound genera of microfungi in schools and offices (air: n = 143; dust: n = 98) (after Gravesen et al., 1986)

	Air	%	Dust	%
Schools	*Penicillium*	81	*Penicillium*	81
	Cladosporium	64	*Fusarium*	74
	Aspergillus	52	*Alternaria*	56
	Alternaria	29	*Cladosporium*	52
	Rhodotorula	24	*Mucor*	40
	Trichosporon	24	*Trichosporon*	30
	Arthrinium	19	*Aspergillus*	25
	Mucor	17	*Rhodotorula*	25
Offices	*Penicillium*	66	*Penicillium*	78
	Cladosporium	57	*Alternaria*	76
	Aspergillus	52	*Aspergillus*	55
	Yeasts	24	*Cladosporium*	45
	Trichosporon	15	*Fusarium*	45
	Alternaria	13	*Trichosporon*	45
	Rhodotorula	13	*Yeasts*	33
	Paecilomyces	9	*Mucor*	29

contribute very significantly to the lung damage which results from invasion of lung tissue (mycosis) (Tobin et al., 1987). Recent Canadian studies indicate that a non-allergic mechanism may account for the increased respiratory symptoms among occupants of damp houses (Dales et al., 1991a,b).

Two types of component in moulds which may be involved in any non-allergic mechanisms are 1,3-β-glucan and mycotoxins. The 1,3-β-glucan is a component of the walls of fungal hyphae and spores, and extracted protein-free β-glucan has been shown to act as an inflammatory agent. It has been implicated in increased reporting of mucous membrane irritation and fatigue by occupants of buildings in which complaints about indoor air quality were made (Rylander et al., 1991).

Mycotoxins are produced by a wide range of moulds and are located in hyphae and spores, often being present in particularly high concentrations in the latter. They are secondary metabolites (Miller, 1990) with molecular weights generally greater than 200, but are considerably smaller than allergens. It is well established that ingested mycotoxins can cause illness and death in humans and animals, but it has also been demonstrated in experi-

mental animal studies that inhalation challenge with trichothecenes is 20–50 times more hazardous than intravenous injection (Miller, 1990). The trichothenes produced by *Stachybotrys atra* and *Fusarium* spp., and patulin and penicillic acid produced by a number of penicillia, demonstrate acute toxicity to pulmonary alveolar macrophages (Sorenson, 1989). Trichothecenes and a number of other mycotoxins are also immunosuppressive. Although there is no definitive proof in humans, it appears distinctly possible that inhalation of high concentrations of mould spores containing these toxins may deleteriously affect macrophage functions such as phagocytosis of living and non-living particles in the lung, and therefore affect respiratory health. The effects on the immune system could also compromise health by reducing resistance to other micro-organisms, perhaps resulting in chronic health problems such as those encountered in a Chicago house where *S. atra* had colonized ducts, insulation and structural materials (Croft et al., 1986). Other toxigenic moulds which may be prevalent indoors include *Aspergillus versicolor*, *Penicillium* spp. (such as *P. brevicompactum* and *P. viridicatum*) and *Phoma* spp., or more rarely *Paecilomyces variotii* and *Trichoderma viride*.

Although toxicological data for many mycotoxins are incomplete, particularly with respect to the dermal and inhalation routes, mycotoxins in airborne particles must certainly be viewed as potentially hazardous factors present in airborne particulates in homes and non-industrial environments. Considerably more research effort will be needed before their real impact on human health can be fully assessed, however (Miller, 1990; Flannigan et al., 1991).

A further factor which may be involved is the complex mixture of volatiles which are produced by fungi and are frequently evident as "mouldy smells". A large number of volatiles have been identified. They occur as complex mixtures of alcohols, esters, aldehydes, various hydrocarbons and aromatics. There is considerable variation in the production of volatiles, even between closely related genera or species. The volatiles 1-octen-3-ol, 2-octen-1-ol and 1,10-dimethyl-9-decalol (geosmin) may be prominent among those which account for the mouldy odours so often encountered in damp houses. According to Samson (1985), some individuals do not react to these, some become nauseous and others may be quite ill. Tobin et al. (1987) have indicated that acute respiratory responses may range from a feeling of stuffiness to wheeze. Low-order toxicity in experimental animals has been demonstrated for 1-octen-3-ol and various other volatiles produced by moulds (Sorenson, 1989). At present, however, there is not sufficient information on which an objective assessment of the health impact of microbial volatiles relative to that of volatiles from other sources (Miller et al., 1988) can be based.

Cases of invasive disease which can be attributed to exposure to moulds in homes and non-industrial workplaces are likely to be rare. However, some

fungi which grow saprophytically in buildings are also opportunistic pathogens. In exceptional cases, pathogens such as *Aspergillus fumigatus* may invade the lung tissue of debilitated individuals, especially immuno-compromised patients, and become systemic. In such patients, the outcome is most often fatal.

5.5 BACTERIA[1]

5.5.1 Occurrence

Although survival of bacteria in air has been studied experimentally, the determinants are still rather obscure. Air is an extreme environment for bacteria, where their survival is limited by environmental stress. Generally speaking, low temperatures permit microbial survival. Bacterial genera differ in their ability to withstand different humidities. For example *Escherichia coli* survives best at high humidities (up to 90%), whereas *Serratia marcescens* and staphylococci survive best at a relative humidity below 40% (Nevalainen, 1989).

In indoor air, the main sources of bacterial aerosols are usually humans and animals, but bacterial aerosols may also be created by disturbing previously settled dust. Furthermore, humidifiers and drainage fans in HVAC systems are potential sources of airborne bacteria.

5.5.2 Typical concentrations

There is very little published work on the numbers and types of bacteria present in the air of houses, but the numbers of viable bacteria are often greater than those of airborne fungi. A century ago, the highest counts obtained by Carnelley et al. (1887) in crowded Scottish homes was approximately 120 000 viable bacteria/m^3 air, but in a more recent investigation the maximum was 22 000/m^3 (Flannigan et al., 1989). Nevalainen et al. (1988) isolated 60–12 200 viable bacteria/m^3 air, including 0–154 actinomycetes/m^3, from Finnish houses which were damp/mould-affected or the subject of building-related health complaints. However, similar levels were obtained in non-problem houses. There are wide differences in numbers of airborne bacteria in other indoor environments also (Table 5.8), but they are usually considerably higher than levels outdoors (Table 5.9). However, compared with an outside maximum of 652 bacteria/m^3, Austwick et al. (1989) recorded an indoor maximum of 512/m^3 in air-conditioned offices.

The bacteria in the indoor air of houses are dominated by micrococci

1 Text excerpted from ECA (1993).

TABLE 5.8

Numbers of viable bacteria in homes and non–industrial premises in France (after Mouilleseaux and Squinazi, 1991)

Location	n	CFU/m^3 (pct)		
		5 pct†	50 pct	90 pct
Air conditioned offices*	918	95	280	900
Hospitals**	50	60	260	860
Class rooms (empty)**	22	300	1050	4900
Class rooms (active)**	118	500	2600	8300
Day care centres**	397	1000	5200	16000
Old homes**	104	1750	4625	14000
Outdoor	296	113	275	775

*Slit sampler.
**RCS sampler.
† Percentile.

(Flannigan et al., 1989, 1991; Nevalainen, 1989), with *Micrococcus luteus* and *Staphylococcus epidermidis* (associated with shed skin scales) likely to be the most prominent, and *Staph. aureus* less abundant (Flannigan et al., 1989). In contrast to outdoor air, where *Pseudomonas* spp. predominate (and micrococci are a minor component), within Finnish houses *Pseudomonas* spp. are much less common (Table 5.10). However, it has also been reported that in houses in Scotland pseudomonads are the next most abundant category to micrococci, with Gram-negative oxidase-positive cocci following (Flannigan et al., 1989). A wide range of other bacteria, including actinomycetes, may be found in houses. A number of these have also been isolated from surfaces and from mould patches on walls, where micrococci and pseudomonads are the most frequently encountered types. A similar range of bacteria has been found in aerosols from humidifying and air-conditioning equipment or in the air of "sick" office buildings (Austwick et al., 1986, 1989), e.g. *Enterobacter*, *Acinetobacter*, *Alcaligenes*, *Bacillus*, *Flavobacterium*, *Micrococcus*, *Pseudomonas* (including *Ps. aeruginosa*), *Staphylococcus aureus* and *Staphylococcus epidermidis*, *Streptomyces* and various thermophilic actinomycetes and coryneforms.

Table 5.11 shows detection frequencies of bacteria resulting from an Italian investigation of 10 naturally ventilated schools (3 nurseries, 1 kindergarten, 3 primary schools) (Maroni et al., 1993). Indoor concentrations of bacteria were found to be higher than outdoor and distributed according to occupant density.

One category of bacterium of particular importance is *Legionella pneumo-*

TABLE 5.9

Levels of viable airborne bacteria (geometric mean (GM), geometric standard deviation (GSD) and range) in suburban houses and in outdoor air during the first three years of occupancy (after Nevalainen, 1989)

Group of samples	n	Bacteria levels CFU/m^3		
		GM	GSD	Range
Total 1986–88				
indoors	183	550	1.32	0–11900
outdoors	89	110	1.38	2–2200
Winter 1986				
indoors	40	140	1.45	0–2400
outdoors	17	16	1.07	0–70
Spring 1986				
indoors	36	680	0.91	100–5900
outdoors	18	145	1.69	2–3100
Fall 1986				
indoors	36	520	0.90	30–2700
outdoors	18	120	1.03	20–850
Fall 1987				
indoors	36	1200	0.80	200–11900
outdoors	18	150	0.75	30–560
Fall 1988				
indoors	35	110	1.12	20–11800
outdoors	18	130	1.14	30–2200

phila. *Legionella* spp. are ubiquitous bacteria in natural waters, and the commonly used methods of water treatment do not eliminate them. Thus they often enter buildings via the potable water system, probably in far too small numbers to cause infection. Amplification has been shown to take place in many locations in buildings such as cooling towers, domestic water heaters, water pipes, shower fittings and whirlpools and also in some indoor equipment such as humidifiers. The optimum temperature for growth under laboratory conditions is 36°C, but growth will take place throughout the range 20–45°C; at temperatures above 60°C legionellae do not survive. The most important of these bacteria is the pathogen *L. pneumophila*. Reported sources of *L. pneumophila* in different outbreaks of these diseases have included cooling towers, humidification equipment and domestic hot-water systems. The presence of algae, protozoa or sludge also promotes the growth of *Legionella* spp. Trans-

TABLE 5.10

Bacteria identified in the indoor air of houses; after Nevalainen (1989) and Flannigan et al. (1989, 1991)

Gram-positive cocci	*Aerococcus viridans*(*)
	Micrococcus spp. (*M. luteus*(*), *M. roseus, M. varians* and *M. viridans*)
	Staphylococcus aureus(*) and *Staph. epidermidis*(*)
	Streptococcus spp.
Gram-positive rods	*Arthobacter* sp.
	Bacillus spp.(*) (*B. licheniformis, B. megaterium* and *B. subtilis*)
	Corynebacterium spp.(*)
	Erysepelothrix sp.(*)
	Kurthia sp.
	Lactobacillus sp.
	Mycobacterium sp.
Gram-negative rods	*Achromobacter* spp.
	Acinetobacter sp.(*)
	Aeromonas hydrophila(*)
	Agrobacterium sp.
	Alcaligenes denitrificans
	Enterobacter agglomerans
	Flavobacterium sp.(*)
	Klebsiella spp.(*)
	Moraxella lacunata(*)
	Proteus sp.
	Pseudomonas spp.(*) (*Ps. fluorescens, Ps. luteola, Ps. maltophilia*, Ps. mendocina, *Ps. oryzihabitans, Ps. paucimobilis, Ps. pickettii, Ps. pseudomallei, Ps. putida, Ps. stutzeri* and *Ps. vesicularis*)
Actinomycetes	*Actinomyces ochroleucus*
	Streptomyces spp. (*S. lepmarii, S. flavogriseus, S. parvus, S. rishiriensis, S. tetanusenus, S. roseus, S. wilkmorei* and *S. nitrosporeus*)

(*) Also detected on surfaces by swabbing or by contact plates.

mission is most likely to occur through inhalation of an aerosol created by, for instance, a shower tap or a cooling tower. In the latter case the aerosol may reach people directly or via the ventilation system.

TABLE 5.11

Frequency of detection and abundance of viable bacteria in air samples of schools (after Maroni et al., 1993)

Identified bacteria	Nurseries and kindergardens		Primary and secondary schools	
	Frequency of detection (%)	Geom. mean (CFU)	Frequency of detection (%)	Geom. mean (CFU)
Staphylococcus spp.	87	13	83	14
Micrococcus spp.	69	8	75	6
Diptheroids	69	7	71	12
Streptococcus non-haemolytic	19	11	25	11
Streptococcus α-haemolytic	12	2.5	12	19
Staphylococcus aureus	12	3.3	8	3
Other Gram-pos. cocci	0	n.d.	4	115
Gram-pos. rods (non-spore-forming)	25	7	62	3
Acinetobacter lwoffi	37	4	46	13
Acinetobacter calcoaceticus	25	2	8	1
Pseudomonas spp.	12	4	25	2.6
Flavobacterium spp.	6	2	12	1.5
Gram-neg. rods not in Enterobacteriaceae	31	4	29	4

5.5.3 Health effects

As mentioned earlier, together with fungi, bacteria (including their antigens and endotoxins) in humidifiers are implicated in humidifier fever, a disease with elements of both toxic and allergic manifestations. The cell-wall lipopolysaccharide (LPS) fractions of Gram-negative bacteria are known as endotoxins, and the symptoms provoked by respiratory challenge with these endotoxins, or the whole cells, are very similar to those found in typical acute episodes of humidifier fever (Rylander, 1986). It is known that LPS enhances IgE-mediated release of histamine in basophil cells which has been induced by housedust mite or birch pollen allergen (Clementsen et al., 1990). Further, dead cells of Gram-positive bacterium *Staphylococcus aureus* also potentiate this release. It has therefore been suggested that inhaled Gram-negative and Gram-positive bacteria could aggravate asthma attacks induced by airborne bioparticles (Clementsen et al., 1990).

Infrequently, bacteria in buildings may also be the cause of extrinsic allergic alevolitis in occupants. Bacteria in the air of houses or offices reported to have caused extrinsic allergic alveolitis among occupants are *Bacillus subtilis* and *Pseudomonas aeroginosa*, and the thermophilic actinomycetes *Faenia rectivirgula* (*Micropolyspora faeni*) and *Thermoactinomyces vulgaris* (Flannigan et al., 1991).

Occasionally, bacteria which cause important infectious diseases and are present in droplet nuclei from individuals shedding pathogenic agents, e.g. *Mycobacterium tuberculosis*, can be rapidly dispersed throughout an enclosed environment via a recirculation system (Riley 1979). Of course, other medically important bacteria such as *Legionella* may also be transmitted in aerosols and can cause respiratory problems.

Of the 30 species of *Legionella* which have been described, *L. pneumophila* is epidemiologically the most important; of 14 serogroups within this species, *L. pneumophila* serogroup 1 is the most important. This Gram-negative bacterium causes two types of disease, Legionnaires' disease and Pontiac fever. Legionnaires' disease is a serious type of pneumonia, which takes its name from a serious outbreak at a meeting of the American Legion. The incubation period ranges from 2 to 13 days (average 5–6 days). Legionnaires' disease may affect other organs as well and requires special antibiotic treatment. The disease has a considerable mortality rate and appears mostly as sporadic or hyperendemic cases. Epidemics are rare. The frequency of sporadic cases is estimated to be 2% of all hospitalized pneumonias in the United Kingdom, and 10% of community-acquired pneumonias in France and Germany (WHO, 1990). Less than 5% of those exposed appear to develop the illness; in 10–15% of these the illness is fatal (Dennis, 1990).

Pontiac fever, named after an outbreak in 1968 in Pontiac, USA, is caused by a number of *Legionella* spp., is milder and appears as a non-pneumonic fever. The incubation period ranges from several hours up to 48 hours. The illness resolves spontaneously in 2–5 days. No fatal cases of Pontiac fever have been reported. This disease mainly appears as epidemics, and 95% of those exposed to aerosols will become ill (Dennis, 1990). However, the incidence of Pontiac fever in the general population is unknown and reports of sporadic cases are unlikely to be made even if they are recognized (WHO, 1990).

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PART II
Health Effects of Indoor Air Pollution

This part of the book presents and discusses the health effects caused by indoor pollution. After a general introduction on the methods to assess exposure and effects related to indoor pollution, different chapters present the effects on the respiratory system, allergic reactions, cancer and effects on reproduction, irritative effects on the skin and mucous membranes, sensory effects and other effects on the nervous system, effects on the cardiovascular system and other specific effects. Each chapter is organised in a similar way, showing the following sequence of subjects for each effect: principal agents and sources, evidence of the causal relation between indoor pollution and the effect under discussion, susceptible groups, public health relevance, and methods for assessment of the effects.

The content of these chapters has been derived, with minor changes, from a document prepared by a working group of the European Collaborative Action "Indoor Air Quality and its Impact on Man" and published in the Indoor Air Journal (Vol. 2, No. 1, March 1992). The group was chaired by Marco Maroni and included B. Berglund, B. Brunekreef, H. Knöppel, T. Lindvall, L. Mølhave and P. Skov.

The current version of the text has been prepared by B. Brunekreef, with the assistance of G. Muzi, L. Mølhave, and B. Berglund.

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Chapter 6

General Aspects of Assessment of Human Health Effects of Indoor Air Pollution

6.1 ASSESSMENT OF HUMAN EXPOSURE TO INDOOR AIR POLLUTION

Human exposure to indoor air pollutants is difficult to quantify because of the many environmental characteristics involved. Pollution levels in one building may be quite different from those in another, depending on the presence and usage of sources of pollutants and on the ventilation habits. Also, many techniques routinely used for measuring ambient air pollution are not suitable for indoor surveys because of cost, bulkiness, noise or amount of air displaced.

A range of miniaturised measuring devices has been developed for indoor use. However, most of these techniques measure average concentrations over several hours or even days, which limits their use in studies of pollutants with acute effects — such as carbon monoxide (CO) — or with effects believed to be related more to short-term peak levels than long-term averages, such as nitrogen dioxide (NO_2). Other pollutants such as environmental tobacco smoke (ETS) and some volatile organic compounds (VOC) are of interest for their suspected chronic effects related to long-term exposure. For such pollutants measuring techniques which average concentrations over extended periods are sufficiently informative. For some substances techniques have been developed for personal monitoring as well, which require subjects to carry the equipment with them wherever they go. In practice, personal monitoring has proved to be most feasible for substances that can be measured with so-called "passive" monitors. These rely on diffusion of pollutants to an absorbing surface without the need of pumps and associated equipment.

If measurement of exposure is not feasible, a modelling approach is sometimes useful. Existing models for estimating human exposure make use of known or estimated time usage patterns for occupants, source presence, strength and use, and other relevant data. They need to be validated in studies in which exposure is actually measured. The advantage of modelling rather than measuring exposure is that modelling can usually be done at a fraction of the cost.

For some pollutants, biological monitoring offers a viable alternative to either measuring or modelling exposure in air. In biological monitoring, substances (or their metabolites) are measured in, for example, exhaled air, blood or urine. If the relationship between the levels in biological media and environmental levels are adequately characterised, biological monitoring offers a unique picture of integrated exposure of humans to pollutants.

6.2 HUMAN SUSCEPTIBILITY TO POLLUTANTS

The human race is extremely diverse, and it is no wonder that there are differences in susceptibility to pollutants between equally exposed individuals. Variations in susceptibility may range from gradual differences to the very dramatic differences shown, for example, by persons sensitised to certain allergens. When there are no specific mechanisms or factors underlying differences in susceptibility these variations in susceptibility are believed to follow a roughly normal distribution. Many specific mechanisms or factors have, however, been shown or suggested to be associated with sometimes large differences in susceptibility. Among these are genetic factors, age, gender, nutritional status, pre-existing disease, allergy and asthma, and tobacco smoking (Brain et al., 1988).

(a) Several genetic factors have been shown to be associated with the development of chronic obstructive pulmonary disease (COPD). The enzyme alpha-1-antiprotease and trypsin, for example, inhibits proteolytic enzymes; deficiency of this enzyme is heritable, and the incidence of emphysema has been shown to be much greater in patients who are homozygous for this particular deficiency (Hutchinson, 1990).

(b) Marked differences in mortality due to ozone exposure have been shown to exist between dogs and some rodent species, presumably due to a more efficient removal of ozone in the upper respiratory system of the dogs (Lippmann, 1989).

(c) Young children, infants and the foetus are known to be more susceptible to adverse effects of lead at a given blood lead level than adults (Lin-Fu, 1973).

(d) Diets deficient in selenium or vitamin E have been shown to increase lung damage due to ozone exposure in laboratory animals (Elsayed et al., 1983, 1988).

(e) Asthmatics are known to be more susceptible to given concentrations of sulphur dioxide (SO_2) and nitrogen dioxide (NO_2) than non-asthmatics (Orehek et al., 1976; Sheppard et al., 1980).

(f) Smokers suffer from COPD more than non-smokers, and to the extent that COPD patients are more susceptible to pollutants than others, smoking causes some people to be more susceptible. It is not clear to what extent

smokers who do not have COPD are more susceptible to effects of other pollutants than non-smokers. Smoking and asbestos exposure have been shown to act synergistically in the causation of lung cancer (Selikoff et al., 1979) although smokers may be predisposed to various forms of occupational asthma (e.g. formaldehyde) (Venables et al., 1988; Zetterstrom et al., 1981).

These are but a few examples of differences in susceptibility to pollutants which may exist between humans with different characteristics. In the separate paragraphs, specific examples will be given of groups thought to exhibit increased susceptibility to the health effect under consideration.

6.3 METHODS OF STUDYING HEALTH EFFECTS

Methods of studying health effects of indoor pollutants can be grouped into three broad categories:

(a) Human studies, subdivided into observational and experimental studies. Epidemiological studies of pollutants are mostly observational, i.e. the investigator has no means of experimentally arranging the exposure of humans to pollutants, or of allocating subjects to exposed and unexposed groups. Critical issues are therefore the validity and precision of exposure assessment, and the control for confounding factors in these studies. Recent developments have stressed the importance of reducing exposure misclassification, and of studying restricted, well defined, homogeneous populations to address these issues. The main advantage is that humans are studied under realistic conditions of exposure. By themselves, observational epidemiological studies are not usually sufficient to support causality of an observed association, so that additional information is needed from other types of studies. Experimental studies are among these; however, these are only suitable for studying moderate, reversible, short-term effects in persons who are healthy or only moderately ill. Their main advantage is that exposure conditions and subject selection are under the control of the investigator.

(b) Animal studies, which can be subdivided into a number of categories depending on the exposure duration (acute, subchronic, chronic) or end-point (morbidity, mortality, carcinogenicity, irritation, etc.). Here, the investigator has full control over exposure conditions and health effects studied. However, the principal limitations are that extrapolation from the studied animal species to man is always necessary. Also, while in human populations health effects with low incidences are often of interest (e.g., specific cancers), it is not feasible to study very large groups of animals to detect these low incidences. In practice, therefore, animal experiments are often carried out using very high experimental doses to compensate for the relatively small number of

animals used and as a consequence, an additional extrapolation from high to low doses is also often necessary.

(c) *In vitro* studies, in which effects of pollutants on cell or organ cultures are studied. These studies have the advantage that they are less costly than animal studies, and that results can generally be obtained in a shorter time. They are useful for studying mechanisms of action, but it is usually impossible in a quantitative way to predict effects on whole organisms from their results.

In the following chapters, additional remarks will be made on specific methods used to study the health effects under consideration.

6.4 SYNDROMES RELATED TO INDOOR AIR QUALITY

6.4.1 "Sick building syndrome"

Since the early 1970s, numerous outbreaks of work related health problems have been described among employees in buildings or offices not directly contaminated by industrial processes. Two broad categories can be distinguished: those characterised by a generally uniform clinical picture for which a specific cause has been identified, and those in which affected workers reported non-specific symptoms occurring only during the time when they were at work.

The former episodes have been defined "building-related illness" (BRI), the latter, "sick building syndrome" (SBS). Symptoms reported in SBS have typically included mucous membrane and eye irritation, cough, chest tightness, fatigue, headache and malaise.

Outbreaks without an identifiable cause have frequently occurred in new, sealed office buildings and have for that reason also been called the "tight building syndrome" (TBS).

Essential for SBS are the concepts of comfort, well-being and air quality. Comfort or well-being refers to a status of optimal physical conditions for the body. Acceptable indoor air quality is described as air in which there are no known contaminants at harmful concentrations and with which a substantial majority (e.g., 80% or more) (ASHRAE, 1989) of the people exposed do not express dissatisfaction.

Thus, "sick building syndrome" is a term used to describe the reduced comfort and health status of occupants in a particular building or part of it where the occupants complain about indoor air quality and manifest symptoms which they assign to the reduced quality of the air.

A recent document by WHO (WHO, 1989a) indicates SBS as a reaction to the indoor environment among a majority of the occupants whose reactions

cannot be related to obvious causes such as excessive exposure to a known contaminant or a defective ventilation system. The syndrome is assumed to be caused by a multifactorial interaction of several exposure factors involving different reaction mechanisms.

The symptoms of SBS are mainly reports of discomfort or the feeling of being "less than well". Most of the symptoms are not usually accompanied by independently observable signs, so that, generally, self-reports are the only means by which incidence and prevalence can be determined.

The criteria for the definition of SBS are summarised in WHO (WHO, 1989a). More details on the SBS can be found in a monograph prepared by a group of experts for the Committee of the COST 613 (CEC, 1989), and the reader is referred to that document for further information.

6.4.2 "Building-related illnesses"

BRI is a term referring to illness brought on by exposure to the building air, where symptoms of a diagnosable illness are identified and can be directly attributed to environmental agents in the air.

In outbreaks of BRI, a wide spectrum of causative factors has been implicated: immunologic sensitizing agents, infectious agents, specific air contaminants, and environmental conditions, such as temperature and humidity (Finnegan et al., 1986; Samet et al., 1988).

Further information on BRI will be found in Chapters 19 and 21 which deal specifically with all the forms of BRI.

6.4.3 Multiple chemical sensitivity or "chemical hypersensitivity syndrome"

Multiple chemical sensitivity is a chronic (i.e. continuing for more than three months) multisystem disorder, usually involving symptoms of the central nervous system and at least one other system. Affected persons are frequently intolerant to some foods and they react adversely to some chemicals and to environmental agents, singly or in combination, at levels generally tolerated by the majority. Affected persons have varying degrees of morbidity, from mild discomfort to total disability. Upon physical examination, the patient is normally free from major abnormal objective findings. Although abnormalities of complement and lymphocytes have been recorded, no single laboratory test, including serum IgE, is consistently altered. Improvement is associated with avoidance of suspected agents and symptoms recur with re-exposure (American College of Physicians, 1989; Marchant, 1992).

The symptoms are non-specific and multiple, e.g., behavioural changes,

fatigue, depression, psychiatric disorders, musculoskeletal, respiratory, genitourinary, and mucous membrane irritation complaints. The history arises after exposure or believed exposure to environmental chemicals or toxins. Because the clinical pictures are various and lack a fundamental scientific basis it is not recognized as a disease entity by clinicians.

There are no agreements on aetiology or prognosis and fundamental features to establish cause/effect between MCS and allergens or toxins are lacking.

For a further discussion on environmental hypersusceptibility the reader is referred to Chapter 21, Section 21.10.

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Chapter 7

Effects of Indoor Air Pollution on the Respiratory System

As the human respiratory system is the organ directly affected by air pollution, the potential respiratory health effects of indoor and outdoor air pollution have been widely investigated. This section is restricted to non-carcinogenic and non-allergic effects on the lower airways (below the larynx). Allergic effects are considered in Chapter 8; carcinogenic effects are treated in Chapter 9; and upper respiratory effects are treated in Chapter 10.

7.1 RESPIRATORY HEALTH EFFECTS ASSOCIATED WITH EXPOSURE TO INDOOR AIR POLLUTION

Several effects on the respiratory system have been associated with exposure to IAP. These include acute and chronic changes in pulmonary function, increased incidence and prevalence of respiratory symptoms, acute worsening of pre-existing respiratory symptoms, and sensitisation of the airways to allergens present in the indoor environment. Also, respiratory infections may spread in indoor environments when specific sources of infectious agents are present, or simply because the smaller indoor mixing volumes allow infectious diseases to spread more easily from one person to the next. The latter mechanism is particularly operative in schools, nursery schools, etc.

Observed changes in pulmonary function due to exposure to, e.g., tobacco smoke in the home, have mostly been due to acute or chronic airway narrowing leading to obstruction of air flow. This is measured as a reduction in the quantity of air that can be exhaled in one second after inspiration (FEV1), and reductions in the various measures of air flow such as Peak Expiratory Flow (PEF), Maximum Mid Expiratory Flow (MMEF), and Maximum Expiratory Flow at x% of Forced Vital Capacity (MEFx) (Brunekreef et al., 1985; White et al., 1980). In growing children, it has also been suggested that lung development may be temporarily impaired by exposure to IAP (Berkey et al., 1986; Tager et al., 1983).

Asthma, manifested by attacks of excessive airway narrowing leading to shortness of breath and wheezing, can be caused by exposure to allergens in buildings, but it has also been associated with exposure to substances such as nitrogen dioxide and environmental tobacco smoke (ETS). Bronchitis, manifested in inflammatory changes in the airways as well as mucus hypersecretion, has been linked to high levels of ambient air pollution in the past (Firket, 1931; Lawther et al., 1970; Ministry of Health, 1954; Schrenk et al., 1948) and to exposure to ETS in the home in recent studies (Colley et al., 1974; Kalandidi et al., 1987). Respiratory symptoms which have been associated with exposure to indoor air pollutants are symptoms mostly related to the lower airways such as cough, wheeze, shortness of breath and phlegm. The distinction between acute and chronic changes in respiratory symptoms is not always clear; this is partly a matter of the methods used to investigate them (see below).

In contrast to the occurrence of chemical pollutants in indoor air, attention to which has grown considerably over the past two decades, the role of infectious agents in indoor air has been known for a long time. Infectious agents may be involved in the inflammatory conditions rhinitis, sinusitis, conjunctivitis and sinusitis, in pneumonia, and in asthma.

7.2 PRINCIPAL AGENTS AND SOURCES

Combustion products, ETS and biological contaminants are the main agents associated with respiratory health effects indoors. Of the combustion products, nitrogen dioxide (NO_2) has been widely investigated, but it has not been unequivocally shown that it actually causes respiratory health effects in the concentration range in which it is normally encountered indoors. NO_2 concentrations are elevated in homes where unvented gas appliances are used (Fischer et al., 1986; Spengler et al., 1983a). Peak concentrations of up to several hundred $\mu g/m^3$, which is well above the 1987 WHO health guidelines, are known to occur relatively frequently in homes. Non-intended usage of unvented gas appliances (heating, clothes drying) may increase concentration levels for extended periods of time.

In homes where unvented kerosene heaters are in use, elevated levels of sulphur dioxide (SO_2) may also occur, when sulphur containing fuels are used. Especially in climate zones with temperate winters where homes may not be equipped with permanent heating systems, unvented kerosene heaters may be a significant source of indoor pollution (Leaderer, 1982; Samet et al., 1987a; Traynor et al., 1985; Wardlaw, 1993).

In industrialised countries, ETS is probably the most important indoor cause of non-carcinogenic adverse effects on the respiratory system. It has been shown that long-term average concentrations of suspended particulate matter in indoor

air are significantly higher in smokers' homes than in homes where the inhabitants do not smoke. In contrast, levels of carbon monoxide (CO) and NO_2 are not markedly increased in smokers' homes, indicating that for these substances, other sources are predominant. Effects of ETS on respiratory symptoms and pulmonary function of children have been observed by many investigators (Berkey et al., 1986; Fielding et al., 1988; Samet et al., 1987a, 1988; Tager et al., 1983). Effects on adults have been demonstrated less convincingly (Kentner et al, 1984; Samet et al., 1987b).

Contaminated humidifiers or humidification sections of HVAC (Heating, Ventilating and Air Conditioning) installations are often sources of infectious agents (such as Legionella pn.). Infectious agents may also proliferate in other locations which, generally speaking, meet their needs in terms of substrate, temperature and humidity. HVAC installations are found to contain and spread biological contaminants quite frequently, due to poor design, operation and/or maintenance (Finnegan et al., 1986). In homes, sources of biological contaminants are more often formed by damp areas on walls and floors (Saltos et al., 1982).

7.3 EVIDENCE LINKING INDOOR POLLUTION TO EFFECTS ON THE RESPIRATORY SYSTEM

The evidence that links indoor exposure to combustion products, ETS and biological agents to non-carcinogenic effects on the respiratory system stems to a large extent from epidemiological studies.

Exposure to ETS has been shown by many investigators to be related to lower respiratory illness in infancy, and to the development of chronic respiratory symptoms in older children. In addition, pulmonary function in exposed children is reduced compared to pulmonary function in unexposed children (Berkey et al., 1986; Tager et al., 1983, 1987). To some extent, this may be a carry over from effects on the foetal lungs caused by smoking in pregnancy, but there is also some evidence which suggests that development of lung function of exposed children is actually lower than that of unexposed children. Among adults, evidence for non-carcinogenic effects of exposure to ETS has been less unequivocal. However, there are some studies which suggest that non-smoking women who have been married to a smoker for a long time, have a reduced lung function compared to unexposed women (Gillis et al., 1984).

Of the combustion products, NO_2 has been studied widely in the past decade. Many studies have used proxy measures of exposure rather than actual NO_2 measurements by contrasting populations living in homes with unvented gas cooking appliances with populations living in homes equipped with

electric cookers. It has been shown that this may lead to a sizable misclassification of exposure to NO_2 (Shy et al., 1978; Spengler et al., 1983b). Some studies have used indoor and personal monitoring of NO_2 instead, usually by employing diffusion samplers which require exposure times of several days to a week. As a result, only long-term average exposure levels were available in these studies. Animal experiments have suggested that repeated exposure to peak concentrations may be more harmful to health than exposure to long-term average concentration levels resulting in the same inhaled dose (Gardner et al., 1979). As peak concentration levels are likely to occur in homes, and as their relationship with long-term average concentration levels is likely to be weak, exposure to NO_2 may have been inadequately characterised even in those studies that have employed large scale passive sampling of NO_2 in homes and on persons. It is conceivable that the inconsistency of the results of epidemiological studies conducted so far is partly related to this issue, as peak concentration levels in homes have been shown to exceed the 1987 WHO health guidelines by a fairly large margin in a fairly large proportion of homes.

Several infectious diseases are known to be transmitted from one person to the next when infectious agents are propelled into the air by coughing, sneezing, singing, talking, etc. The major reason why airborne infections are much more likely to spread indoors than outdoors is, that the dilution in outdoor air is usually so great that chances of inhaling enough infectious droplet nuclei to become infected, are negligible. Also, people spend much more time indoors than outdoors, especially in winter when respiratory infections are generally more prevalent than in summer. Several studies have linked Legionnaires' disease, humidifier fever and bronchopulmonary aspergillosis to agents spread by contaminated HVAC systems or resulting from refurnishing of hospitals (Finnegan et al., 1986). There is little evidence that biological contamination of homes can lead to these specific diseases, although isolated cases have been known to occur.

7.4 SUSCEPTIBLE GROUPS

The respiratory system of young children is considered to be more susceptible to environmental insults than that of adults. Also, children have faster respiratory and metabolic rates compared to adults. Elderly people with impaired pulmonary function and/or weakened defence systems may also be more at risk, as an insult of a given size will affect them more than people with a larger reserve capacity. Smoking may also increase susceptibility to indoor air pollutants. In addition, patients already suffering from Chronic Obstructive Pulmonary Disease (COPD) are considered to be more susceptible than healthy individuals.

Once sensitised, people suffering from allergies are orders of magnitude more sensitive to allergens and to some other pollutants than the non-sensitised population. A special population at risk are unborn children whose mothers smoke, as they are exposed to noxious substances in utero. Other susceptible groups are people exhibiting increased non-specific bronchial reactivity and/or asthma, also some persons are susceptible to contracting respiratory infections as they have impaired ability to fight off infections. This group includes persons with immunodepressive conditions as associated with, for instance, AIDS and cancer; young children; the elderly; persons with existing disease such a chronic obstructive lung disease; and possibly those who may be more susceptible to infections due to exposure to irritating agents such as NO_2 which may damage mucociliary cells etc.

7.5 PUBLIC HEALTH RELEVANCE

A large segment of the population is exposed to ETS, to combustion products from unvented combustion appliances, and to biological contaminants in the home. The smoking habit is still so prevalent in many European countries that smokers are present in over 50% of the households. Effects of exposure to ETS on the respiratory system of children are well documented, and sizeable public health benefits can be achieved when smoking in private homes is discouraged. Respiratory disease is relatively common among children, and a reduction in exposure to ETS could result in a significant decrease in incidence (Burchfiel et al., 1986; Samet et al., 1987a, 1988; Ware et al., 1984). Even if relative risks are small, as seems to be the case with risks associated with exposure to NO_2 in the home, attributable risks may be of some concern when many are exposed. Dampness in homes is a recurrent area of concern, partly due to building practices which, in reducing ventilation, may cause moisture to be trapped inside new homes. Home dampness may enhance growth of mites and fungi, which produce substances that may sensitise susceptible individuals.

The public health relevance of infectious disease caused by indoor contamination is not easy to assess. It has been noted that a substantial proportion of disease and absenteeism from work or school is associated with infectious episodes caused by indoor air exposure to infectious agents. To the extent that such exposure is preventable by reducing proliferation of infectious agents in buildings or by immunisation, air irradiation or minimisation of crowding, the public health relevance of indoor factors leading to infectious diseases could be sizable.

7.6 METHODS FOR ASSESSMENT OF EFFECTS ON THE RESPIRATORY SYSTEM

For the investigation of respiratory health effects, human epidemiological and experimental studies, animal and other laboratory experiments can be employed.

Epidemiological studies have been widely used, mostly focusing on pulmonary function and respiratory symptoms. A fairly high level of standardisation has been reached in this field. In the United States of America, an extensive set of guidelines on pulmonary function testing as well as the use of respiratory symptom questionnaires was published by the American Thoracic Society (ATS) in 1978. In 1987, the guidelines for pulmonary function testing were updated (American Thoracic Society, 1987). In Europe, the European Community for Coal and Steel (ECCS) published guidelines for pulmonary function testing in 1983. An up-dated version of the European document has recently been published (Quanjer, 1993). For the assessment of respiratory symptoms, the questionnaire of the British Medical Research Council (BMRC) has been used for decades to investigate symptoms in adults. Questionnaires for children have been developed by the ATS, WHO and ECCS. The WHO and ECCS questionnaires have both been used in international collaborative studies in the 1970s. Despite the existence of these questionnaires, many investigators use their own or include modifications. Validation of these questionnaires has been incomplete.

Less frequently applied in epidemiological studies are measurements of hypersensitivity and non-specific hyperreactivity. Histamine, methacholine and cold air have been used to test airway responsiveness in such studies followed up by bronchodilators to test reversibility of obstructions (Abramson et al.,1990; Cartier et al., 1989; Cockcroft et al., 1977). To study hypersensitivity, specific extracts of allergens have been used in skin tests and in bronchial provocation tests. Also, measurement of antibodies in serum has been performed. Short-term changes in respiratory symptoms can be studied by using symptom diaries, in which day-to-day changes in symptoms are recorded by or for study subject and by using daily peak flow recordings (Burge P.S., 1982; Lebowitz, 1991). The advantage of epidemiological studies is that human populations are studied under normal living conditions, which makes their findings highly relevant from a public health point of view. The major disadvantage is that it is difficult to control for other potential determinants of the health effect which is being studied.

Human experimental studies have been used to investigate short-term, reversible changes in pulmonary function and respiratory symptoms in volunteers (Frampton et al., 1991; Hackney et al., 1984). The methods to study

effects can be more sophisticated than in epidemiological studies. Exposure conditions are under control of the investigator. The major disadvantage is that only short-term, reversible changes can be studied.

Small airway dysfunction as measured by pulmonary function tests (MEF25, N2-washout curves) has been suggested as a marker for early damage to the lung; tests of non-specific bronchial hyperreactivity have been suggested as a means to identify a susceptible subpopulation; skin tests can be used to identify sensitised individuals.

As the agents responsible for infectious diseases are of biological origin, the methods to measure effects do not only include confirmation of signs and symptoms, but also isolation, culture and identification of the microorganisms involved.

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Chapter 8

Allergy Associated with Indoor Air Pollution

The immune system is characterised by the ability to recognise and react specifically with foreign macromolecular material. This ability is mostly beneficial and plays an important part in resistance to infectious disease, but sometimes results in adverse effects such as allergic or hypersensitivity diseases of the respiratory and other systems.

Indoor as well as outdoor air pollutants may induce an immunological sensitisation in susceptible individuals in whom any future contact with the pollutant (the allergen) may elicit an adverse response.

This chapter is restricted to the description of allergic effects in the respiratory system.

8.1 ALLERGIC DISEASES ASSOCIATED WITH EXPOSURE TO INDOOR AIR POLLUTION

Allergic asthma and extrinsic allergic alveolitis (hypersensitivity pneumonitis) are the two most serious immunological diseases caused by allergens in indoor air. Allergic rhinoconjunctivitis and humidifier fever are other important diseases; it is not clear if or how the immunological system is involved in humidifier fever.

Asthma, a heterogeneous condition, is characterised by reversible narrowing of the lower airways and airway inflammation. Pulmonary function during an attack shows an obstructive pattern, in serious cases together with reduced ventilation capacity. Asthma may be caused by exposure to indoor air pollutants, either acting as allergens or as irritants (Burge et al., 1985a; Finnegan et al., 1984). Immunological specific IgE sensitisation to an airborne allergen is a major component of this disease, but non-specific hypersensitivity is also important for the asthmatic attacks occurring on exposure to irritants in the indoor air.

The prevalence of asthma varies considerably from country to country. Asthma may cause death. The costs of medical care are also considerable in terms of hospital admissions, medication, and lost work days.

Allergic rhinoconjunctivitis is also an IgE-mediated disease, and is especially prevalent among children and young adults. The main symptoms are itching of the eye and/or the nose, sneezing, watery nasal secretion and some stuffiness of the nose. The severity of the symptoms varies with the exposure to the allergen and to certain irritants. Individuals often suffer from both allergic asthma and allergic rhinoconjunctivitis and are seldom sensitive to only one allergen. Aeroallergens from house dust mites, pets, insects, moulds, and fungi in the indoor air have been shown to be associated with allergic asthma and/or rhinoconjunctivitis. Rhinitis as well as asthma may also have a non-allergic basis and be triggered by non-specific stimuli.

Extrinsic allergic alveolitis, also called hypersensitivity pneumonitis, is characterised by recurrent bouts of pneumonitis or milder attacks of breathlessness and flu-like symptoms. Studies of the pulmonary function during an acute episode will usually show a restrictive pattern with a decreased diffusion capacity. The disease is an inflammatory reaction in the alveoli and bronchioles involving circulating antibodies and a cell-mediated immunological response to an allergen. For example, it occurs in farmers as a result of handling mouldy hay ("farmer's lung") and in pigeon breeders. However, the disease has also in a few cases been associated with exposure to IAP, most frequently related to humidifiers in homes and offices contaminated with bacteria, fungi, or protozoans (Arnow et al., 1978; Ashton et al., 1981; Bansazack et al., 1970; Bernstein et al., 1983; Kohler et al., 1976; Sweet et al., 1971).

Allergic asthma and extrinsic allergic alveolitis resolve with cessation of exposure to the allergen, but continued exposure in sensitised persons may result in permanent lung damage and death from pulmonary insufficiency.

Humidifier fever is a flu-like illness associated with breathlessness, in which X-ray abnormalities are usually absent (Finnegan et al., 1986; McSharry et al., 1987; Pickering, 1982; Rylander et al., 1978). The exact cause is not clear. The disease normally occurs among persons exposed to humidification systems contaminated with microbial growth. The symptoms typically occur 4–8 h after exposure on the first day back at work after a weekend, but resolve within 24 h. Despite continuous exposure the disease does not recur until after the next weekend. Even though physiologic changes are seen during attacks of humidifier fever, the disease does not lead to permanent lung damage.

8.2 PRINCIPAL AGENTS AND SOURCES

House dust mites, pets, insects, plants and moulds in the indoor environment are important causes of allergic asthma and rhinoconjunctivitis (H.A. Burge et al., 1982; Lowenstein et al., 1986). Outdoor allergens such as pollens

and moulds may penetrate into the indoor environment through open windows, doors, or ventilation systems. The airborne allergens vary with seasons, weather conditions, geographical location, and the local indoor environment.

Among house dust mites (Kang et al., 1989; Platts-Mills et al., 1992), *Dermatophagoides pteronyssinus* and *D. farinae* are prevalent in climates where winters are humid and mild, but they can also live elsewhere as long as a microenvironment with a high humidity (> 45%) and temperatures between 17 and 25°C are provided. House dust mites and their debris and excrements that contain the allergens are normally found in the home in beds, mattresses, pillows, carpets and furniture stuffing, but they have also been found in office environments. The dust mite allergens may become airborne during indoor activity.

Domestic animals such as cats, dogs, birds, rodents and horses may cause allergic asthma and rhinoconjunctivitis (Platts-Mills et al., 1988). Allergens are found in different amounts in dandruff, hair, saliva, and urine from the animals. The exposure usually occurs in homes, but also in schools and kindergartens where domestic animals are kept as pets or for education. Close contact with people keeping animals may also give rise to allergic reactions. Shed skin scales, dried secretions, and faecal particles from insects may also cause allergic asthma and rhinoconjunctivitis. Cockroaches are an important source of allergens in homes with poor sanitary conditions.

Moulds require a high humidity (>70%) to grow. There is a large variety of moulds and many of them have very specific growth requirements (Finnegan et al., 1986; Lowenstein et al., 1986). Mould allergens are mostly found outdoors in living mould organisms, spores, and particles even smaller than spores. However, they may penetrate into the indoor environment as in the case of those vehiculated by pollens. Persistent damp areas, particularly bathrooms and basements, may support abundant mould growths indoors, but also water seepage in building material causing damp ceilings, walls, carpeting, and furniture, may provide favourable conditions of growth for moulds. Furthermore, draught-proofed ("tight") buildings may offer ideal places for mould growths when the indoor humidity is high and moisture condensation on cold areas on walls and windows occurs.

Contaminated humidifiers in homes, industrial and non-industrial buildings, and cars have been associated with allergic asthma, humidifier fever, and extrinsic allergic alveolitis. The contaminated humidifiers generate aerosols loaded with microorganisms and debris of microorganisms, and a wide range of microorganisms, including thermophilic actinomycetes, moulds, bacteria, amoebae, and nematodes, have been described as sources of offending allergens.

8.3 EVIDENCE LINKING INDOOR AIR POLLUTION TO ALLERGIC EFFECTS

Most of the evidence that links IAP to allergic effects comes from individual case reports and small series of outbreaks which resulted in comprehensive clinical descriptions.

The diagnosis of the allergic disease is based on the clinical history and signs, evidence of exposure, the presence of specific antibodies, response to inhalation challenge, and improvement with cessation of exposure.

In many cases the major allergens for allergic asthma or rhinoconjunctivitis have been identified, while identification of the causative allergens for extrinsic allergic alveolitis has often been uncertain. For humidifier fever it is debatable whether the disease is caused by exposure to allergens, bacterial endotoxins or other toxins, but it is known that the cause lies in the biological contamination of the humidifiers.

Epidemiological studies have shown that exposure to mites in homes during childhood is a major risk factor for the development of allergic asthma (Buscinco et al., 1988; Sporik et al., 1990). Reports suggest that for mite allergy a level of 100–500 mites/g house dust should be regarded as a maximum acceptable contamination (Platts-Mills et al., 1990, 1992). Otherwise, there is for non-industrial indoor air allergens no knowledge of the dose response relationships apart from the fact that allergy to animals is very unlikely to develop among occupants when not exposed daily at home or at work.

8.4 SUSCEPTIBLE GROUPS

All human beings have IgE antibodies and can make specific IgE antibodies against a number of allergens. A minor part (10–12%) of the population respond easily to allergen exposure by making specific IgE antibodies and developing allergic asthma and/or rhinoconjunctivitis. This ability (atopy) is genetically determined although not in a simple way. Outdoor air pollution, ETS, and certain types of infections possibly contribute to the allergic breakthrough. There are no cases of extrinsic allergic alveolitis being hereditary, but non-smokers seem more susceptible to the disease (Anderson et al., 1988; Finnegan et al., 1985).

8.5 PUBLIC HEALTH RELEVANCE

The medical expenses associated with allergic diseases are considerable. The prevalence and incidence of allergic diseases due to IAP have not been studied. However, the overall prevalence of allergic asthma and rhinoconjuc-

tivitis may be as high as 20%, and a large proportion of the patients suffering from asthma who are referred to a clinic are allergic to house dust mites and pets. A high prevalence of allergy to mould has also been reported in some series of patients with allergic asthma. Establishing the diagnosis of allergy to a certain allergen requires standardised allergen preparations which are lacking for most allergens; therefore, the estimation of the relative importance of the different allergen exposures is difficult. Extrinsic allergic alveolitis and humidifier fever are rare diseases in the indoor environment but epidemics in office buildings have been seen.

One of the major concerns is what happens when buildings are made more energy-efficient by reducing ventilation and increasing insulation. This may lead to condensation of water from cooking, showering, etc. and, therefore, higher indoor humidity, creating a favourable environment for house dust mites and moulds.

As the major causes of allergic asthma seem to be house dust mites, pets, and in some areas, cockroaches, this disease is highly preventable by appropriate building design and building use. Likewise, selection of better humidifier principles and proper maintenance of humidifiers will reduce the occurrence of extrinsic allergic alveolitis and humidifier fever in the indoor environment.

8.6 METHODS FOR ASSESSMENT OF ALLERGIC EFFECTS OF INDOOR AIR POLLUTION

Even though the usual investigation methods are applicable for assessing the allergic effects of IAP, clinical studies are essential for these diseases. As already mentioned, the diagnosis of the allergic disease is based on the clinical history and signs, evidence of exposure, the presence of specific antibodies, response to inhalation challenge, and improvement with cessation of exposure. A thorough examination of every individual is, therefore, only achievable in clinical and small epidemiological studies. However, the major methodological problems are to quantify the exposure, standardise the allergen preparations used in inhalation challenges, and establish the IgE antibody sensitisation to the allergen, either with skin testing or serum assays for IgE antibodies or for other antibodies.

Different methods have been used to sample airborne allergens, i.e., gravity sampling, impactors, and suction devices. Also vacuum cleaning of surfaces has been used for indirect estimation of exposure. Methods varying greatly in specificity and sensitivity have been used for determination of the allergen content of the sample, i.e., light microscopy, culture of spores, immunoassays, and measurements of enzymatic activity.

Determination of precipitating antibodies to an antigen can only be used to indicate that exposure to the antigen has taken place, not to establish the diagnosis of extrinsic allergic alveolitis *per se*.

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Chapter 9

Cancer and Effects on Reproduction of Indoor Air Pollution

A few indoor air pollutants, notably asbestos, radon, formaldehyde, and environmental tobacco smoke (ETS) have been associated with cancer although in the indoor air context formaldehyde is probably not a human carcinogen. Very few studies have tried to evaluate whether IAP affects human reproduction.

9.1 CARCINOGENIC AND REPRODUCTIVE EFFECTS ASSOCIATED WITH EXPOSURE TO INDOOR AIR POLLUTION

Lung cancer is the major cancer which has been associated with exposure to IAP (radon or ETS) (National Council on Radiation Protection and Measurements, 1984; National Research Council, 1986). Asbestos exposure has been linked to cancer in workers and also in workers' family members, presumably due to asbestos fibres brought into the home on workers' clothing (Epler et al., 1980; Li et al., 1978; Muncharek et al., 1989). However, there are no studies associating asbestos exposure in homes or public buildings from asbestos used as a construction material, to the development of cancer.

Mesothelioma is a malignant tumour of the pleura and of the peritoneum associated with asbestos exposure. The relevance of this to normal indoor air is unclear.

Effects on human reproduction have been associated with exposure to chemicals in the environment, but it is unclear to what extent (if any) exposure to IAP is involved.

9.2 PRINCIPAL AGENTS AND SOURCES

The principal agents present in indoor air associated with lung cancer are ETS and radon decay products. Tobacco smoke has been known to cause cancer in man for a long time, and although sidestream smoke has a different composition from

the mainstream smoke which is being inhaled by a smoker, carcinogens have been identified in sidestream smoke as well. Some investigations suggest that the concentration of carcinogens in sidestream smoke is higher (relative to other substances) than in mainstream smoke (Adams et al., 1987; Guerin et al., 1987). It is well known that in smokers' homes, the long-term average concentration of particulate matter in the air is considerably higher than in non-smokers' homes (Brunekreef and Boleij, 1982; Moschandreas et al., 1981; Spengler et al., 1981). Often the concentration in smokers' homes is also higher than in outdoor air. Few studies have measured carcinogens resulting from tobacco smoking in indoor air.

Radon and radon decay products are present at different levels of concentration in indoor air of all dwellings. High concentrations can be found in homes built on soils which are rich in uranium and/or having a high permeability. In several countries, areas have been identified in which indoor radon concentrations in homes approach levels which have been associated with an increased lung cancer incidence in miners. An overview of radon levels measured in homes in EC countries is given in the report "Radon in Indoor Air" (Commission of the European Communities, 1988) and in Section 3.4.3. When radon and radon decay products are inhaled the radiation dose to lung tissue is dominated by the alpha particles emitted by the short-lived decay products deposited in the lung, especially the decay products attached to small size aerosols or in unattached form. A synergistic effect seems to occur, to a greater or lesser degree, between radon and cigarette smoking both in mines and dwellings, so that smokers exposed to radon probably have a higher risk (6–10 times) than non-smokers (see also Section 3.4 and Chapter 16).

Of potential further interest are asbestos, polycyclic aromatic hydrocarbons, benzene, formaldehyde, some pesticides, and nitrosoamines (which may form on filters of recirculating kitchen exhaust fans), all of which have been found in indoor air, and all of which are known or suggested to be human carcinogens (I.A.R.C., 1989; Lewtas et al., 1987; Matsushita et al., 1990; U.S. Environmental Protection Agency, 1989; WHO, 1987). However, there are no firm indications that the levels of these pollutants normally encountered indoors warrant much concern. This is also true for exposure to electromagnetic fields, such as those generated by electric conductors.

Some chemicals which have been associated with adverse effects on human reproduction are tobacco smoke, solvents, chlorinated pesticides, and metals such as lead. Although most of these may occur in indoor air, apart from ETS there is basically no information to decide whether the levels normally encountered in indoor air warrant concern.

9.3 EVIDENCE LINKING INDOOR AIR POLLUTION TO CANCER AND EFFECTS ON REPRODUCTION ON HUMANS

Although several of the substances mentioned in the previous section are considered to be human carcinogens by the International Agency for Research on Cancer, only few of them have been linked to human cancer in epidemiological studies on specific indoor exposures. The main emphasis in these studies has been on the relationship between exposure to ETS ("passive smoking") and lung cancer, a topic on which a relatively large number of studies have been published. Although only a few of these show a significantly increased chance of dying from lung cancer associated with ETS exposure, several meta-analysis of all available information have concluded that the increase is real (UK Department of Health, 1988; US Surgeon General, 1986). The risk of dying from lung cancer associated with living with a smoker, relative to living with a non-smoker, has been estimated at 1.35. Most studies of the issue have considered women only, so that at present, it is not clear to what extent non-smoking men living with a smoking partner are at increased risk.

The carcinogenicity of radon decay products for humans has been firmly established in studies among miners, at exposure levels exceeding those commonly found in the air in homes (Lundin et al., 1971; National Research Council, 1980; Samet and Hornung, 1990). It should be noted that in a small fraction of dwellings the cumulative exposures reach values comparable with those found in miner epidemiology studies. Recently, some epidemiological studies have addressed radon in homes more directly, and some (but not all) of these suggest that exposure to radon decay products in the home is indeed associated with an increased lung cancer incidence (Edling et al., 1984). One case-control study from Sweden included measurement of indoor radon concentrations in the homes where study subjects had actually lived, and a significant dose–response relationship was found for all histological types of lung cancer combined, and for small cell carcinoma in particular (Svensson et al., 1989). In this particular study the elevated risk was restricted to smokers, suggesting an interaction between smoking and radon decay product exposure as has been found in the occupational studies among miners too. An interaction between "passive smoking" and radon decay product exposure has also been suggested.

From occupational studies, asbestos fibres are known to be able to cause cancer (mesothelioma and lung cancer) in humans. Several case reports have shown that asbestos carried home on workers' clothes can lead to high exposures in the home, and some fatal mesothelioma cases among non-occupationally exposed partners have been documented (Epler et al., 1980; Li et al., 1978; Vianna et al., 1978;). Due to the widespread use of asbestos in the past, many buildings contain asbestos in some form, and low-level exposures occur in

these buildings. However, there is ongoing discussion on whether these low levels of exposure are associated with a sizable increase in the risk of mesothelioma or lung cancer.

Benzene is a known cause of leukaemia in occupationally exposed humans and may be present in indoor air at low concentrations. A large recent study indicates that the main sources of benzene exposure to the general population are personal activities or sources inside the home. The most important of these, accounting for over half of the population exposure to benzene is smoking. Other important sources are exposure to ETS and to certain consumer products. However, a number of occupational studies with low exposure levels have failed to detect significant increase in leukaemias among the exposed workers, and it is questionable whether increased risks exist at the even much lower levels encountered in homes. In areas with heavy automobile traffic, ambient concentrations of benzene are probably higher than those encountered indoors.

The mutagenicity of indoor air, as measured by *in vitro* techniques (see below) has been studied by several investigators. Mutagenicity refers to the capacity of a substance to induce a permanent change in the amount or structure of the genetic material in an organism, resulting in a change in the phenotype characteristics of the organism. The alteration may involve a single gene, a block of genes, or a whole chromosome. Tobacco smoke is a major source of mutagens in the air in homes. In addition, wood smoke from fire places and fumes produced by cooking activity have been shown to increase the mutagenicity of indoor air.

Occupational studies have shown that exposure to lead, ethylene oxide and some pesticides can lead to effects on human reproduction such as spontaneous abortions, infertility and chromosomal aberrations. There is no evidence that at the lower levels of exposure to these chemicals normally encountered in the non-occupational indoor environment, hazards of this type exist. Smoking has also been implicated, but there is little evidence that passive smoking is associated.

9.4 SUSCEPTIBLE GROUPS

Susceptibility to cancer is believed to vary among individuals due to genetic factors. However, methods so far available do not allow susceptible individuals to be reliably identified.

9.5 PUBLIC HEALTH RELEVANCE

Many non-smokers are exposed to ETS in the home, so the public health

relevance of an increased lung cancer risk associated with ETS exposure are potentially large. In some countries, large numbers of homes have elevated levels of radon decay products in the air, and the risks estimated to be associated with these levels are sometimes appreciably higher than those deemed maximally acceptable for other chemical exposure (Edling, et al., 1984; Nazaroff et al., 1990). For example, the lifelong risk of developing lung cancer due to exposure to a radon decay product equilibrium concentration of 1 Becquerel/m^3 has been estimated by WHO (1987) at 0.7–2.1 per 10 000. In countries such as Sweden, Norway and Finland many homes can be found with indoor concentrations exceeding 100 Becquerel/m^3, and those living in these homes, therefore, are at a remarkable level of risk. In several countries, risks for life-long exposure to environmental carcinogens in the order of one per million only are considered acceptable, which makes clear why exposure to indoor radon decay products has become such a prominent issue in the past decade.

ETS and radon may account for a sizable fraction of lung cancer cases among non-smokers. As mentioned before, radon may, through synergistic interaction, account for a sizable number of lung cancer cases among smokers as well.

Formaldehyde causes nasal carcinomas among rats after exposure to high concentrations (Anonymous 1984, 1992; Kerns et al. 1983; WHO, 1987). The mechanisms have not at present been fully described, but existing evidence indicates that acute peak formaldehyde exposure is more important for the occurrence of nasal tumours than the accumulated doses (IARC, 1989).

In 1991 the EPA made cancer risk evaluations based on the possible DNA–protein cross-links which may appear as a result of formaldehyde exposure. At present, this model seems to be the most acceptable description of the mechanism involved in the development of human cancers due to airborne exposures to formaldehyde. These investigations have been made on rats and monkeys, the latter being considered more relevant to humans because monkeys are more similar to humans than rats with respect to the respiratory mechanism, absorption, etc. Using investigations of monkeys, a value of 36 $\mu g/m^3$ seems to correspond to a lifetime risk of 10^{-5} (Blair et al. 1986; Casanova et al., 1991).

For other human and animal carcinogens found in indoor air, there is insufficient epidemiological evidence to decide to what extent these are of concern at levels normally encountered.

Data at present are insufficient to assess the public health relevance of potential effects of IAP on human reproduction.

9.6 METHODS FOR ASSESSMENT OF CARCINOGENIC AND REPRODUCTIVE EFFECTS

The carcinogenicity of substances can be studied in animal experiments but usually relatively high doses are required to induce cancer in a sufficient number of animals in the exposed groups. As a consequence, assessment of risk for humans from such data only, requires extrapolation from experimental animals to humans, and also from high to low doses. The uncertainty involved in both extrapolations is large. Thus, no reliable and precise estimate can be made of the cancer risks associated with human exposure to the substance on the basis of animal experiments alone.

The mutagenicity of substances can be tested using a variety of *in vitro* and *in vivo* tests. Bone-marrow assays for chromosome damage are an example of the latter, the well-known Ames test is an example of the former. A mutagen is not necessarily a carcinogen, and by themselves, tests of mutagenicity do not permit risk assessment of human exposure.

Epidemiological studies can be used to detect whether human exposure to a substance is actually associated with an increased cancer incidence. Usually, large numbers of people (cohorts) need to be followed for a long time to unequivocally document changes in cancer incidence associated with exposure. Therefore, the case-control approach is widely used in cancer epidemiology, in which cancer cases are compared to suitable control subjects with respect to past exposure to the suspected agent and to other potential causes of cancer. In both types of study, confounding factors may produce spurious associations (or mask true associations) between exposure and disease, so that suitable treatment of confounders is a major issue. Besides ethical considerations on keeping human beings exposed to suspected carcinogens, it must be noted that to obtain the epidemiological proof about the carcinogenicity of a chemical may be very difficult unless the potency of the substance (or the power of the study) is high.

Various specific approaches are used in toxicology to detect effects of chemical substances on reproduction. Among these are in vitro tests on whole embryo systems, and mammalian three generation studies in which three consecutive generations of experimental animals are exposed to various dose levels of the substance under study. Human epidemiological studies have utilised study endpoints such as spontaneous abortion frequency, number of miscarriages, birth weight, sperm counts, and birth defects.

Again, the complexity of these studies has to be noted as to the possibility of confirming experimental data that are more easily obtained.

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Chapter 10

Irritative Effects of Indoor Air Pollution on the Skin and Mucous Membranes of Eyes, Nose and Throat

10.1 IRRITATIVE EFFECTS ASSOCIATED WITH INDOOR AIR POLLUTION

Exposure of the skin or mucous membranes to indoor air pollutants stimulates sensory systems and may cause dysfunctions and tissue changes. Each of these may subsequently lead to the other. Two types of sensory irritation appear in the literature on indoor climate and air quality: a primary sensory irritation, caused by direct stimulation of sensory cells by environmental exposures, and a secondary irritation following inflammatory changes in the skin, mucous membranes or other tissues (Mølhave, 1991).

This chapter deals with irritative effects associated with tissue changes while primary sensory irritation will be dealt with in the following chapter. The most common effect of indoor environmental agents on the tissues of skin and mucosal membranes is inflammation (Mølhave, 1991). It is characterised by a sensation of heat ("calor"), redness ("rubor"), swelling ("tumour"), pain ("dolor") and a certain loss of function ("functio laesa") in the tissues affected. Irritative effects on tissues can be a considerable annoyance either in terms of severity of effects on individuals or in terms of the number of persons affected. Host factors such as hyperreactivity may play a role (Mølhave, 1991).

Signs and symptoms of effects on skin and mucous membranes may appear at the site of contact of the exposed skin, mucosa, etc. or manifest themselves in other tissues due to nervous reflexes, although they have seldom been seen to follow exposure to indoor air (Mølhave, 1991; Mølhave et al., 1991). The symptoms and signs are unspecific and may be caused by several exposure factors other than those present in indoor air. Also, some exposures may cause a number of additional signs and symptoms, such as physiological or sensory reactions, psychological reactions and subacute changes in sensitivity to environmental exposures.

10.2 PRINCIPAL AGENTS AND SOURCES

Formaldehyde

Formaldehyde is highly water soluble and causes irritation of the mucous membranes of the eyes and the upper respiratory tract. Symptoms of irritation include dry and/or sore throat and a tingling sensation of the nose, usually coexisting with watering and painful eyes. Irritation occurs over a wide range of concentrations and the interindividual variation is known to be large (Bernstein et al., 1984; Imbus, 1985; WHO, 1989b). Sensory irritation usually begins at approximately 0.1 ppm, but is reported by more than 50% of the exposed at 0.6 ppm (Ahlstrom et al., 1986). Increased tearflow and eye-blinking is reported to be caused by formaldehyde (WHO, 1989b). This is not strictly dose-dependent and appears only in some people. At high exposure levels, formaldehyde may act as an allergen and provoke IgE antibodies (Burge et al., 1985b; Kramps, 1989; Patterson et al., 1989). Oedema and inflammation appear after formaldehyde exposure exceeding that usually occurring in indoor air.

Other aldehydes

Acetaldehyde, acrolein and other aldehydes are known irritants. However their relation to irritation of the skin and mucous membranes in indoor environments has not yet been extensively investigated. ETS is a major source of exposure to these compounds. Acrolein may produce conjunctivitis (Weber-Tschopp et al., 1977).

Volatile organic compounds (VOCs)

Many VOCs are mucous membrane irritants and have been implicated as a cause of SBS. Studies of the acute effects of VOCs indicate that concentrations of VOCs found in new buildings may cause irritative tissue changes in the eyes (WHO, 1989a).

Environmental tobacco smoking (ETS)

ETS is a complex mixture of pollutants whose source is primarily cigarette smoking. Various components of this mixture have been actively monitored including respirable suspended particles, CO, nicotine, nitrogen oxides, acrolein, nitroso compounds and benzo(a)pyrene. The major sites of irritative changes caused by ETS are the eyes and nasopharynx. Eye and conjunctival irritation, nasal discomfort, sore throat, sneezing, and cough are frequently reported symptoms in both smokers and non-smokers (Weber, 1984). Increased tearflow and eye-blinking are also reported to be caused by ETS (Cain et al., 1987; Muramatsu et al., 1983; Winneke et al., 1990).

Other exposure

Temperature and humidity variations may themselves cause effects on skin and mucous membranes (Williamson and Allison, 1967). The intensity of symptoms due to irritative effects may vary due to interactions with other exposures. Temperature and humidity have been demonstrated to influence the level of eye and nose irritation experienced by non-smokers exposed to ETS. Changed mucosal clearance is known from exposure to NO_2 or particulate matter. Irritation due to agents such as biological contaminants or other factors has not been described in the literature to the level where a conclusion can be drawn.

10.3 EVIDENCE LINKING EXPOSURE TO INDOOR AIR POLLUTION TO IRRITATIVE TISSUE CHANGES

Apart from formaldehyde and ETS, chemicals which are likely to produce irritative changes are found at levels in the normal indoor environment which are orders of magnitude below those known to produce irritation in the industrial indoor environment. Few agents other than these two are known to cause irritative effects at concentrations found indoors. However, the effects observed in industrial environments may be different from the effects in the non-industrial indoor environment.

Most experimental studies of non-carcinogenic effects of potential irritants do not involve exposure periods longer than a few hours and may, therefore, not show a relation between long-term exposures and health effects.

Several experimental studies of the acute effects of VOC have been undertaken to study building related illness, and SBS in humans. These experiments have shown irritative effects of VOC at exposure levels encountered in new buildings, but interpretation of these studies is limited by the non-specific nature of the effects. The biological mechanisms of the relationship between VOC exposure and building related illness or SBS remain unclear.

10.4 SUSCEPTIBLE GROUPS

There is a wide range of individual susceptibility to formaldehyde exposure. The exact proportion of people with increased susceptibility to formaldehyde is unknown.

Very few investigations have dealt with increased susceptibility to irritation due to indoor air pollutants other than formaldehyde. No conclusion can therefore be made with respect to the existence of other groups at risk. However, it might be argued that persons with allergic disorders, chronic irritative skin diseases (such as eczema), and ocular diseases, might be at higher risk of developing symptoms or worsening existing diseases.

10.5 PUBLIC HEALTH RELEVANCE

Studies have been undertaken to determine the magnitude and extent of formaldehyde exposure of the general population in the home environment. However, most studies have dealt with specific problems such as mobile homes, so that the results are difficult to extrapolate to the whole population.

The effects of other factors and their interactions — such as cigarette smoking history, variability of health status, gender, age and genetic predisposition (which may modify responses to formaldehyde) — have not been adequately evaluated. This makes it difficult to accurately assess the health risks due to irritative effects attributable to exposure to formaldehyde indoors.

More than half the population is estimated to be exposed to formaldehyde as a component of ETS. Even though dose–response effects for irritative tissue change are not known, this exposure may be of public health concern.

Fibres (i.e. glass fibres), ozone and NO_2 are other possible agents causing irritation of tissues. These exposures have been even less well characterised.

10.6 METHODS FOR ASSESSMENT OF IRRITATIVE EFFECTS

The possible effects of indoor air pollutants on the skin and mucous membranes can be measured at high exposure levels. The available methods have varying sensitivity, precision and accuracy. Most objective methods have not yet been documented at exposure levels relevant to the non-industrial indoor environment and there is a general need for development and validation of such methods (Imbus, 1985). Objective measurements of irritation of the eyes are at present the most promising.

Some of the methods relate to those surface phenomena of the eye, which are part of the eyes protective mechanisms. Increased blinking rate seems to be a useful protection against medium to high levels of irritants. The constituents and stability of the tear film formed during blinking also seems to be affected by exposures found indoors (Johnson et al., 1991; Kjærgaard, 1992a) and, furthermore, seem to be correlated with sensory irritation in affected buildings (Franck, 1986; Kjærgaard et al., 1993a). Damage of eye epithelium and irritation seems to be related to exposures (Franck, 1986; Kjærgaard et al., 1993a; Wolkoff et al., 1992a). Methods used to test these inflammatory responses on both the eyes and the nose seem promising.

Increased inflammatory response in the form of inflammatory cells (and of mediators in the nose) has been shown for *n*-decane, mixtures of VOC, house dust, and nitrous acid exposure (Kjærgaard et al., 1990, 1992b, 1993b; Koren et al., 1992). In the nose, measurements of the mucociliary flow rate showed effects at medium to high exposure levels, but not at low levels. Most of these parameters has only been documented in few cases and not all results are consistent. Therefore, they need further documentation and validation.

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Chapter 11

Sensory and other Effects on the Nervous System due to Indoor Air Pollution

11.1 SENSORY EFFECTS ASSOCIATED WITH INDOOR AIR POLLUTION

Sensory effects are defined within the context of indoor air science as the perceptual response to environmental exposures. Sensory perceptions are mediated through the sensory systems. These systems all contain various receptors, from which signals are transmitted to the higher levels of the central nervous system (CNS) where the message results in a conscious experience of odour, touch, itching, freshness, etc.

Sensory effects are typically observed in buildings with indoor climate problems because many chemical compounds found in the indoor air have odorous, mucosal, and skin irritation properties. It is important to notice that some indoor air chemicals with a measurable vapor pressure will be odorous even at concentrations below their chemical-analytical detection limit. The odour of a few others may only be detectable at concentrations exceeding the thresholds for other adverse health effects.

Sensory effects are important in indoor air quality control for several reasons. They may appear as:

(1) adverse effects on sensory systems (e.g. environmentally-induced sensory dysfunctions);

(2) adverse environmental perceptions, which may be adverse *per se* or constitute precursors of disease to come on a long-term basis (e.g. annoyance reactions, triggering of hypersensitivity reactions);

(3) sensory warnings of exposure to harmful environmental factors (e.g. odour of toxic sulfides, mucosal irritation due to formaldehyde);

(4) important tools in sensory bioassays for environmental characterization (e.g. using the odour criterion for general ventilation requirements or for screening of building materials to find those with low emissions of volatile organic compounds).

11.2 MECHANISMS INVOLVED IN SENSORY PERCEPTION

In the indoor environment, two main classes of sensory perception can be identified. The first class includes perceptions attributed to the surrounding physical environment (environmental perception), for example perceptions of draft and odour. Environmental perception can be adverse or non-adverse.

The second class includes perceptions of events inside the body or on the body surface (body perceptions). The body perceptions (e.g., perceived eye irritation or dry skin), may or may not be attributed causatively to the surrounding physical environment. The sensory systems are tuned towards registering environmental changes rather than the absolute exposure levels. The senses responding to environmental exposure are not only hearing, vision, olfaction and taste, but also the cutaneous and chemical senses of the skin and mucous membranes.

As pointed out by WHO (1989a), many different sensory systems which respond to irritants are situated on or near the body surface. Some of these systems tend to respond to an accumulated dose and their reactions are sometimes delayed. On the other hand, in the case of odour perception the reaction is immediate but also very much influenced by olfactory adaptation after prolonged exposures. The sensory irritation response has been shown to be facilitated rather than adapted over time (Cain et al., 1987). It is also well-known that the odour response will facilitate over time at low concentrations due to cross-adaptation, a phenomenon that may be relevant for indoor air.

Responders are often unable to identify a single sensory system as the primary base of sensory irritation by airborne chemical compounds. The sensation of irritation is influenced by a number of factors such as previous exposures, skin and mucous temperature, competing sensory stimulation, etc.

Since interaction and adaptation or facilitation processes are characteristic of the sensory systems involved in the perception of odour and mucosal irritation, the duration of exposure influences the perception.

Most of the airborne contaminants are odorous irritants because they involve both olfaction and chemesthesis at one concentration or another. Chemesthesis refers to the chemical sensibility mediated by free nerve endings in mucosal membranes and the skin. Perceptually, it seems that irritation inhibits odour so that with increasing exposure concentration, olfaction and chemesthesis shift in dominance towards less odour and more irritation (Cain, 1976; Cometto-Muñiz et al. 1991). Recordings of olfactory and chemosensory evoked potentials in anosmics and non-anosmics confirm the existence of such perceptual odour-to-inhibition shifts (Cometto-Muñiz, 1993).

The critical question for environmental pollutants is what perceptual attributes associated with olfaction and chemesthesis will appear and how will

these share the perceptual images resulting from inhaled gases and vapours at various concentrations. The research on sensory irritation has mainly been concerned with relatively high concentrations and has also been limited to the problem of isolating the trigeminal effects by using anosmic subjects who lack the sense of smell (Cometto-Muñiz et al., 1990). It is not known to what extent sensory irritation perceived by anosmic subjects corresponds to irritation perceived by non-anosmic occupants.

Humans integrate different environmental signals to assess total perceived air quality and comfort or discomfort. Comfort and discomfort by definition are psychological concepts and for this reason even when severe, cannot be documented without using subjective reports.

Sensory effects reported to be associated with indoor air pollution are in most cases multisensory and the same perceptions or sensations may originate from different sources. It is not known how different sensory perceptions are combined into perceived comfort and into the sensation of air quality. Perceived air quality is mainly related to stimulation of both the nerves trigeminus and olfactorius. Most of the odorous compounds are also significant mucosal irritants, especially at high concentrations. However, a few compounds produces sensory irritation at low concentrations. The olfactory system continuously signals the presence of odorous compounds in the air and has an important role as a warning system. In the absence of instrumentation for chemical detection of small amounts of some odorous vapours, the sense of smell remains the only sensitive indicator system.

The integrated effects of complex mixtures of odorants and irritants have been investigated in several studies. These studies include:

(a) Chamber exposure studies using exposures such as tobacco smoke (Weber, 1979), VOC (Kjærgaard et al., 1991; Mølhave et al., 1986), and building materials (Johnson et al., 1991; Wolkoff et al., 1992b).

(b) Symptoms reporting and questionnaires in the field.

(c) Studies of the correlation with other measures including indicators of sensory irritation (Franck, 1986; Kjærgaard et al., 1993a).

(d) Relationship between symptom reports and exposures (Mølhave et al., 1986, 1991; Mølhave, 1991).

(e) Interaction with other environmental factors in causation (Kjærgaard et al., 1993a).

11.3 TOXIC EFFECTS ON THE NERVOUS SYSTEM

It is well known that environmental pollution can affect the nervous system. The effects of occupational exposure to organic solvents can be mentioned as an example. A wide spectrum of effects may be of importance,

ranging from those at molecular level to behavioural abnormalities.

Since the nerve cells typically do not regenerate, toxic damage to them is usually irreversible. The nerve cells are highly vulnerable to any reduction in oxygen supply. Furthermore, the nerve cells can be exposed for a long time to chemicals that are able to enter the CNS. The risk of accumulation of hazardous compounds within the CNS is higher than in other body tissues since the nerve cells are slow in metabolizing intruding chemicals. Many solvents affect the nerve cells by interfering with the transmission of nerve signals, e.g., by inducing narcotic effects.

Although a number of adverse health disorders of the CNS are suspected to be associated with exposure to hazardous pollutants in the environment, there is so far no documentation that non-industrial exposure in homes or offices to indoor air pollution produces these endpoints.

11.4 PRINCIPAL AGENTS AND SOURCES

Sensory effects have been linked to various indoor air pollutants. In particular, many organic compounds may cause odours and/or mucosal and skin irritation at concentrations encountered indoors. Formaldehyde is a strong mucosal irritant and the most common pollutant occurring in the indoor air at concentrations known to cause sensory irritation in the eyes and upper airways (>100 ppb). These symptoms have been reported in buildings with particle board or urea formaldehyde foam insulation (UFFI) after remodelling or after installation of new furnishings or carpets.

A great number of volatile organic compounds are emitted from indoor materials and products. Many indoor materials, including paints, stains, adhesives, chalks, contain petroleum-based solvents. Such solvents are composed of a variety of organic compounds which often subsequently are found in indoor environments.

Volatile organic compounds enter the indoor environment also as mixtures of pollutants from cigarette smoke and from unvented combustion appliances. Not all of these substances have been identified individually. However, environmental tobacco smoke is known to produce sensory irritation and odour complaints, and is a predominating contributor to the perception of bad indoor air quality.

Several agents present as pollutants in the indoor environment are known to be neurotoxic; however, their effects generally have only been seen at high exposure levels in occupational settings.

The most important among the potentially neurotoxic substances found in the indoor air are the volatile organic compounds, such as acetone, benzene,

toluene, cyclohexane, *n*-hexane, formaldehyde, styrene, chlorinated solvents, and several other organic solvents. Another compound causing adverse CNS effects, which can be fatal, is carbon monoxide (CO), which may interfere with oxygen supply of the nervous tissue. Impaired vigilance function is one of the possible effects at low concentrations.

Some pesticides are also well-known neurotoxins. Most are poisonous to insects and parasites because of there neurotoxicity and may act on mammals through the same mechanisms. Prolonged exposure to some pesticides may cause irreversible effects in the central or peripheral nervous system. The extent of exposure to these chemicals in indoor environments are not clear and vary much according to geographical areas, proximity to agricultural settings and local habits of use of pesticides.

Important indoor exposure has been documented for pentachlorophenol, chlordane and other chlorinated insecticides; however no cases of nervous system effects have been associated with these environments.

11.5 EVIDENCE LINKING INDOOR AIR POLLUTION TO SENSORY EFFECTS AND EFFECTS ON THE NERVOUS SYSTEM

Many indoor air pollutants are odorous, even at the low concentration which are appearing indoors and there is ample evidence that their sensory effects are playing an important role in the occupant's acceptability of indoor air quality. However, it should be noted that some air pollutants lack sensory warnings and behave differently from the odorous ones.

One of the important characteristics of an odorant or irritant is the concentration at which it can be barely detected by a subject (detection threshold) or recognized (recognition threshold). The latter thresholds are up to four times larger than the detection thresholds. However, reported detection and recognition thresholds may vary considerably between studies from different laboratories using different measuring techniques; differences of 10 000 ppb are not uncommon. Very little information is available about dose–response relationships of airborne mucosal irritants, somewhat more of odours. On the average irritation is detected at about 6 times higher concentration than odour (Berglund and Shams, 1992). However, recent research on formaldehyde and pyridin indicate a very large interindividual variation in irritation thresholds as compared to odour-thresholds.

As mentioned, effects on the nervous system have not been linked to exposure to indoor air pollution. Practically all organic solvents are able to interfere with nervous system functions in man at high concentrations. How-

ever, the exposures associated with neurotoxicity are generally orders of magnitude higher than exposures usually encountered in non-industrial indoor environments.

Controlled human exposures to complex mixtures of VOC have suggested the possibility of functional memory impairment and sensory irritation at concentrations comparable to those detected in newly constructed buildings (Mølhave, 1991). These studies, moreover, showed effects at a range of concentrations at which such effects would not have been predicted on the basis of existing toxicological information about the individual components. Conversely, controlled field exposures to a healthy and sick preschool environment failed to show any effect on memory, motor and vigilance tests (Berglund et al., 1992). One important factor in such studies is the selection of subjects, for example, previously exposed or unexposed, visitor or occupant, trained or untrained, etc.

Interference of carbon monoxide with the performance of complex sensory-motor tasks or with functions such as visual perception, manual dexterity and ability to learn has been demonstrated to occur at carboxyhaemoglobin levels between 5 and 17% in several human studies (Davies et al., 1981; Dolan, 1985; Luria and McKay, 1979). It cannot be excluded that such effects may appear at lower levels in susceptible subjects. Concentrations of carboxyhaemoglobin associated with effects on the CNS are of the same order of magnitude as those reported to occur in some indoor environments polluted from sources, such as improperly ventilated or adjusted combustion appliances or garages.

The neurotoxicity of several pesticides is well documented from animal studies. Human investigations are few and their results are not always easy to interpret. Specific investigations on the neurotoxic potentials of non-industrial, indoor pesticide exposure are totally lacking.

11.6 SUSCEPTIBLE GROUPS

There are large differences in sensory sensitivity between individuals, especially for sensory irritation in the airways, and also for odour if compared with vision or audition. By aging, the sensitivity is known to decrease in most individuals for odours but for sensory irritation data is lacking.

Foetuses and children are particularly at risk for neurotoxic effects as a result of the sensitivity of the brain during its growing stages.

Many field studies and some experimental exposure studies indicate significant difference in sensitivity between woman and man (gender effect). Sometimes this gender effect disappears when proper corrections are made in the analysis regarding cofactors such as occupational position, age, location of workplace in building.

11.7 PUBLIC HEALTH RELEVANCE

As pointed out by WHO (1987) in their "Air Quality Guidelines for Europe", many substances in the indoor environment may cause sensory effects at concentrations far below those at which toxic effects occur. Also, sensations are the integrated net response of the body to a large number of interacting components and the effects appear at an early stage. Some pollutants lack sensory warnings and behave differently from the sensorily stimulating ones, precluding the sole use of sensory perception as an indicator of effect. In these cases, the pollutants must be controlled by other means.

An example of guideline to control human health risk based on sensory criteria is the WHO document on environmental health criteria for formaldehyde (WHO, 1989b). The argument applied is that human exposure to formaldehyde should be minimized not only for its possible carcinogenic effects (nasal cancer in rats), but also for its potential to cause irritation. WHO recommends that in order to avoid strong sensory reactions in work-place environments where formaldehyde is being produced or used, peak concentrations above 1.0 mg/m^3 should not be allowed and mean concentrations should be kept below 0.3 mg/m^3. With regard to exposure outdoors and in the non-industrial indoor environment, formaldehyde concentration should not exceed 0.1 mg/m^3 in order to avoid odour and sensory irritation for the general population. In the case of highly sensitive groups, that show hypersensitivity reactions without immunological signs, formaldehyde concentration should be kept to a minimum and not exceed 0.01 mg/m^3.

The prevalence of neurotoxic effects in the population as a result of indoor exposure to neurotoxic air pollutants is impossible to estimate because there is no documentation of neurotoxic effects (or absence of them) clearly linked to indoor exposure.

A large part of the general population is exposed to low concentration of VOCs. Some individuals may experience higher exposures during indoor use of certain products or after refurnishing or home redecoration.

Carbon monoxide exposure may be important depending on the type of heating and combustion appliances in use and on the ventilation of the building.

Pesticide exposure may be a risk factor in homes in rural areas as well as in homes where pesticides have been used indoors. Pentachlorophenol, a neurotoxic wood preservative has been found at elevated concentrations in the urine of subjects living in houses with indoor wood surfaces, even 10 years after the application of this chemical (Maroni et al., 1987).

11.8 METHODS FOR ASSESSMENT OF SENSORY EFFECTS AND NEUROTOXICITY

Sensory effects such as odour and mucosal irritation are perceptions and therefore, by definition, subjective in nature. The assessment of such subjective aspects of sensory stimulation must involve humans. The olfactory system adapts during prolonged exposure and olfactory measurement should control for this adaptation. As a consequence of adaptation to any odour in indoor air, two different responses may be identified: that of the visitor and that of the occupant. A WHO expert group (WHO, 1982) has recommended that odours should be measured through the immediate response of the unadapted olfactory system (visitor situations). It should be noted that odour intensity measured by visitors does not necessarily correlate with the perceptions of the occupants.

A number of indicators or substitute measures may be used to estimate or predict the odour or mucosal irritation potency of environmental chemicals. The concentration of total volatile organic compounds has been suggested as a possible indicator of the overall perceived indoor air quality. Carbon dioxide emitted by human metabolism is often used as an indicator for perceived indoor air quality as affected by bioeffluents.

The science of psychophysics offers a variety of sensory models for studying indoor air quality effects and for indoor air quality characterization (bio-assays). Regulatory agencies now require sensitivity, validity, reliability and biological meaningfulness of sensory methods applied for indoor air quality control. Therefore, quality assurance in sensory measurements is mandatory (Berglund, 1990).

Individuals, panels and populations do differ in sensory sensitivity, response behaviour and value judgements. Some of these differences are environmentally induced. It is important to specify the target groups of indoor air quality control based on sensory effects and how they relate to large population groups.

Methods for assessing neurotoxicity in animal and human studies include a number of neurophysiological and behavioural diagnostic techniques designed to study selected central or peripheral nervous functions. Examples of neurobehavioural tests are reaction time, memory, manual dexterity, etc., and examples of electrophysiological techniques are measurements of visual or auditory evoked potentials, nerve conduction velocity, etc. These tests are increasingly being used in the field because of their non-invasive nature. Neurobehavioural tests can be used in experimental laboratory settings as well as in epidemiological studies. However, the attribution of abnormal results to acute reversible impairments or to irreversible brain lesions may be difficult.

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Chapter 12

Effects of Indoor Air Pollution on the Cardiovascular System and Other Systemic Effects

Cardiovascular effects have only infrequently been described as being associated with exposure to IAP. Only exposures to ETS and carbon monoxide (CO) have been implicated in cardiovascular symptoms, and in changes in cardiovascular disease (CVD) morbidity and mortality.

12.1 CARDIOVASCULAR EFFECTS ASSOCIATED WITH INDOOR AIR POLLUTION

Organs with a high oxygen demand, such as the heart and the brain, are particularly susceptible to reduced oxygenation caused by CO exposure. Early effects include reduction of time to onset of chest pain in exposed exercising heart-disease patients. At levels of exposure higher than those normally occurring indoors, myocardiac infarctions may be triggered by CO (Anderson et al., 1967; WHO, 1987). Carbon monoxide (CO) exerts its influence primarily through binding to the haemoglobin (Hb) in blood. The affinity of CO and Hb is about 200 times higher than the affinity of oxygen to Hb, so that at relatively low levels of CO in the air, oxygen is replaced by CO. The percentage of Hb bound to CO (% carboxyhaemoglobin) is a measure of recent exposure to CO.

Increased mortality due to CVD has been associated with exposure to ETS in some groups of non-smoking women married to smokers (Garland et al., 1985; Humble et al., 1990; Steenland, 1992). Some investigators have also addressed the question whether total mortality is influenced by exposure to ETS, but results have been contradictory. As any effect on mortality would not be expected to occur until after many years of exposure, a problem in these types of study is the accuracy and reliability of the exposure classification. Attempts have also been made to relate ETS to electrocardiographic abnormalities and cardiovascular symptoms, but results have been inconclusive.

12.2 OTHER SYSTEMIC EFFECTS ASSOCIATED WITH INDOOR AIR POLLUTION

Systemic effects are defined as adverse effects on one or more target organs caused by absorption and distribution of a toxic agent within the body. They include effects such as gastrointestinal, hepatic or renal effects, immunosuppressive effects and a group of miscellaneous effects. Such systemic effects have never been documented but have only been suggested in relation to IAP. At present no firm evidence exists for a causal link between these effects and exposure to non-industrial IAP.

The gastrointestinal tract is the first system in contact with chemicals contained in food and drinks. In addition, through the liver and biliary system, the gut provides a route for excretion of toxic chemicals, drugs, and products of metabolism. More important, in relation to indoor air, is the secondary swallowing of inhaled pollutants originally trapped in the airways (mucociliary clearance) and then transported to the throat. Due to transportation time in the body and dilution of the pollutant in body fluids, few acute systemic effects are expected as a result of exposure to the low levels of pollutants occurring indoors. The main effects, if they exist, would be expected to be subchronic or chronic, due to long-term accumulation of chemicals or to accumulation of small, irreversible effects at cellular or molecular level.

12.3 PRINCIPAL AGENTS AND SOURCES

ETS and carbon monoxide (CO) are the main pollutants, of indoor air that have been associated with cardiovascular effects (Anderson et al., 1967; Garland et al., 1985). Active smoking is a well-known cause of cardiovascular disease. Carbon monoxide present in tobacco smoke is one of the likely causal agents, as smokers are known to have chronically elevated levels of carboxyhaemoglobin in their blood. Whether or not CVD effects resulting from exposure to ETS are related to CO is questionable, as CO concentrations are not very elevated by tobacco smoking unless ventilation is restricted. Non-smokers exposed to ETS, generally, do not have significantly elevated levels of COHb in their blood.

Exposure to lead in the general environment has been related to high blood pressure in adults, but exposure of adults to lead is mostly through the food chain, and not so much via indoor air.

Gastrointestinal system

Gastrointestinal symptoms caused by inhalation of toxic substances at concentrations found in normal indoor air have not been documented.

Hepatic effects

These effects have been related to pesticides and other organic compounds such as pentachlorophenol applied in and around buildings to control insects or microbiological growth. Investigations have not been extensive and reliable exposure estimates or exclusion of other potential causes were lacking in the cases reported. Hepatotoxic effects might be possible from other types of organic compounds, such as halogenated VOC. No epidemiological investigation has, however, been performed to relate exposures to such health effects.

Renal effects

Renal damage can be caused by many chemical compounds. Cadmium is among the most well known environmental contaminants linked to renal disease. No renal effects of exposure to normal indoor air have been documented.

Other health effects

ETS has been related to impaired growth and reduced general health of children exposed to it. Most studies, however, have been unable to differentiate effects of *in utero* exposure from childhood exposures to ETS. It has recently been suggested that non-smoking pregnant women exposed to ETS for several hours on a daily basis are at increased risk for producing babies of low birth weight (Lazzaroni et al., 1990; Rubin, et al., 1986).

Exposure to ETS in homes has been linked with increased incidence rates of acute and chronic ear infections and middle-ear effusions in young children (Iversen et al., 1985; Kraemer et al., 1983; Pukander et al., 1985).

Studies on systemic immunosuppressive effects of indoor pollutants are lacking.

12.4 EVIDENCE LINKING INDOOR AIR POLLUTION TO EFFECTS ON THE CARDIOVASCULAR SYSTEM

Few studies have addressed the issue of IAP and CVD, although some epidemiological studies have addressed the relationship between exposure to ETS and mortality and morbidity due to CVD. As discussed in recent, major reviews (Samet et al., 1987a,1988; US Surgeon General, 1986), the evidence obtained from these studies is limited or inconclusive. However, recent cohort studies among non-smokers have suggested that living with a smoker is associated with a significantly increased relative risk of dying from CVD (Garland et al., 1985; Hole et al., 1989).

The effects of CO on cardiovascular symptoms such as time to onset or aggravation of angina symptoms in angina pectoris patients have been well

documented in human studies (Anderson et al., 1973; Aronow et al., 1984).

The level of knowledge about systemic effects on humans due to indoor air is very low and, at present, does not allow conclusions about the risks associated with exposure to indoor air in normal non-industrial buildings.

12.5 SUSCEPTIBLE GROUPS

Persons with angina pectoris or with obstructed coronary arteries should be considered to be a susceptible group. Other susceptible groups are those with disorders (e.g. anaemia) which reduce the oxygen carrying capacity of the blood.

There is no information about susceptible groups in relation to systemic effects caused by indoor air. It may be expected that groups showing hypersusceptibility to other types of environmental exposures causing systemic effects may be risk groups for exposure to the same compounds in the indoor air. Such groups will often be characterised by age, genetic factors such as gender or race, smoking habits, hypersensitivity status, general health conditions and occupation.

12.6 PUBLIC HEALTH RELEVANCE

Many people are exposed to ETS. CVD is a major cause of morbidity and mortality in most developed countries. Even when relative risks are moderate, the number of people which could be affected by ETS in this respect is potentially large. The final assessment of the public health relevance awaits conclusive studies of this issue.

CO levels indoors do not usually exceed recommended levels as published by WHO (1987). Therefore, the public health relevance of indoor CO pollution in causing or aggravating CVD is probably limited. Nevertheless, CO is known to reach high levels in some homes when a number of unfavourable circumstances combine. Effects of CO are unspecific and may go undetected by physicians and other health professionals. It is considered by some to be "an old enemy forgot", and it is one of the few indoor air pollutants that kills several hundred and possibly thousands of persons each year (Anonymous, 1981; Meredith et al., 1988).

No documentation is available to assess the relevance of systemic effects caused by indoor air for public health.

12.7 METHODS FOR ASSESSMENT OF EFFECTS ON THE CARDIOVASCULAR SYSTEM AND OTHER SYSTEMIC EFFECTS

The main study types mentioned in the introductory chapter of this section are applicable here as well. Mortality studies typically rely on death certificates which may not always contain the correct diagnosis. Morbidity studies use symptoms questionnaires, ECG measurements, and measurement of serum cholesterol and of blood pressure. CVD has been the subject of an extremely large number of studies, and the methods to investigate symptoms and physiological and biochemical variables are well developed and standardised.

There are several tests available for the detection of gastrointestinal diseases and some for the identification of persons at risk. The applicability of these methods for detection of possibly subtle effects associated with IAP has yet to be documented.

A large number of tests of hepatic function are available and could be tailored to the particular objective of any experimental or epidemiological study. Again, the applicability of these methods for detection of possibly subtle effects associated with IAP has to be documented.

The early stages of renal damage are seldom accompanied by symptoms. Thus, questionnaires are useless in the early detection of renal impairment, and laboratory tests, such as those measuring changes in urinary sediments and glomerular and tubular function, are necessary for carrying out investigations.

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PART III
Risk Assessment

This part presents the methodology of risk assessment for indoor pollutants. The various steps of the method are discussed, highlighting the available procedures and the specific characteristics of their application to the indoor environment. Examples of application are provided for radon, environmental tobacco smoke and carpets.

The contents of this part have been excerpted from presentations made at the NATO/CCMS–CEC workshop "Methods of Risk Assessment for the Indoor Environment" held in Kloster Banz, Germany in 1991, the proceedings of which are available.

The text has been prepared by T. Pierson and D. Moschandreas. The originating authors and sources of each chapter are indicated in the text.

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Indoor Air Quality
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Chapter 13

Risk Assessment: Hazard Identification and Dose Effect Assessment[1]

13.1 GENERAL ASPECTS OF RISK ASSESSMENT

13.1.1 Introduction

Risk assessment is a tool that has been used increasingly in the fields of environmental science and health to identify the probability of injury, disease, or death from exposure to agents (chemical, physical or biological) under specific circumstances.

Risk assessment can also be used to evaluate the net risk associated with a particular policy. For example, before water was chlorinated, many deaths occurred from infectious disease outbreaks such as typhoid and cholera. More recently, the use of chlorine to disinfect water resulted in an increase of the rate of formation of halogenated hydrocarbons. Risk analysis can be used to compare the risk of illness associated with not chlorinating water to the increased risk of cancer from chlorination by-products. When risk assessment is used, the uncertainty associated with it must always be acknowledged.

Useful references for additional information on risk assessment include several articles on cancer risk assessment (Aust, 1991; Hoffman, 1991; Cerutti et al, 1990; Krewski, 1987; OSTP, 1985; IARC, 1984) and guidelines published by EPA for conducting exposure assessment and assessing risks from carcinogens, mutagens, teratogens and chemical mixtures (U.S. EPA, 1986a–e). EPA's risk assessment methodologies are currently under review and revised guidelines are anticipated.

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1 A part of the text of this chapter has been derived from W. Wosniok "Dose–Response Models for Cancer-Inducing Agents" in: Report on a Joint Workshop "Methods of Risk Assessment for the Indoor Environment", pp. 29–46, Kloster Banz, Germany, 15–17 October 1991. Ed. B. Seifert. NATO/CCMS Pilot Study on Indoor Air Quality and ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man").

13.1.2 Assessment versus management of risk

Once the risk associated with exposure to an agent has been defined, a policy can be developed to manage that risk. In the environmental and health fields, risk assessments are generally performed by scientists and engineers, and the management of risk is usually a decision made by a governmental agency.

Risk management is a socio-political decision on whether or how much to control future exposure to the contaminant or activity under consideration. The risk assessment portion of the decision-making process should be carried out independently from considerations of the consequences of risk management. The risk management decision depends in part on the risk assessment, but also on the consideration of social, economic, and political factors. An example of economic factors to be considered in the risk management process is given in Chapter 38.

13.1.3 Measures of risk

Risk assessments are used to measure the likelihood of a specific effect, such as death from acute hazards (in early deaths/year), cancer (in early deaths/year), and various types of chronic conditions (in cases/year).

Risk can be measured for a population or an individual. A population risk is the number of occurrences of a hazard per year within a given population. For example, assume there were 15 million automobile accidents in the U.S. in 1988; of these, assume that about 1 in 300 resulted in the death of an individual. The population risk is $1/300 \times 15$ million, or 50,000 deaths/year.

The individual risk is the probability of a single occurrence affecting an individual during a year or life time. For the automobile accident example, the individual risk of dying as a result of an automobile accident is about 50,000 deaths/year/250 million people. This means that an individual has a 2 in 10,000 chance (or 1 in 5000) chance of dying as a result of an automobile accident.

The above examples are quantitative estimates of risk, but risk can also be expressed in qualitative terms using categories such as "low", "medium", or "high".

13.1.4 Perception of risk

There is a difference between the measure of risk and the perception of risk. The measurement of risk is based upon an estimated likelihood or frequency of an effect. The perception of risk is based upon a societal-political or personal interpretation and acceptance of the hazard posed by a situation or chemical exposure. For example, people who drive cars, but do not wear seatbelts, may

perceive their risk to be less than the actual risk of driving without wearing seatbelts. On the other hand, the perceived risk of flying may be considerably higher than the actual risk for those people who are afraid to fly.

Risks can be voluntary or involuntary and may result from natural or synthetic substances. In general, people are willing to accept higher risks from voluntary exposure to agents (for example, smoking) than from involuntary exposure (for example, pesticide residues in food). Also, people might be willing to accept a cancer risk of 1/10,000 or 1/100,000 as a result of eating peanut butter containing low levels of naturally occurring aflatoxin, but they might reject having a synthetic chemical with a cancer risk of 1/100,000 in their water supply.

13.1.5 Risk communication

The goal of risk communication is to produce an informed public that is involved and interested in the issues and willing to work jointly to produce reasonable and thoughtful solutions. Effective risk communication is particularly important for the issue of indoor air quality.

Effective communication of indoor air risks is important for three major reasons. First, because indoor air pollution affects all individuals there is a high level of public interest in the problem. This interest creates a need for information on the overall risk to the public and how this risk compares to other environmental and non-environmental risks. Effective risk communication, therefore, allows the public to put the risks in perspective so that they can deal with the risks rationally.

Second, remedial actions for many indoor air problems are outside of regulatory controls and rely heavily on public participation. Public understanding of the potential risks associated with indoor air quality problems and active participation by individuals are necessary to facilitate solutions.

Third, both personal and institutional credibility depends on the ability to communicate risk. If the public perceives that a balanced and rational approach has been used in assessing indoor air risks and their possible solutions and in relaying this information to them, they will be more likely to accept the information provided as reliable and respond positively.

Risk communication for indoor air problems relies on the same basic principles used for all risk communication strategies. Risk communication experts have developed the following seven cardinal rules of risk communication.

- *Accept and involve the public as a legitimate partner.* People and communities have a right to participate in decisions that affect their lives, property, and the things they value. Respect for the public's right is demonstrated by involving all interested parties early.

- *Plan carefully and evaluate your efforts.* Different risk communication goals, audiences, and media require different risk communication strategies. Clear explicit risk communication objectives (e.g., motivating individuals, contributing to the resolution of conflict) are a necessity, as is knowledge of the strengths and weaknesses of the risk information.
- *Listen to the public's specific concerns.* People are often more concerned about issues such as trust, credibility, or compassion than about mortality statistics. It is necessary to find out what people are thinking, to put yourself in their place, and to recognize "hidden agendas", symbolic meanings, and broader economic or political considerations that often underlie and complicate risk communication.
- *Be honest, frank, and open.* Once lost, trust and credibility are almost impossible to regain. Risk information should be disclosed as soon as possible, along with its associated uncertainties, strengths, and weaknesses. Mistakes or incomplete knowledge in certain areas should be admitted.
- Coordinate and collaborate with other credible sources. Few things make risk communication more difficult than conflicts or public disagreements with other sources. Coordination within the organization and with other organizations and joint communications with other trustworthy sources increase credibility.
- *Meet the needs of the media.* The media are prime transmitters of information on risk; they play a critical role in setting agendas and in determining outcomes. Complex risk information should be prepared in advance, along with background materials, and tailored to meet the media's needs.
- *Speak clearly and with compassion.* Technical language and jargon are barriers to successful communication with the public. Risk comparisons to put risks in perspective should acknowledge distinctions that the public views as important, such as voluntariness, controllability, benefits, and catastrophic potential. The tragedy of any illness, injury, or death should be acknowledged.

Several other basic points can be made regarding risk communication. For example, environmental risk communication is based on the concept of voluntary vs. involuntary risks. The public is more likely to accept risks that are voluntary (e.g., from the use of a consumer product) than those that are involuntary (e.g., from passive smoking or radon), because certain personal benefits are usually obtained from the voluntary exposure.

In addition, everything is either safe or unsafe to the public. Caveats and shadings of grey are seldom heard or accepted by the public. This may be a problem in making risk comparisons or relaying information on uncertainties to the public.

Finally, to the news media, if it is not bad news, it is not news. The media is more likely to publish information on the risks of an environmental problem than on any subsequent risk reductions. In addition, if risks are overstated in the media, it may be difficult to inform the public of the actual risks.

13.1.6 The risk assessment process

The subject of this part of the book is risk assessment. The basic elements of risk assessment which are identified in the guidelines include one or more of the following: hazard identification, dose response evaluation, exposure assessment, and risk characterization. Examples used in the discussion of these elements below are taken predominantly from carcinogenic risk assessment, not because it is more important, but because this area is the most developed.

13.2 HAZARD IDENTIFICATION

Hazard identification is the process of determining whether or not exposure to an agent can cause an increase in the incidence of a health condition. This involves a review and analysis of available scientific information to determine whether or not an agent poses a particular risk.

Information which is used to identify hazards is obtained from cellular and tissue studies, animal studies, controlled human exposure studies, case studies of humans exposed in accidents or in the workplace, and epidemiologic studies.

13.2.1 Carcinogenic agents

A hazard identification to determine whether or not a contaminant poses a carcinogenic risk should include information on the nature of contaminants, degradation products, and metabolites. The available scientific literature should be reviewed for the following key elements:

- physical and chemical properties which affect the distribution and decay of the chemical in the body;
- routes and patterns of exposure;
- structural or activity properties of the chemical or its metabolites that support or argue against the prediction of potential carcinogenicity;
- metabolic and pharmacokinetic properties of the chemical which determine how the chemical is distributed, metabolized, and excreted in the animal or human;

- toxicologic effects other than carcinogenicity, interactions with other chemicals or agents and with lifestyle factors, and other factors relating to toxicologic effects;
- short-term predictive tests which detect chemical interactions with DNA and assess mutagenic activity;
- long-term animal studies which identify the tumorigenic or
- carcinogenic potential of the chemical; and
- human epidemiologic studies which examine the association between the chemical and the incidence of cancer.

Of these elements, animal and human epidemiologic studies have been the most widely used sources of data to evaluate hazards, but they do have limitations.

Animal studies are used because it is assumed that effects in humans can be inferred from effects in animals. This assumption is generally accepted because all the chemicals that have been demonstrated to be carcinogenic in humans (except possibly arsenic) have been shown to be carcinogenic in some, but not all, animal studies. However, scientists must be careful in how such studies are interpreted because there are potential differences in the way different species metabolize, distribute, and excrete chemicals.

The overall confidence that an agent which is carcinogenic in animals will be carcinogenic in humans increases with the following types of evidence:

- an increase in the number of animal species and strain showing an effect, and with both sexes showing an effect;
- an increase in the number of tissue sites affected by the agent;
- the presence of a clear-cut dose–effect relationship;
- a high level of statistical significance of the increased tumour incidence in treated compared to control groups;
- a dose-related shortening of the time for tumours to form or death to occur as a result of tutors; and
- a dose-related increase in the proportion of tumours which become malignant.

Human epidemiologic studies which are properly designed and conducted provide the most direct information about the toxicity of a particular contaminant. These studies, however, are not available for most chemicals because of the complexity and cost associated with gathering these data. Epidemiologic studies require large sample sizes for statistical reasons, and long time periods are needed to observe effects such as cancer. It is also difficult to quantify exposures because confounding variables such as socioeconomic effects, personal habits such as smoking and drinking, and other risk factors may not be known or accounted for quantitatively.

Because of the problems associated with epidemiologic studies, animal

studies and the other types of evidence listed above are usually used to derive estimates of cancer risk in humans.

The weight of evidence is reviewed carefully for technical adequacy and then given an overall classification system including 5 groups in descending order of overall weight of the evidence:

Group A (Carcinogenic to Humans): Used when there is sufficient evidence from epidemiologic studies to support a finding that a causal relationship exists between exposure to the agent and cancer;

Group B (Probably Carcinogenic to Humans): Used when there is enough evidence of carcinogenicity based on animal studies and limited epidemiological evidence (Group B1) or inadequate or no human data (Group B2);

Group C (Possibly Carcinogenic to Humans): Used when there is limited evidence for carcinogenicity in animals and inadequate or no human data;

Group D (Not Classifiable as to Human Carcinogenicity): Used when there is inadequate animal evidence of carcinogenicity and inadequate or no human data; and

Group E (Evidence of Non-carcinogenicity in Humans): Used when there is no evidence for carcinogenicity in at least two adequate animal tests in different species or in both adequate epidemiologic and animal studies.

13.2.2 Non-carcinogenic agents

Hazard identification for non-carcinogenic agents operates in a very different environment. The outcomes of concern are usually not dichotomous, but show grades and progression over time. There are many outcomes, which at least in the beginning, are reversible, and often there is a considerable range of sensitivity in the population. This means that the determination of the outcome is much more complex, and the outcomes are not immediately life-threatening. Often several outcomes may be associated with a single contaminant, and similarly, a single outcome may be affected by several contaminants.

The above general considerations can be expanded into a number of important factors for non-carcinogens:

1. The adverse outcomes range from odour annoyance to debilitating effects in persons with pre-existing disease. This makes the specification of a standard outcome to be assessed very difficult. A population will contain a wide range of outcomes. Background levels of these outcomes are difficult to establish, so that the baseline or threshold for a population is also difficult to describe.
2. Many exposures can produce adverse consequences in a number of systems, so that several outcomes need to be assessed simultaneously.

13.3 DOSE–EFFECT ASSESSMENT

Dose–effect assessment is a critical part of the overall risk assessment process. It attempts to quantify the relationship between the dose and adverse effects expected in humans. Ideally, human epidemiologic data should be used to develop the dose–effect relationship for carcinogenic and non-carcinogenic effects. In practice, however, these data are usually not available, and data from animal studies are used.

13.3.1 Toxicology and animal factors

There are three problems associated with the use of animal data which must be considered in the dose–effect assessment. First, animals are usually exposed at high doses, which means that effects at low doses must be extrapolated. Second, animals and humans differ in susceptibility. Third, there are some individuals in the population who may be more susceptible to effects than the average person.

The extrapolation of effects at high doses to those at low doses usually involves one of two assumptions: (1) biological effects occur after a threshold dose has been reached; or (2) there is a linear relationship between the dose and the effect. Effects which involve the alteration of genetic material (genotoxic effects) including the initiation of cancer are assumed to be non-threshold effects; that is, an effect is predicted at all non zero exposure levels. A threshold dose is assumed for effects which are not genotoxic.

These assumptions, however, may need to be revised for some substances as new methods of assessing dose–effect relationships are developed. One of these new methods is the use of physiologically based pharmacokinetic modelling, which compares the sensitivity of animal and human cells and then relates these via dosimetry models to human and animal species sensitivity (Menzel, 1987). Whenever these new models appropriately simulate real effects, they may be preferable to the traditional threshold versus non-threshold models.

Regardless of the assessment methodology, numerical estimates of the dose-assumptions and uncertainties should be included. The risk characterization should include a discussion and interpretation of the numerical estimates that provide the risk manager some insight into the degree to which the estimates are likely to reflect the true magnitude of the risk to humans.

13.3.2 Non-carcinogen dose–effect assessment

For non-carcinogens, the threshold dose in animal studies is approximated by the no-observed-adverse-effect level, NOAEL (expressed in mg/ kg/day).

The NOAEL is selected in the context of the entire data set. Uncertainties in the NOAEL are compensated by dividing the NOAEL by an uncertainty factor (previously called the safety factor) which may be 10 (or less), 100, 1000, or 10,000. The uncertainty factor dates to the early days of food additive legislation when it became clear that there was no universally accepted quantitative method of extrapolating from animal data to humans. The selection of the uncertainty factor is a professional judgment that depends on the nature and quality of the data, the seriousness of the effect, the type of effect, and the population to be protected.

The NOAEL can be used to derive the reference dose (RfD) for humans. The reference dose, which was formerly called the acceptable daily intake (ADI), can be expressed as an oral reference dose (mg/kg/day) or inhalation reference concentration (mg/m^3). The RfD is a dose (from contaminants in food, water, and air) that is anticipated to be without risk to humans over a lifetime of exposure. The RfD is derived by dividing the NOAEL for the toxic effect appearing at the lowest dose by the uncertainty factor. An additional modifying factor which comments on the quality of the data may also be included. The RfD is an estimate of risk (with uncertainty that spans perhaps an order of magnitude). It is not a guarantee of absolute safety, and it may overestimate the risk in some instances.

In those instances when the NOAEL cannot be identified, the RfD might be calculated with the lowest observed-adverse-effect level (LOAEL).

The assessment should be accompanied by a confidence statement that comments on the quality of the study that drives the assessment, the underlying data base and the degree to which the selected study agrees with the data base, and overall adequacy of the assessment.

13.3.2.1 Models for non-carcinogens

General dose–response modelling methods have been proposed for non-cancer endpoints (Crump, 1984; Dourson et al., 1985). These methods were originally proposed as alternative methods to the traditional Reference Dose (RfD) approach for establishing protective exposure levels. Additionally, however, the use of dose–response modelling allows for the estimation of risks to various types and severities of health effects due to varying exposure levels within a population. Models have been proposed for use with quantal and continuous endpoints for non-cancer health effects.

With models for both quantal and continuous endpoints, dose–response curves can be estimated for any given exposure duration for which there are adequate dose–response data. When responses vary by exposure duration, dose–response models can be extended to incorporate exposure duration as a variable

in the model. Below is a brief description of several dose–response models which may be used with each of continuous, binary and ordinal-type response diverse, complex, and not well understood. Therefore, non-cancer dose–response models do not attempt to describe these biological mechanisms, but rather statistically based "generic" models are used to describe experimental data.

Dose–response model for continuous response data

For modelling continuous responses one can assume that the responses within each dose group of animals are normally distributed, and apply maximum likelihood (ML) estimation methods to estimate the parameters of the dose–response models. Several models can be used including the continuous linear regression model (CLR), the continuous polynomial regression model (CPR) and the continuous power model (CP). Expressions for these models are given below.

The continuous linear regression model (CLR):

$$m(d) = c + q_1 (d - d_0) \quad \text{for } d \geq d_0$$

$$= c \quad \text{for } d < d_0$$

where: $m(d)$ = mean (or expected) response at dose d; c = background response rate; d_0 = threshold dose $d_0 \geq 0$; and q_1 = the dose–response parameter.

The continuous polynomial regression model (CPR):

$$m(d) = c + q_1 (d - d_0) + \ldots + q_k(d - d_0)^k \quad \text{for } d \geq d_0$$

$$= c \quad \text{for } d < d_0$$

where the $\{q_1\}$, $i = 1,2,\ldots,k$, are all positive or all negative, and the continuous power model (CP):

$$m(d) = c + q_1 (d - d_0)^k \quad \text{for } d \geq d_0$$

$$= c \quad \text{for } d < d_o$$

Another option is to model the mean of the log of the response variable as a linear function of the log of the dose. This model is referred to as the log–log linear model.

Dose–response models for binary response data

Models which have been proposed for modelling binary response outcome (i.e., presence or absence of an effect) for non-cancer data include:

the quantal linear regression model (QLR):

$$p(d) = c + (1 - c)\left[1 - \exp[-q_1(d - d_0)]\right] \quad \text{for } d \geq d_0$$

$= c$ for $d < d_0$

where: $p(d)$ = probability of response at dose d; c = background response rate $0 \leq c \leq 1$; d_0 = threshold dose $d_0 \geq 0$; q_1 = the dose–response parameter;

the quantal polynomial regression model (QPR):

$$p(d) = c + (1 - c)\left[1 - \exp[-q_1(d - d_0) - \ldots q_k(d - d_0)^k]\right] \quad \text{for } d \geq d_0$$

$$= c \quad \text{for } d < d_0$$

the quantal Weibull model (QW):

$$p(d) = c + (1 - c)\left[1 - \exp[-q_1(d - d_0)^k]\right] \quad \text{for } d \geq d_0$$

$$= c \quad \text{for } d < d_0$$

where $k \geq 1$;

the quantal log-normal (probit) model (LN):

$$p(d) = c + (1 - c)\, N[a + q_1(\log d) - (\log d_0)] \quad \text{for } d \geq d_0$$

$$= c \quad \text{for } d < d_0$$

where $N(t)$ = the standard normal distribution function evaluated is

$$N(t) = \int_0^t \frac{1}{2\pi} \exp\left(\frac{1}{2} y^2\right) dy$$

the logistic model:

$$p(d) = c + (1 - c)\left[1 - \exp[-a - q_1 (d - d_0)]\right]^{-1} \quad \text{for } d \geq d_0$$

$$= c \quad \text{for } d < d_0$$

the log–logistic model:

$$p(d) = c + (1 - c)\left[1 - \exp[-a - q_1 \log(d - d_0)]\right]^{-1} \quad \text{for } d \geq d_0$$

$$= c \quad \text{for } d < d_0$$

and the hockey-stick model (e.g., see Cox, 1984):

$$= c \quad \text{for } d \leq d_0$$

$$p(d) = c + q_1(d - d_0) \text{ for } d_0 < d < (1 - c)/q_1 + d_0$$

$$= 1 \quad \text{for } d \geq (1 - c)/q_1 + d_0 = d_u$$

where d_u is the upper threshold dose or saturation point.

Models for ordinal response data

Non-cancer health effects involve multiple target organs and, consequently, are characterized by multiple endpoints with varying levels of severity. Many times the severity of these non-cancer effects is not measured quantitatively, but reported in a descriptive or qualitative manner. This qualitative description of effects can be used to categorically define varying levels of severity. Severity of effects are typically divided into none, mild (non-adverse), moderate, and severe, corresponding to the common-effect exposure levels of No Observed Effects Level (NOEL) No Observed Adverse Effects Level (NOAEL), Lowest Observed Adverse Effects Level (LOAEL), and Frank Effects Level (FEL). Severity levels can be a useful means of describing the information contained in each animal's set of responses.

Typically, published accounts of toxicity studies report only the types of effects seen in each dose group rather than the effects seen in each animal (Hertzberg, 1989). Consequently the dose group is forced to be the unit of study, rather than the individual animal, when such data are used.

Categorical regression techniques based on severity level responses have been proposed as a means of estimating a dose–response relationship using the dose group as the unit of analysis (Hertzberg, 1989). The response variable under this methodology is based on a multiple categorical variable describing the overall health response within a dose group. The dose–response relationship is estimated using ordinal regression techniques with the dependent (response) variable defined by categories of severity, which are ordinal by nature. Initial efforts have focused on defining severity levels as NOEL, NOAEL, LOAEL and FEL. These severity levels are assigned scores of 1 to 4, respectively, which reflects the increasing severity over the defined levels. Severity for each dose group, thus defined, is used as the response variable in the ordinal regression techniques described below.

Define $P(S \leq i)$ to be the probability (or risk) of observing a response in severity level i or a lower severity level, $i = 1, 2, 3, 4$. Then, $P(S >) = 1 - P(S \leq i)$ is the probability of observing a response more severe than level i. A general class of models based on a linear relationship between some function f (called the "link" function) of $P(S \leq i)$ and dose is defined by McCullagh (1980) as:

$$f[P(S\ i)] = c_i - q_i\, d, \quad i = 1,2,3,4$$

where c_i is a constant for level i. Note that d can be replaced by $\log d$. Model (E.1) is a non-threshold model; the corresponding threshold model is

$$f[P(S \ i)] = c_i - q_i(d - d_0) \quad \text{for } d > d_0$$

$$= c_i \quad \text{for } d \leq d_0$$

There are several choices for the link function. One widely used is the logit transformation, corresponding to a logistic model for model (E.1):

$$\log[P(S \ i)/(1 - P(S \ i))] = c_i - q_i \, d$$

which can be rewritten as:

$$P(S > i) = [1 + \exp(c_i - q_i \, d)]^{-1}$$

This is referred to as the proportional odds model since the odds of event ($S \leq i$) at different dose levels is independent of i, the severity level.

The fitted dose–response curve allows estimation of the probability that a given dose will result in an adverse effect (i.e., under the previously defined severity levels, $P(S > 2)$, the probability that an effect would be either moderate or severe). If data on different exposure durations as well as dose levels are available, a dose–duration response curve can be estimated. A description of such models follows in the next subsection.

The application of categorical regression techniques for dose–response modelling of non-cancer health effects is not limited to the scoring of severity as described above. The assignment of dose groups into the four severity levels relies on professional judgment and, therefore, problems of inconsistency may arise. A more detailed severity scoring system can be used, such as those proposed by Hartung and Durkin (1986) and Tallarida et al. (1979) which increase the number of severity levels to five by adding a level corresponding to an Adverse Effect Level (AEL).

13.3.3 Carcinogen dose–effect assessment

There are three main components to the dose–effect assessment for carcinogens:

1. the appropriate data base is selected based on the quality of the data, its relevance to human modes of exposure, and other factors;
2. mathematical models are used to extrapolate from high to low doses of exposure; and
3. appropriate scaling factors are used to extrapolate from animal studies to human exposure.

In the absence of human data, data are typically used from a species that responds most like humans or has in the long-term studies shown the greatest sensitivity.

Several mathematical models (linearized multistage model, one-hit model,

gamma multi-hit model, log-probit model, and other time-to-response models such as the Weibull model) have been used to extrapolate the risks from high doses to those at low doses. No single model is recognized as most appropriate for all carcinogens (U.S. EPA, 1986a). The selection of a specific model depends on the underlying mechanism that results in the development of cancer, and if such data are available, the selected model should be consistent with these data. Unfortunately, the way in which cancer develops is not understood for most carcinogens. In the absence of these data, EPA recommends the use of the multistage model, which assumes that cancer originates in a single cell as a result of an irreversible and self-replicating process that involves a number of different random biological events.

EPA uses a linearized modification of the multistage model which assumes that the time rate of occurrence of each event is in strict linear proportion to the dose. This model, the linearized multistage model, is considered to be the strongest of the available models (U.S. EPA, 1988). The selection of the model for extrapolation is important because at low doses the predicted values from the models diverge significantly. For example, for a given dose there may be a factor of 10^5 or 10^8 difference in the estimation of lifetime cancer risk at a given concentration by the most conservative and least conservative models. This is uncertainty equivalent to not knowing if one has enough money to buy a cup of coffee or pay off the national debt (Cothern et al., 1986). Even so, it is highly likely that the projections of the more protective models will not underestimate risk and they may strongly overestimate it (U.S. EPA, 1988).

The risk estimates at low doses derived from animal studies must also be adjusted for the differences between the human and the animal test species. Some of these differences include body size, genetic variability, population homogeneity, health status, life span, pharmacokinetic effects such as metabolism and excretion patterns, and the exposure regimen.

The most common approach for making these adjustments is to use standardized scaling factors which are related to the type of exposure pathway and sensitivity of the target organs. Scaling factors include mg per kg body weight per day, ppm in the diet or water, mg per m^2 body surface per day, and mg per kg body weight per lifetime. Because detailed toxicological, physiological, metabolic, and pharmacokinetic data may be limited for a specific agent, EPA considers a scaling factor based on surface area (mg per m^2 body surface area per day) to be appropriate because certain pharmacological effects scale according to surface area (U.S. EPA, 1986a).

Carcinogenic risk assessment must be accompanied by a weight-of-evidence statement that is professional judgment of the quality of the assessment. The system for classifying the evidence is given under the Hazard Identification section.

13.3.3.1 Models for carcinogens

A variety of models has been proposed for this purpose. The fact that there are different models is due to different aims which are pursued with these models, and it reflects different assumptions and/or degrees of certainty about the process of carcinogenesis. Examples for the aims the models are used for can be found among the following:

- to assess the carcinogenic potency of chemical agents;
- to gain insight into the process of carcinogenesis or to check biological assumptions about this process;
- to assess the accuracy of experimental findings;
- to interpolate: estimation of the response to be expected for an intermediate dose which was not included in the observations;
- to extrapolate: estimation of the response to be expected for a very low or very high dose outside the observed range;
- to extrapolate: estimation of the response to be expected for a dose–time pattern different from the observed one; and
- to extrapolate: estimation of the response to be expected for mixtures of agents.

Before going into details it is necessary to fix some definitions. The term *dose–response* originally refers to the relation between the *number of responses* (i.e. cancers) as the dependent part and the *dose* as the explaining part. The effect of time is not considered. It is eliminated by looking at the response at a given time point only and by considering only doses which are constant over time. The inclusion of the time factor gives rise to *time–response* or *failure-time* models, which in most cases describe not only the relation between response (i.e. failure) and time, but also the effect of dose. Figure 13.1 shows, in the three-dimensional space of dose, time and response, a pure dose–response relation (time t fixed), and a pure failure-time curve (dose d fixed).

Data background

The formulation of dose–response models or failure-time models relies on the availability of data on dose, time, and response. Such data either result from experiments on animal, tissue, or cell level, or from epidemiological sources describing human cancer incidence or mortality.

Gaining data from experiments has the advantage that the dose–time pattern can be fixed by the experimenter according to his needs. However, the sample size is, in practice, always restricted due to financial and operational limitations. This has severe consequences for investigations concerning the effect of small doses, which are of particular interest for the assessment of general environmental burdens. A further problem is the transfer of results

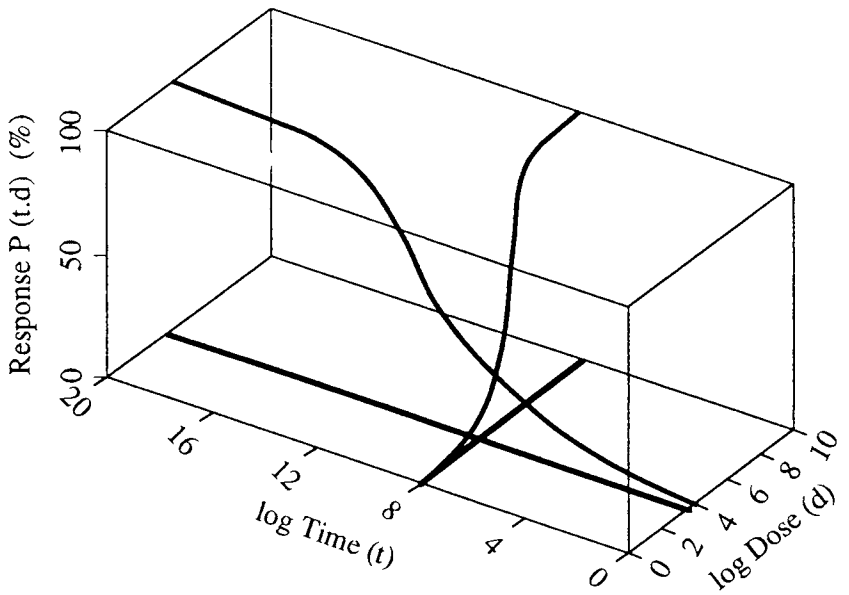

Fig. 13.1: Dose–response relationship and failure–time relationship.

from animal experiments to man.

Data from epidemiological sources have the advantage that they refer to real human conditions, as far as response and dose range are concerned, and that no transfer problem arises. Here the problems lie in the usually bad availability, especially of dose data and in the long induction times which elapse before conclusions can be drawn. Prospective testing of possibly carcinogenic agents is completely impossible. Dose–response relations thus are mainly derived from animal experiments, and consequently, the discussion here will focus on this type of data.

The notation used to describe dose–response or failure-time models is shown in Table 13.1.

Dose–response models

Dose–response models describe the relation between dose and response, where response from dose d_i is expressed by the proportion P_i:

$$P_i \frac{r_i}{n_i}$$

An observed value P_i is the result of a random experiment, which generates a response under dose d_i with probability $P(d_i)$. Several choices for the function $P(d)$, which relates dose and response, will be introduced in the next para-

TABLE 13.1

Notation to describe dose–time–response data

Experimental conditions	
n	total number of individuals
k	number of groups in the sample
n_i	number of members in group i
ij	unique index of each individual, $j = 1,2,...,n_i$; $i = 1,2,...k$
d_i	time-constant dose of agent to which each member of group number i is exposed
t_{max}	maximum time of observation
Recorded result from a pure dose–response experiment	
r_i	number of individuals with caner at time t_{max} (the only result in a pure dose–response experiment)
Recorded results from a failure-time experiment	
ij	= 0 if cancer occurred before t_{max}
ij	= 1 if not
t_{ij}	time of cancer development or censoring for individual ij

graphs. All models presuppose that cancer induction is not a deterministic, but a stochastic process. This means that a certain dose only corresponds to a certain *probability* of cancer occurrence rather than to guarantee a fixed proportion of cancer cases. Hence $P(d)$ will have the mathematical form of a distribution function.

The doses will, as before, be assumed to be constant over time, unless otherwise stated. The event of cancer development is identified with the event that the first cell in a tissue has become a cancer cell (Pike 1966).

Dose–response models: the probit model

Biological motivation. The biological background of the probit model is the assumption that there exists a response threshold for each individual: a dose above the threshold produces a response (i.e. cancer), a dose below does not. The threshold itself is assumed to have a certain small random variation from one individual to another. Individuals are assumed to behave independently from one another, i.e. cancer development in one individual does not influence the behaviour of another individual. A model of this kind is called a *threshold model* or a *tolerance model*. Both terms are equivalent, because saying that an individual has response threshold d_0 means the same as saying that the individual tolerates a dose below d_0.

Mathematical aspects. From the assumption that individual thresholds vary randomly follows that for a dose d all those individuals with thresholds smaller than or equal to d will show a response. This means that the proportion of responding individuals corresponds to the cumulative distribution function of the individual thresholds. Practical experience has shown that in many cases the log-normal distribution of individual thresholds is a good approximation. This leads to the following relation between dose d and expected proportion of responses $P(d)$:

$$P(d) = \frac{1}{\sigma\sqrt{2\pi}} \int_0^d \frac{1}{s} \exp\left(-\frac{(\log s - \mu)^2}{2\sigma^2}\right) ds$$

or, with $a = -\mu/\sigma$ and $b = 1/\sigma$,

$$P(d) = \frac{b}{\sqrt{2\pi}} \int_0^d \frac{1}{s} \exp\left(-05 \cdot (a + b \cdot \log s)^2\right) ds$$

This model is known as the *probit model* (Finney 1971). The term *probit* is shorthand for probability unit. The probit of $p = r/n$ is defined by $N_{01}^{-1}(p)$, where N_{01} is the standard normal distribution function. Probits may be used in a nonlinear regression procedures additionally, the plot of probits vs dose provides an easy graphical check for the assumption that threshold levels are log-normally distributed.

Application of the probit model is justified from the biological point of view only if the existence of thresholds and their log-normal distribution is assumed. Experimental evidence whether or not such thresholds exist is hard to provide, because thresholds could be so small that they cannot be detected by experiments with realistic sample sizes. Nevertheless, from the numerical point of view, the log-normal distribution is a satisfactory description of many empirically found dose–response relations. This, of course, does not allow me to conclude that any thresholds exist.

Dose–response models: the logit model

Problems arise with the probit model, if control groups with $d = 0$ are present, or if the model is to be used for low-dose extrapolations. In such cases, the use of the *logit* model should be considered, because the logit dose–response relation is numerically very similar to the probit, numerical difficulties are avoided, and calculations are easier. The term *logit* is derived from logistic unit (Ashton, 1972). In analogy to probits, logits are defined as transformations of

probabilities p: $\text{logit}(p) = \log(p/(1-p))$. The logit model for log dose predicts the following proportion of responses for dose d:

$$P(d) = \frac{1}{1 + \exp[-(a + b \log d)]}$$

$$= \frac{\exp(a + b \log d)}{1 + \exp(a + b \log d)}$$

$$= \frac{e^a \cdot d^b \, 1}{1 + e^a \cdot d^b}$$

The last version of the logit model is defined for all $d \geq 0$, so that no problems arise if control groups with $d = 0$ exist.

Differences between probit and logit curves lie mainly in the tails of the distributions, as will be shown in Figure 13.2.

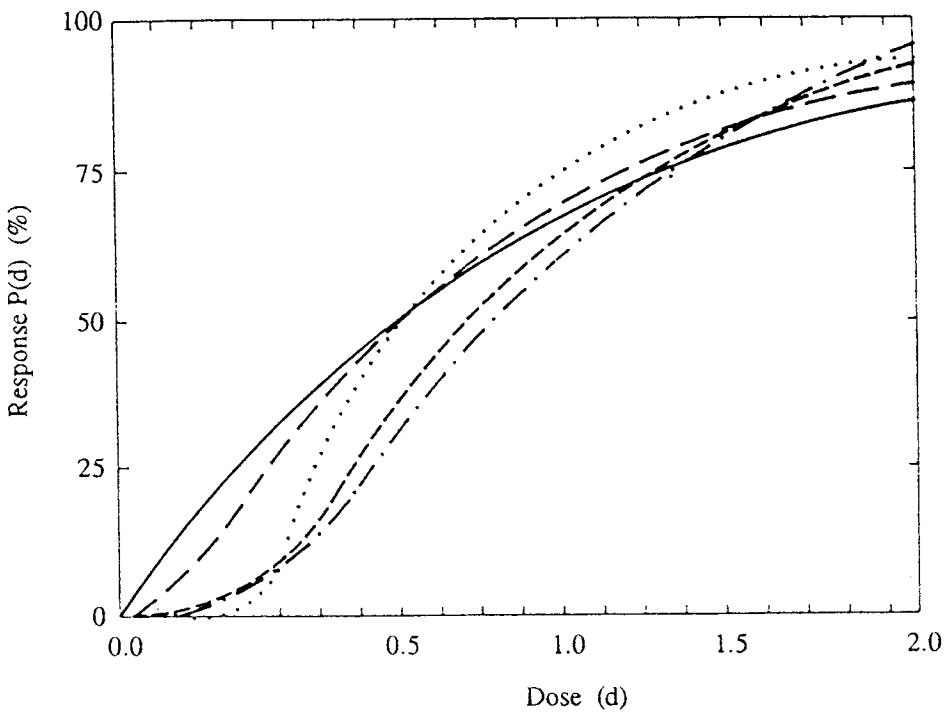

Fig. 13.2: Some dose-response functions: (a) probit (long dashes); (b) logit (closely spaced dots); (c) one hit (solid line); (d) multi-hit (short dashes); (e) Weibull distribution (dots and dashes). Displayed distributions have means equal to 1 and, where possible, variances equal to 1.

Dose–response models: one hit model

Biological motivation. Hit models are based on the assumption that cancer is caused by the entry of an agent into a "target area". A one-hit model describes the risk of cancer origination, if one single entry in one target area is sufficient to cause cancer. The term "hit" has been coined in the framework of radiation-induced cancer, where "hit" has an obvious meaning. In the framework of chemically induced cancer, a "hit" can be identified with the entry of a molecule of the agent into the target area.

Mathematical aspects. If the target area can be assumed to be small compared with the total cell area, and if there is a constant number of hit attempts per time unit (i.e. dose is constant over time), then the relation between dose d and probability of response is

$$P(d) = 1 - \exp(-a \cdot d)$$

This distribution is known as the exponential distribution.

Dose–response models: multihit model

Biological motivation. The multihit model is a direct generalization of the one-hit model. Here it is assumed that several hits are required to induce cancer. Only the number of hits, not their distribution on possibly several target areas, is crucial.

Mathematical aspects. The formal assumptions (small target area, constant number of hit attempts per time unit, independent hit probabilities) are the same as with the one-hit model. The difference lies in the assumption that $k \geq 1$ hits are necessary. This leads to the following relation between response P and dose d:

$$P(d) = 1 - \sum_{j=0}^{k-i} \exp(-a \cdot d) \frac{(a \cdot d)^j}{j!} \tag{13.1}$$

This contains the exponential distribution as a special case ($k = 1$).

If the coefficient a is very small, then the sum in Eq. (13.1) is dominated by the highest-order term and $P(d)$ may be approximated by

$$P(d) = 1 - \exp(-a \cdot d^k) \tag{13.2}$$

the Weibull distribution. In principle, the parameter k in Eq. (13.2) should attain only integer values, as it corresponds to the number of necessary hits. In practice, all values are accepted, because this allows a closer fit to empirical data.

Dose–response models: multistage model

Biological motivation. The multistage approach assumes that development of cancer requires several transformations per cell, i.e. a single cell must undergo several successive changes. The effect of the agent is at least to start this transformation process, which subsequently might proceed independently from the presence of the agent. Alternatively the model allows one to account for agent effects during later stages of the transformation process. In the general version, the multistage approach models three possible events for each cell: transition to another stage, death, or division. Each event may or may not be affected by the agent, which means that potentially not only the carcinogenic effect, but also toxic and proliferation changing effects may be studied. The multistage model allows a natural incorporation of time-varying doses.

Mathematical aspects. In the classical version of the multistage model proposed by Armitage and Doll (1954, 1961) it is assumed that the tissue under consideration consists of a large number of initially normal cells and that the number of hit attempts per time unit by the agent is constant over time. Toxic and proliferation effects are assumed to stay in an equilibrium, so that they may be omitted from explicit modelling. The event of cancer development is set equal to the event that the first cell undergoes the final transformation. These simplifying assumptions lead to the following relation between probability P of response and dose d:

$$P(d) = 1 - \exp(poly(d)) \tag{13.3}$$

where $poly(d)$ denotes a polynomial of d. Degree and coefficients of the polynomial depend on the number of stages and the possible transitions between stages. In a simple version with two transformation stages, which must be passed consecutively and where no other transitions are possible, the dose–response relation is

$$P(d) = 1 - \exp[(a_1 + b_1\, d) \cdot (a_2 + b_2\, d)] \tag{13.4}$$

The a-coefficients in Eq. (13.4) describe spontaneous transition rates, while the bs model the dose-dependent rates. These coefficients cannot be identified from simple dose–response data, but if Eq. (13.4) is expressed in a simpler form by multiplying out the exponent, giving

$$P(d) = 1 - \exp(a + b\, d + c\, d^2) \tag{13.5}$$

the unknown coefficients a, b and c can be estimated from dose response data. Note that if $a = b = 0$ or $a = c = 0$ in Eq. (13.5), then the multistage

dose–response relation has the Weibull form (Eq. 13.2).

Several versions of the multistage model have been proposed. They differ mainly in details concerning the biological interpretation of the stages and in their way of dealing with cell cycle behaviour.

Low-dose extrapolation

One particular use of dose–response models in the framework of environmental risk assessment is the estimation of the response to be expected from doses comparable to those which can be found in the actual environment. This usually is an extreme extrapolation problem (at least if occupational exposure is left out of consideration). The smallest non-zero effect which can appear in an animal experiment of group size n corresponds to an empirical probability of $1/n$. For realistic sample sizes this means that risks of order below 10^{-3} are hardly observable, while from the regulatory point of view risks of much lower order than 10^{-3} are relevant.

Most of the dose–response models introduced so far can, for small d, satisfactorily be approximated by a log–linear relationship:

$$\log P(d) = a + b \log d \quad \text{for } d \text{ small} \tag{13.6}$$

An exception is the probit model, which has as low-dose extrapolation

$$\log P(d) = -0.5(a + b \log d)^2 - \log[(a + b \log d)c] \tag{13.7}$$

where c is some constant.

The result of a low-dose extrapolation depends heavily on the chosen dose–response model. Though the low-dose approximations have the same form for most of the models, the resulting extrapolations are not, due to different coefficients (e.g. a and b), which are specific for each model. Experience has shown that extrapolations from different models may differ considerably, even for models which fit equally well in the range of observed data (Drescher et al., 1983).

Time-dependent doses

The previous discussion assumed that dose is constant over time, unless the opposite was stated explicitly. While a constant dose is helpful in the formulation of models, for carrying out an experiment, and for the comparison of agents, it is not very near to what happens in the real environment, where time-varying doses are the rule. With time-varying dose, the term "dose" must be redefined. One now has to deal with a dose function $d(t)$ of time, where $d(t)$ is the dose level at the time point t. The total dose up to time t is:

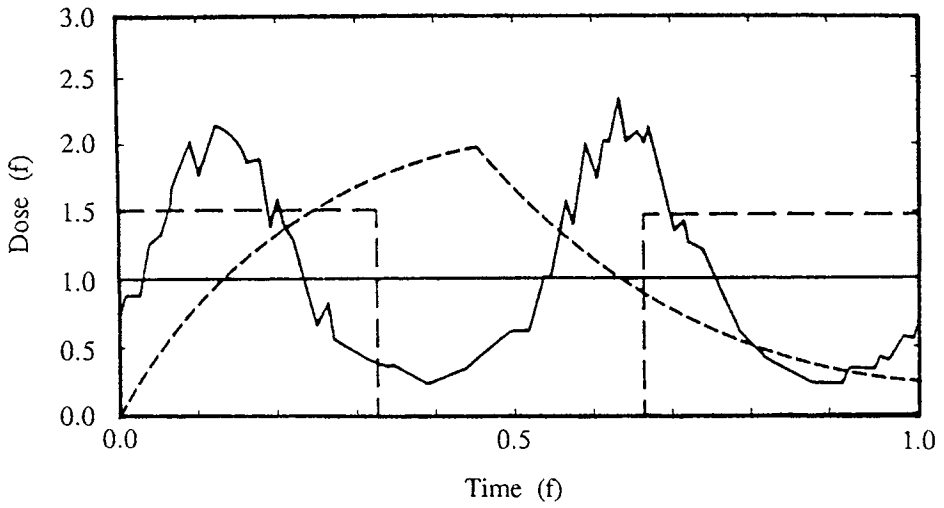

Fig. 13.3: Some dose–time functions: (a) constant dose (solid line); (b) fractionated dose (long dashes); (c) fractionated dose transformed by pharmacokinetics (short dashes); (d) dose with circadian rhythm superimposed by random fluctuations (dotted). The dose until $t = 1$ is the same for (a)–(d).

$$D(t) = \int_0^t d(s)ds \tag{13.8}$$

If $d(t)$ is constant as before ($d(t) = d$), the integral in Eq. (13.8) reduces to $D(t) = d \cdot t$. Some typical dose–time functions are shown in Figure 13.3.

A time-varying dose is mostly realized experimentally by breaking up the total dose in several portions (fractionating) as shown in Figure 3b. The response to be expected from a fractionated dose cannot be concluded from the models introduced so far without further assumptions. The first problem is with $d(t)$; it is not clear what to insert for d into the model equations. Some suggestions are

$d = \max_{t \le t_{max}} d(t)$ maximal dose,

$d = D(t_{max})$ total dose,

$d = D(t_{max})/t_{max}$ mean dose.

Neither of these suggestions has been found to be universally satisfactory. The most promising approach is to use a version of the multistage model, which allows to directly incorporate a time-varying dose by letting the rates depend on dose $d(t)$. This leads to time-varying rates $a_i(d(t))$, $b_i(d(t))$ etc. The model can

further be extended to account for repair events, which could play a central role in explaining effects of different fractionation schemes. The disadvantage of the multistage model with time-dependent rates is that in general a closed form of $P(d)$ can no more be given, and that the numerical effort to compute $P(d)$ as numerical solution of a system of differential equations might become formidable (though tractable).

One way to deal with dose–time relations of possibly different shapes is to include the dose–time relation into the mathematical model. This leads to dose–time–response models, which have a graphical representation of the kind displayed in Figure 13.1. The use of dose–time–response models requires that appropriate data is collected besides the number of responses now the time t_{ij} of each event must be recorded, as well as the type $-ij$ of the event (response or censoring).

Parametric dose–time–response models

Most of the dose–response models have an immediate generalization to dose–time–response models. As the biological and mathematical background remains essentially unchanged, it is sufficient to present the time dependent counterparts of the models. One difference is that hit models with different numbers of target areas may now be discriminated. The models are given as dependent on $\log d$, analogous to Eq. (13.6).

one-hit model

$$P(d) = 1 - \exp[-\exp(a + \log d + \log t)]$$

multihit — one target

$$P(d) = 1 - \exp[-\exp(a + b \log d + \log t)]$$

one hit — multitarget

$$P(d) = 1 - \exp[-\exp(a + \log d + k \log t)]$$

multihit — multitarget model

$$P(d) = 1 \exp[-\exp(a + b \log d + k \log t)]$$

multistage model

$$P(d) = 1 - \exp[-\exp(poly(\log d, \log t)]$$

where $poly(x,y)$ denotes a polynomial with arguments x and y.

The multistage models and the Moolgavkar–Venzon model mentioned before include time already by their basic definitions.

Computational aspects

All practical applications of dose–response or dose–time–response models require that some unknown parameters (coefficients in the model equations) be estimated from experimental or epidemiological data. The estimation itself follows the maximum likelihood principle or some least squares approach. As the estimation for most of the models means to perform lengthy numerical calculations, this task is normally left to appropriate software packages.

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Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 14

Risk Assessment: Exposure Assessment[1]

The exposure assessment estimates the exposures to which the population of interest is likely to be subjected. It must be combined with the dose–effect assessment in order to obtain a quantitative estimate of the risk.

At the present time there is no single approach to exposure assessment that applies to every case. Rather, the method that is used must match the individual case based on the available data. As in all phases of risk assessment process, the assumptions, approximations, and assertions made for the exposure assessment should be stated.

The major elements of the exposure assessment include identification and characterization of the following areas:

Sources. The production, distribution, uses, disposal, and environmental releases of the chemical or biological agent should be assessed.

Exposure pathways and environmental fate. The transport, transformation, principal pathways of exposure, and predicted environmental distribution should be evaluated.

Measured or estimated concentrations. The concentrations available for exposure should be evaluated using measurement, mathematical models, or a combination of the two.

Exposed populations. Populations and subpopulations at potentially high exposure should be identified, and subpopulations of high sensitivity may be studied separately.

Integrated exposure analysis. An exposure profile is developed from the estimation of environmental concentrations and the description of the exposed population. This profile should include the size of the exposed population; duration, frequency, and intensity of exposure; and routes of exposure.

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1 A part of the text of this chapter has been derived from D. Mage "A Comparison of the Direct and Indirect Methods of Human Exposure" in: Report on a Joint Workshop "Methods of Risk Assessment for the Indoor Environment", pp. 21–28, Kloster Banz, Germany, 15–17 October 1991. Ed. B. Seifert. NATO/CCMS Pilot Study on Indoor Air Quality and ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man"); B.R. Weir "Specification of Indoor Air Model Characteristics" in: Proceedings of the 5th International Conference on Indoor Air Quality and Climate, Vol. 4, pp. 231–235, Toronto, Canada, 29 July–13 August 1990.

EPA currently assumes that receiving a high dose of a carcinogen over a short time period is equivalent to receiving a low dose over a lifetime. This assumption is used to calculate a lifetime average daily exposure (LADE) that is incorporated into the risk characterization.

14.1 DIRECT AND INDIRECT METHODS FOR ASSESSMENT OF HUMAN EXPOSURE

Biological samples can be either a measure of dose of pollutant or a measure of the effect of the pollutant. Although we here refer to surveys that can use biological dosimetry to assess human exposure to the pollutant, the concepts can also be applied to a survey that uses biological dosimetry to measure the health effects of the pollutant.

Human pollutant exposure assessments have been performed initially by comparison to ambient air quality, water supply quality, and market basket food data. With the introduction of personal exposure monitoring and sampling of individual dietary uptakes it has been found that personal exposures are determined by individual activities and these exposures, when computed from ambient type data, are most often underpredicted and only weakly correlated. Consequently the World Health Organization (WHO) and the United Nations Environment Programme (UNEP) have initiated the Global Environment Monitoring System (GEMS) Human Exposure Assessment Locations (HEAL) Programme to encourage the use of personal monitoring and biological dosimetry for evaluation of how well people are protected by the various air pollution, water pollution and food contamination control programmes which are evaluated using ambient data.

For a given population there will be a distribution of exposures and resulting doses of pollutant. By summing the exposure/intake over all pathways (air, water, food, dermal) for each individual we can model this distribution. This distribution provides information useful for defining the severity of the pollutant problem and allows judgements to be made on the necessity for future action. Furthermore, when routes and magnitudes of exposure are known, the effectiveness of various control strategies can be evaluated.

14.1.1 Methods of exposure assessment

Human exposure surveys can be performed in two basic ways. In the *direct method*, people carry personal air monitors, collect duplicate diets of food and water, and in some cases wear skin patches for dermal exposure. By completion of a diary/questionnaire we can follow their time, location and physical activity trajectory through the study. This allows us to estimate their ventilation rate

(litres/minute) and pollutant intake by inhalation which can then be summed with the dietary intakes and dermal uptake if any to create their total intake for the study period. In the *indirect method*, people only complete the diary/questionnaire to indicate time, location, activity and what was eaten or drunk. The exposure is then computed from available ambient air and water data, market basket surveys of food contamination and microenvironmental[1] (5E) data to adjust the ambient data for the type of indoor location the subject may be in.

The choice of type of survey depends on the accuracy and precision required for the results and of course the budget available for conducting the study. In practice one designs a study to meet an initial set of objectives and if the budget required is too great the accuracy and precision requirements may be relaxed until an acceptable study is designed. Suppose we wish to determine the percentage of the nonsmoking population with carboxyhaemoglobin (COHb) greater than 3%. There are several ways this can be done using a randomly sampled population either directly or indirectly, such as:

1. collect blood samples and measure COHb;
2. collect end tidal breath measurements of CO and estimate COHb;
3. measure personal CO exposures and activities and compute COHb; and
4. measure activity patterns and estimate CO exposures, then compute COHb.

In the measurement of an individual personal exposure, accuracy refers to the correct values of the pollutant concentration in a biological media or in the correctly measured amount of food, air and water which the subject ingests. However in a population study, accuracy refers to these measured quantities plus the percentage of the population with that exposure. For instance the true population value may be 5% of the people having exposures leading to 3% COHb. The result of the study may lead to results such as 3% of the population are at 3% COHb or 5% of the population are at 2.5% COHb. These differences from the true value may be caused by errors in both the concentration and the population variables. The following sections discuss major components of exposure accuracy for both the *indirect* and *direct methods*.

14.1.2 Errors in assessment of human exposure

A. Concentration measurement error

In the *direct method* the exposure concentration in air, water and food is measured by established analytical techniques. Two components of error in the analysis are non-random error or bias caused by incorrect calibration

1 A microenvironment (5E) is a volume of space for a given time interval during which the concentration variance within the 5E is less than the variance between it and its neighbouring 5Es. A home without any operating sources of CO can be a single 5E. If a gas stove is operating in the kitchen, the kitchen and the rest of the home can now be treated as two distinct 5Es.

standards and random error caused by fluctuations in the analytical results about the central value. These errors can be minimized by use of independent quality control standards and good laboratory practice.

In the *indirect method* the pollutant concentration in the air is estimated by use of the ambient concentration plus a correction to allow for the microenvironment effect in the location that the subject is in. The diary questionnaire defines where the subject is, indoors or outdoors, and in what type of surroundings. If the subject is in a residence with a gas stove in use and smokers present, the exposure may be estimated as the ambient exposure at the nearest monitoring station plus a correction for the presence of gas stove and smokers. This correction comes from studies of similar residences with similar sources and sinks where the difference between the indoor and outdoor concentrations are noted. This difference can then be added to the current ambient value to estimate the concentration the subject is exposed to. Note there are two errors involved here. The concentration outside the home is not identical to the ambient value measured several kilometres away and the correction for that particular home is not identical to the average correction for the similar locations developed in another study. These other reference data had experimental errors as described above for the direct method and the overall error will be a composite of all of them.

B. Sampling error

Sampling error occurs because a random sample of a population does not have the identical distribution of the whole population. If we choose different random samples of identical size from the population the values of any parameter such as the percent of the sample with a characteristic or the mean value of a parameter will be variables that fluctuate about the population means. These errors cannot be eliminated but only reduced by increasing the size of the sample.

In the *direct* and *indirect method*, sampling error refers to the size of the sample. Because the cost of the indirect method per participant is much less than for the direct method, many more people can be enrolled for use of a diary and questionnaire which would minimize the sampling error for the study.

C. Non-response error

In any population survey people often refuse to participate. This may be for one or more of several reasons. Some people don't like giving blood. Many people are too busy, uninterested in the subject, illiterate, too ill or weak to carry a monitor, unable to follow instructions, away from home during the

study, unable to get permission to take the monitor to work, and many other reasons. Because the exposure characteristics of these people may be different than those who agree to participate this will produce an error in the estimate of the population parameters. If the true mean of all people is Z, the mean of x responders is X, and the mean of y non-responders is Y, the bias in the estimate of Z is equal to $y(X - Y)/(x + y)$. Since $X - Y$ is unknown one can only reduce the bias by reducing the fraction of non-responders in the study, $y/(x + y)$.

Because the burden on the subject in the indirect method study who only carries a diary and completes a questionnaire is much less than the burden on the subject in the direct method study who carries a diary, completes a questionnaire, but also carries a portable monitor and collects a duplicate diet, the indirect method study will have a lower non-respondent error because fewer people will refuse to participate.

It is always important to convert non-responders to responders which may require giving special incentives (such as a cash sum). Finally, if at all possible, the non-responders should be asked a minimal number of questions that can help define whether they as a class have similar or different characteristics to the responders and whether they have some special exposure characteristic that is not in the response group (e.g. traffic policeman, welder, etc.)

D. Design effect error

In practice we are unable to randomly sample a complete population where the sample is drawn from a list of the total eligible population. Even if such a list existed it would be very impractical and costly to have to go to 100 different locations in an urban area to recruit 100 subjects compared to going to 10 randomly chosen locations and cluster recruiting 10 subjects in each area. The process of drawing the sample is very complicated. An interviewer must go to the home to find out if the occupant is eligible to participate (e.g. nonsmoker), capable of participating (not ill) and willing to participate. If no one is home in the evening, the interviewer must return until contact is made. If the next person on the list to interview is far away, it takes too much time and effort for the overall process to succeed. If no one is home another person next door cannot be substituted as this could lead to a sample with under-representation of those who work in the evenings.

The underlying principle in drawing the sample is that each choice of person is independent. However, when cluster sampling is used the people in each neighbourhood may not have independent exposures. Their air quality may be similar, their water supply source may be the same, they may shop in the same markets and have the same food supply, their homes may be of similar quality and age, and their socio-economic status may be similar which

relates to home product usage, diet, etc. Thus 10 clusters of 10 each provides less information on overall community exposure than 100 randomly distributed samples over the entire community. This is called the design effect which arises from the practical requirements of a study design. The design effect does not cause a bias but does increase the sampling error as the number of independent samples is reduced.

E. Information error

People may give incorrect information on questionnaires and diaries. This may be purposeful, such as stating that they are non-smokers when in fact they smoke (perhaps marijuana). A serious error may occur if a subject forgets to turn on a monitor or leaves it at home and doesn't take it to work but doesn't report it in order to avoid an uncomfortable situation. They may forget to report some things that they did, or recall some things they did outside the study period. "Have you eaten any shrimp (an important dietary source of cadmium) in the past month?" might get a positive response when it was 6 weeks ago that they were eaten. People may not keep a running diary as instructed, but complete it at night and not recall exactly what they did and when. Dietary recall may be incomplete or impossible to complete accurately, as one does not know the ingredients of the food, only the generic name (pizza).

These information errors can lead to erroneous estimates of exposure in the indirect method studies and to wrong classification of exposures. For instance if someone smokes but is reported as a nonsmoker a high COHb would be found in the nonsmoking categories with the implication that one of the locations or vehicles may be considered the source or the participant may be attributed a blood disorder such as sickle cell anaemia.

These errors can not be eliminated, but it may be possible to minimize them by careful checks and random visits to participants to verify they are wearing monitors and that their diaries are filled in.

F. The Hawthorne Effect

The act of being in a study in itself can influence a persons actions and performance. In the classic Hawthorne study, the productivity of an assembly line group of employees was studied to see if an improved lighting system was effective for improving production. The results of the study showed increased productivity but when the study was over the productivity returned to the prior level in spite of the continued use of the improved lighting. It was concluded that the subjects improved their functions because they were being studied, not because of the lighting. In an exposure study the danger is that

the subjects alter their behaviour because they are wearing a personal monitor or because they have to collect a duplicate diet. The change in behaviour may be either conscious or unconscious. In the worst case the subject might blow cigarette smoke into the inlet of the air sampler or stand behind an idling vehicle to purposely influence the study (they think pollution is bad and they want the study to show it). Another conscious action may be to try to help us find pollution by going to places that they think are interesting (a smoky restaurant or a parking garage) or to deliberately not go somewhere because they are wearing the monitor as it might attract unwanted attention.

Collection of a duplicate diet may be embarrassing or difficult in a restaurant or as a guest so the subject may alter his or her eating habits because of it. Some of these choices may be unconscious or conscious but they all provide the same result, e.g. the exposure is an artificial exposure in that this subject would have been exposed differently if he or she were not involved in the study. This cannot be prevented although subjects are cautioned to act normally and not to change their behaviour. This error is greatest in the direct approach where samples are collected and monitors are worn, and probably negligible when the subject of an indirect study only carries a diary.

G. Selection bias

In almost every situation where a sample of a population is to be chosen the selection of the participating subjects must be randomized. Otherwise the choice of the subject may be influenced either consciously or unconsciously by the person selecting the sample. For example, the person selecting the subjects for the study may subconsciously want to choose those people who may have a high exposure and thus the sample would be over-represented by that group. To prevent any such bias error occurring from the sample selection procedure must be firmly fixed before the people are contacted and stringently followed. If an interviewer is to enrol one nonsmoking adult in each home, all such adults must be numbered and a random number chosen to determine which adult is asked to participate.

14.1.3 Measurement accuracy

In exposure assessment studies quality control can establish confidence intervals on physical measurements of pollutant concentration in food, air, water, on skin, and in biological media and the amount of food, air and water ingested. However, the confidence intervals on population estimates are much more difficult to obtain for the reasons discussed above. If the exposure distribution in a city of 1 million people is measured by drawing a random

TABLE 14.1

Relative comparison of direct and indirect methods

Exposure category	Direct method	Indirect method
Concentration error	low	high
Sampling error	high	low
Non-responder error	high	low
Design effect error	high	low
Information error	equal	equal
Selection bias error	equal	equal
Hawthorne effect error	high	low
Overall study accuracy	high	low
Overall study cost	high	low

sample of 100 people, each person represents ten thousand (10,000) people. If one person in the sample has an exposure above some established action level the implications are that approximately 10,000 people in the city have a similar exposure which might be large enough to justify control measures. Thus the activities of a single individual in such a study can have importance for the interpretation of the results. Even though this individual's exposures were measured correctly, the implication for the population would be incorrect if the subject had altered behaviour from the normal pattern in such a way as to increase the exposure.

As shown in Table 14.1, the *direct method* and the *indirect method* of exposure assessment each have different advantages and disadvantages.

The choice between these two study types is essentially between low cost–low accuracy and high cost–high accuracy. It is proposed that careful combinations of the elements of each study can lead to an acceptable study design of moderate accuracy and moderate cost that meets the study requirements. The design of such a hybrid study is discussed in the following section.

14.1.4 The hybrid exposure assessment

It is proposed that a study design be used in which some subjects carry a personal monitor, collect a duplicate diet, give biological samples and complete an activity diary and questionnaire. Other subjects would only complete the activity diary and the questionnaire. This type of design has been used in some health studies where the measured exposures of a small group of subjects in a cluster is taken to represent the exposures of the local population who are

reporting activities and symptomatology by a questionnaire. It is proposed that this information collected by questionnaire be combined with exposure measurements on the subsample to make a corrected estimate for their exposure and that these results could be combined into a final estimate.

The study would operate on a design in which the total area to be sampled is divided into clusters and a number of clusters are chosen at random for sampling. In a given cluster a small number of people would be chosen randomly for the exposure measurements and a large number of people would be randomly chosen for the exposure estimation by questionnaire. Since the people carrying monitors and collecting duplicate diets are completing the same questionnaire and diary as the second group their exposures can be computed by the indirect method using the same ambient values and market basket data. Their measured media exposures can then be compared to their estimated media exposures as an independent check of how accurately the indirect method is able to compute the media exposures of the second group. The exposures of the second group in the various media determined by the indirect method could be adjusted by the bias between the first group's measured exposures and estimated exposures.

There are a number of advantages to this combined or hybrid type study design. The major advantage is that the number of people whose exposures are estimated is now increased and the sampling error is decreased. The non-response error may be more easily corrected since people who refuse to carry monitors may be willing to complete questionnaires and diaries so that their exposures as a class can be compared with the people carrying the monitors. The population parameters, such as the mean exposure, could be computed from both groups giving higher weights to those whose exposures are measured, and lower weights to those whose exposures are estimated.

A further advantage to this type of split design is that the cluster measures of air route exposures may be used to compute the microenvironmental (5E) exposure increments in the same cluster for those who only complete the questionnaires and diaries. The 5E concept is that a concentration in a 5E is equal to the concentration that would exist there if there were no sources or sinks (e.g. the ambient value at that time and place) plus an increment that accounts for sources and sinks in the 5E. This increment is also a function of the air exchange rate between the 5E and the ambient air (or in some cases between the 5E and other bordering 5Es). Because of the design effect these 5E increments may be more similar in the cluster because of the similarities in housing stack (air-conditioning, age, etc.) and local meteorological parameters such as wind speed and direction which can influence air exchange rates. The water quality measured in the cluster homes would give appropriate values to use to estimate the indirect method subjects' water exposure.

In regard to the dietary exposure, the analysis of the duplicate diet collected in the direct method measures the average concentration of pollutant in the mixture of all foods eaten so that each item is not measured individually. Thus, a direct item-by-item correction to use for the estimated dietary exposures may not be possible. However, by computing the dietary intake of the direct subjects and comparing it to the actual measurement, a rough estimate of the accuracy of the prediction could be obtained. By making these corrections on a cluster basis there may be an advantage in that the type of diet may be similar in the cluster and the subjects may purchase foods from the same supplier. One difficulty is that there may be food items with high pollutant levels that are reported by a person in one group but not in the other group, e.g. an ethnic food or an out-of-season item such as cranberry sauce. In this case the market basket value uncorrected may be a better estimate.

14.1.5 Comments on practical use

Population studies to measure or estimate the distribution of pollutant exposures, doses and effects can combine the direct measurements of these quantities with estimates of these quantities based on questionnaire information. The loss in accuracy in using estimates is offset by the financial gain in using a less expensive method and reduction of sampling error by increasing the sample size. By adjusting the total number of subjects and the percentage of direct method subjects the cost and estimated accuracy of the study can be varied so that an acceptable study design can be achieved. For developing countries where resources are scarce, a study with a large number of subjects and a small (10%) percentage using the direct method may be useful for problem definition where an approximate answer is needed. For a developed country with environmental quality standards and complex implementation plans and emission controls to meet them, a moderate number of subjects and a large (50%) percentage using the direct method may give the required accuracy and precision. In conclusion, it is recommended that future exposure assessment studies consider combining these two techniques of direct and indirect exposure assessment into their design as a practical and cost effective way to achieve their study goals.

14.2 EXPOSURE MODELLING

The field on indoor air modelling has suffered from a lack of categorization and classification. Models that were developed to evaluate single rooms, multiple rooms, indoor sources, entire populations of cities and indoor/outdoor differences have all been called "indoor air models". This has led to confusion as to the applicability of each model for specific situations. Many of the

currently used models have overlapping strengths and weaknesses; no one model addresses the entire range of indoor problems and issues.

In the following text nine models are reviewed, in brief, and systematically categorized as to the particular features inherent in each.

14.2.1 Classification of models

Outdoor air quality models are normally categorized as dispersion models or source-receptor models. Sub-division include plume, grid and data base empirical models. This categorization is adapted here for indoor air models. Since personal and "normal" ambient air quality monitoring data are becoming more prevalent (Wallace, 1989; Kelly, 1989) empirical models may address these features. Modelling of source strength emission characteristics may also be included.

Personal exposure is difficult to estimate form either indoor or outdoor models. The difficulty is determining the probability of anyone being in a particular location and time. Several indoor and outdoor models have incorporated population mobility patterns into their structure; this capability and its effectiveness are also included as parameters to be evaluated. The capabilities described below are adapted from those developed by the California Air Sources Board (California Air Resources Board, 1989).

1. Level 1 capability: Given personal exposure data, estimate the population exposure distribution for the population as a whole and for particular subgroups.
2. Level 2 capability: Given microenvironment concentrations and activity patterns, estimate population exposure. This includes concentrations calculated as part of the model.
3. Level 3 capability:
   (a) Given air exchange rates, removal rates, source strength, and outdoor concentrations, estimate microenvironment concentrations.
   (b) Optionally calculate or sample air exchange rates from a data set.
   (c) Estimate source strength based on emission rate, load factor and source duration.
   (d) Estimate source strength for four broad categories of indoor sources: combustion sources, consumer products, material sources and water.
4. Ability to analyze both gaseous and particulate pollutants.
5. Capability of estimating for several averaging times.
6. Capability of integrating exposure in several different microenvironments.
7. Assessment of the effects of potential mitigation measures.
8. Estimation of exposure by non-inhalation pathways.
9. Operate on a PC with a user-friendly interface.

TABLE 14.2

Summary of capabilities of nine exposure and indoor air quality models

Model	1	2	3a	3b	3c	3d	4	5	6	7	8	9
NEM, NEM/SAI	X	X	X		X	X	X	X	X	X	X	
PAQM	X	X	X		X	X	X	X	X	X	X	
REHEK	X	X					X		X			
SHAPE	X	X					X	X	X			
MACROMODEL			X	X	X		X			X		
IAQPC			X		X	X	X			X		X
CONTAM		X		X	X	X	X		X			
IAQ		X	X		X			X		X		
CIT			X		X	X	X	X		X		

A summary of the capabilities of the nine indoor models is presented in Table 14.2.

14.2.2 Description of individual models

National Ambient Air Quality Standards Exposure Model (NEM):

Capabilities: 1,2,3a,3c,3d,4,5,6,7,8.

NEM is a urban-scale exposure model developed for EPA (Paul, 1981), to estimate population exposure to criteria pollutants (Johnson, 1981a,b; Paul, 1986). Diurnal patterns of movement between six microenvironments, such as outdoors, inside a home, inside an office, etc., are defined for each age and occupation group. Model results include hourly exposures for each census district for each age-occupation cohort.

Systems Applications Inc. has enhanced NEM for ozone exposure, NEM/SAI (Austin, 1988) with addition of an explicit indoor air quality model (also a stand-alone model; see PAQM). A version of this model has been developed for the South Coast Air Quality Management District (Los Angeles, California) as SCREAM-II (South Coast Risk and Exposure Assessment Model).

Personal Air Quality Model (PAQM):

Capabilities: 1,2,3a,3c,3d,4,5,6,7,8.

PAQM was developed by Systems Applications Inc. to estimate the indoor exposures due to population groups' daily activities (Hayes, 1989a). PAQM uses 56 different population groups with distinct activity patterns for weekdays, Saturdays and Sundays. Eleven different microenvironments are used with a

single-compartment indoor module (Hayes, 1989b). Building parameters and source terms are user-defined and optionally time-varying.

Regional Human Exposure Model (REHEX):

Capabilities: 1,2,4,6.

REHEX developed by Winer et al. (1989) is similar to NEM except that it utilizes frequency distributions of air quality data for each hour of the day, rather than a time series. This enhances computational efficiency, but prohibits estimations of multi-period averages. The model addresses three microenvironments: outdoors, in-transit and indoors. Indoors and in-transit concentrations are estimated as in NEM, i.e., as a fraction of the outdoor concentration plus a contribution from indoor sources. Population mobility among exposure districts is included.

The nine age–occupation groups are divided into numerous subgroups, each with a different activity pattern. Thus variability in activity patterns is more detailed. The likelihood of capturing the upper tail of the exposure distribution, associated with an atypical activity is thus increased.

Simulation of Human Activity and Pollutant Exposure Model (SHAPE):

Capabilities: 1,2,4,5,6

SHAPE developed by the U.S. EPA (Ott et al., 1988), uses activity pattern data in conjunction with microenvironment concentrations. SHAPE's activity patterns may be either deterministic or generated through a Monte Carlo sampling process. The authors state that application of the sampling option to other than CO exposures requires "more extensive analysis of the data base than has been carried out thus far". The microenvironment concentrations for each period are sampled from an empirical or statistical frequency distribution. The population is tracked sequentially so that multi-period average exposures may be estimated. It is noted by the authors that the assumed time-independence of microenvironment concentrations may underestimate the upper tail to the exposures.

Macromodel for Assessing Residential Concentrations of Combustion Generated Pollutants (MACROMODEL):

Capabilities: 3a,3b,3c,4,7

The MACROMODEL (Traynor, 1989)) estimates indoor air concentrations, but not exposures. It contains detailed modelling of combustion sources. It assumes well mixed and steady-state air masses, which may underestimate the upper tail of the exposure distribution. The authors note that open windows or doors are not addressed so summer air exchange may be underestimated, and concentrations overestimated. It uses a fixed averaging time of one week.

Indoor Air Quality Model Personal Computer Version (IAQPC):

Capabilities: 3a,3c,3d,4,5,7,9

The IAQPC model, developed at the U.S. EPA by Sparks and others (Ensor, 1989; Sparks, 1988) also addresses indoor air quality and not exposure. It estimates concentrations in multi-compartments on the basis of air exchange, removal rate, source strength, and outdoor concentrations. Air exchange parameters are user-defined rather than sampled. The actual airflow distribution is determined according to pressure balance equations, but not on the basis of external factors. The model allows for 2 models of air flow (i.e., HVAC system on or off) with user-defined switching times. Source strength is modeled for a number of different source types, as is removal due to reactivity or deposition with the possibility of re-emission. Like PAQM, the timing of combustion source emissions is user-defined rather than estimated from building parameters and external conditions. However, tobacco smoke emissions are randomly distributed within each particular hour to simulate smoking behaviour, this prohibits reproducibility of model results.

Concentration-time profiles are determined, as well as user-defined. Mitigation measures affecting source duration, load, air exchange, or HVAC functioning may be assessed. The model utilizes user-friendly menus.

CONTAM:

Capabilities: 3a,3c,3d,4,5,7

CONTAM (Axley, 1988) also estimates the microenvironment concentration based upon air exchange rate, removal rate, source strength, and outdoor concentration for a multi-compartment structure. Air exchange rates are determined from user-defined flow rates, rather than from sampled parameters or calculations. Source strength is user-defined and time variable. Pollutants may be removed or generated by chemical transformations or deposition at user-defined rates. Mitigation measures affecting building configuration, airflow rates, HVAC system parameters, source emission rates, or source emissions duration may be assessed. The model may find a steady state or the time trajectory of concentrations. Multi-period averaging is possible. CONTAM also utilizes a well-mixed assumption. It is not an exposure model.

Indoor Air Quality Model (IAQ):

Capabilities: 2,3a,3c,5,6,7

McKone's IAQ model (McKone, 1987; 1989) estimates the concentration of volatile organic compounds (VOCs) from contaminated tap water in a 3-compartment structure on the basis of source strength and air exchange. It also estimates lifetime average inhalation exposure, utilizing simplified activity data and 3 microenvironments: shower, bathroom, and rest of the house. Air

exchange is calculated from user-defined air residence times and compartment volumes. Sources are from user-defined emissions and timing. Multi-period exposure concentrations would be possible with hour-by-hour activity patterns. Mitigation measures affecting source duration, emission rates, and air exchange could be investigated.

California Institute of Technology Indoor Model (CIT)

Capabilities: 3a,3c,4,5,7

CIT (Nazaroff, 1986; 1989) is designed to estimate concentrations of chemically reactive compounds or particulate matter in a single or multi-compartments on the basis of air exchanges, removal and generation, source strength, and outdoor concentrations. Air exchange is calculated from used-defined airflows. Source strength is from user-defined emission and durations. Mitigation measures such as building design, air exchange rates and source strength are included. Both models utilize a well mixed assumption.

In the reactive version, pollutants may be generated or transformed on the basis of chemical kinetics. These relationships are only available for the species included. In the particulate version, size transitions and depositions by aerodynamics are treated. Multi-period averaging is possible for both versions.

14.2.3 Comments on practical use

Most of the indoor air models reviewed here calculate the air concentration of either a single or multi-room building. They generally utilize air exchange rates, removal rates and source strengths in the same manner. Differences in mixing, source terms and averaging times are evident. The CIT model includes sophisticated chemical and particle calculations that may be important in certain situations. IAQ and MACROMODEL contain sophisticated source estimation algorithms.

By far the largest difference among the models is the inclusion of population activity patterns in order to estimate personal exposure. This allows different issues, such as exposures to particular pollutants to be addressed. For specific concerns such as environmental tobacco smoke (ETS) or ozone exposures in heavily polluted regions, populations exposures are necessary for understanding the issues involved.

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Chapter 15

Risk Assessment: Risk Characterization[1]

The final step in the risk assessment process is the characterization of risk. In this step, numerical estimates of risk are presented along with a framework for evaluating those risks. The risk characterization section should summarize the hazard identification, exposure assessment, the dose–effect assessment, and the public health risk estimates. Major assumptions, scientific judgements, and to the extent possible, uncertainties should be included.

Non-carcinogen Risk Characterization: The risk for non-carcinogens may be characterized by a margin of safety (MOS) approach that is estimated by dividing the reference dose (oral or inhalation) by the estimated daily human dose (U.S. EPA, 1989). The MOS quantifies the ratio between potential exposures and a presumed safe level; it is not an absolute statement of risk, but a surrogate for risk. The larger the MOS, the smaller the risk. The MOS that is needed to protect human health will vary depending on the agent, and its selection is similar to the process used to select the uncertainty factors for the reference dose calculation.

Carcinogen Risk Characterization: The numerical estimates for carcinogenic risks can be calculated in one of three ways. The unit cancer risk, under an assumption of low-dose linearity, is the excess lifetime risk due to a continuous constant lifetime exposure of one unit of carcinogen concentration. Typical exposure units include ppm or ppb in food or water, or ppm or $\mu g/m^3$ in air. When the dose, expressed as mg/kg/day is used rather than the concentration, the term slope factor is used and represents the risk per unit dose. One can convert a slope factor to unit risk by multiplying the slope factor by the inhalation rate (20 m^3/day) and dividing by body weight (70 kg). The dose

1 A part of the text of this chapter has been derived from R.A. Rudel and B.D. Beck "Risk Assessment for Indoor Air: Evaluating Risks to Susceptible Populations" in: Report on a Joint Workshop "Methods of Risk Assessment for the Indoor Environment", pp. 67–80, Kloster Banz, Germany, 15-17 October 1991. Ed. B. Seifert. NATO/CCMS Pilot Study on Indoor Air Quality and ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man"); D.F. Naugle "Possibilities and Limitations of Indoor Environment Risk Assessment" in: Report on a Joint Workshop "Methods of Risk Assessment for the Indoor Environment", pp. 67-80, Kloster Banz, Germany, 15–17 October 1991. Ed. B. Seifert. NATO/CCMS Pilot Study on Indoor Air Quality and ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man").

corresponding to a given level of risk can be used in some instances-for example, when using nonlinear extrapolation models where the unit risk would differ at different dose levels. Finally, risks can be characterized either in terms of individual risks, population risks, or both.

Individual risks provide an estimate of the additional lifetime risk for individuals exposed to average and maximum levels of an agent. Individual risks can be obtained by multiplying the incremental unit risk (from the dose effect assessment) by the lifetime average daily exposure of that person (U.S. EPA, 1989).

Population risk estimates provide a measure of the possible total expected excess number of cancers in a population during a lifetime of exposure to a given agent. The population risk is derived by multiplying the individual risk by the total exposed population over some selected time period (U.S. EPA, 1989).

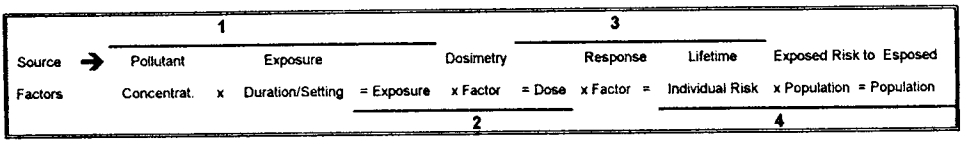
The usefulness of these estimates must be balanced with the likelihood that the agent is a human carcinogen. The likelihood is reflected by the letters A, B_2, B_1, C with "A" being the most certain and "C" being only a possible human carcinogen. The availability of a unit risk estimate does not modify the uncertainty associated with "B" or "C" designations nor are risks for A carcinogens necessarily more accurate than risks for "B" and "C" agents.

15.1 RISK CHARACTERIZATION FRAMEWORK

Increased emphasis on indoor air issues is likely to emerge with the realization that pollutant concentrations in indoor environments are generally greater than air quality levels measured outdoors and the fact that people spend a majority of their time indoors (Wallace et al., 1985). Indoor air quality (IAQ) strategies will likely focus on decentralized programs with risk reduction as a prime objective. Such strategies may emphasize materials substitution over air cleaning; localized mitigation options over rigid regulations; and mitigations (often by individual homeowners) motivated by the desire to reduce health risks. A Risk Characterization Framework is suggested to help organize complex technical results of research in a way which is useful to diverse audiences.

15.1.1 Predictive risk equation

A detailed review of studies which characterize risk attributed to indoor air pollutants has been conducted (Naugle et al., 1989). Not surprisingly, these studies utilized a variety of methodologies. Analysis of the common aspects of



1. Exposure

Pollutant Concentration (mg/m^3) x Exposure Duration (days) = Exposure (mg/m^3 x days)

Total exposure over a lifetime

2. Dose

Exposure (mg/m^3 x days) x Dosimetry Factors = Dose $\left(\frac{mg}{Kg \times day}\right)$

(contact rate: m^3 days)

(absorption rate: %)

(inverse average body Weight: $\frac{1}{Kg}$) or

(inverse days in lifetime: $\frac{1}{days}$)

Average dose per day over a lifetime

3. Lifetime Individual Risk

Dose $\left(\frac{mg}{Kg \times day}\right)$ x Response Relationship $\left(\frac{mg}{Kg \times day}\right)^{-1}\left(\frac{probability}{lifetime}\right)$ = Lifetime Individual Risk $\left(\frac{probability}{lifetime}\right)$

Individual Rrsk over a lifetime

4. Risk to Exposed Population

Lifetime Individual Risk $\left(\frac{probability}{lifetime}\right)$ x Exposed Population $\left(n^{\circ} people \times \frac{1 lifetime}{n^{\circ} years}\right)$ = Risk to Exposed Population $\left(\frac{n^{\circ} cases}{years}\right)$

Estimated cases in exposed population over a lifetime

Fig. 15.1: Elements of the predictive risk equation.

these studies lead to the formulation of a simple Predictive Risk Equation which could be used to express most if not all of the key elements utilized.

The four general equations that comprise the Predictive Risk Equation (PRE) are presented in Figure 15.1. Representative units are shown and are useful to illustrate dimensional consistency. The general equations shown are relevant to carcinogenic risk assessments, and may be useful in visualizing ways to address non-carcinogenic health risks.

Source → Pollutant		Exposure		Dosimetry		Response		Lifetime	Exposed Risk to Esposed	
Factors	Concentrat. x	Duration/Setting	= Exposure	x Factor	= Dose	x Factor =		Individual Risk	x Population	= Population
(B)	(C)	(D)	(E)	(F)	(G)	(H)		(I)	(J)	(K)
AC Systems	Environ.	Short Duration	Integrated	Contact	Pollutant	Carcinogenic		Incidence	Infants	
Ambient Air	Tabacco		by Direct	Rate	Mass per	Potency			Children	
Building Material	Smoke	Long Duration	Measur.	Absorption	Body				Home	
Copying Machines	Nonionizing			Rate	Weight				School	
Earth or Ground	Radiation		Calculated	Ave. Body	per Time				Adults	Incidence
Furnishing	Organics	Indoors, work	from	Weight					Male	
Gas stoves	Asbestos	Indoors, home	Columns		or			Endpoint	Female	
Household Prod.	inorganics	Outdoors	(C) x (D)	Ave.	Pollutant				Worker	
Insulation	Biologicals			Lifetime	Mass per				Homemaker	
Heaters, Kerosene	Radon/	Other		Regional	Surface				Smoker	
Tobacco Smoking	Progeny			Surface	Area per				Nonsmoker	
Vehicle Exhaust				Area of	Time				Other	
Woodstoves				Lung						
HAZARD IDENTIFICATION										
EXPOSURE ASSESSMENT					DOSE RESPONSE			RISK CHARACTERIZATION		
RISK ASSESSMENT										

Fig. 15.2: Risk characterization framework (for cancer health effects).

15.1.2 Framework for a systematic approach

The traditional risk assessment approaches were combined with elements of the Predictive Risk Equation (PRE) to produce the Risk Characterization Framework as presented in Figure 15.2. It has been developed to encourage a systematic approach for analysis and presentation of risk characterization study results. This framework subdivides the four components of the risk assessment process (as defined by EPA and the US National Research Council) into ten elements (Columns B–K in Figure 15.2) to provide a refined and systematic way of describing a very complex risk estimation process. Descriptions of each column are given here.

Column B (source factors) is the starting point for the PRE. The estimation of risk can be based on the study of a single source emitting one or more pollutants of concern or the study of a pollutant or mixture that is emitted from one or more sources.

Column C (pollutant concentration) of the framework records the numerical data of average or peak concentrations for each pollutant under study.

Column D (exposure duration and setting) of the framework combines the identification of each environmental setting in which exposure occurs and an estimation of the time spent in that environment.

For carcinogenic effects, duration is typically expressed as total hours or days of exposure during one's lifetime. For non-carcinogenic effects, one is also concerned about acute health effects from short-term exposures at elevated concentrations. Short-term durations of hours or even minutes may be important.

Column E (exposure) is frequently expressed as the product of the concentration of pollutant to which one is exposed in a particular setting times the duration of that contact.

Column F (dosimetry factor) of the framework addresses factors which influence how much of the exposure to a pollutant is available to, or passes through, an absorption barrier and enters the body. These factors include: contact rate, ingestion or inhalation rate, absorption rate, average body weight, average lifetime, regional surface area of the lung, and regional deposited dose ratio. Other factors may also be required in specific analyses.

As shown in Figure 15.2, Column G (dose) of the framework is the product of exposure estimates and the variety of modifiers discussed above. It represents the amount of a substance available for interaction with metabolic processes or biologically significant receptor. For toxicological studies involving animals under controlled conditions, the pollutant dose can be directly measured. For epidemiological studies correlating human disorders with exposures, the dose must be estimated.

Column H of the framework (response factor) describes the magnitude of the response of an individual to a given dose of the substance. For practical reasons, human observational data are usually available for very few of the different possible chemicals and exposure routes. It is therefore necessary to derive mathematical models of the dose–response relationship based on the best understanding of the mechanism of action of the toxic substance.

Column I (lifetime individual risk) of the framework gives estimates of the lifetime excess risk of cancer for an individual exposed at the given lifetime exposure. In their simplest form, carcinogenic risks are estimated by multiplying a lifetime average daily dose (Column G) by a potency factor (Column H).

Column J (exposed population) of the framework is provided for the types and number of affected subpopulations included in a risk analysis. A numeri-

cal estimate of the number of individuals in each subpopulation is therefore required. Also, each subpopulation must be linked to a specific exposure scenario. Ideally, in characterizing an exposed population or subpopulation, a distribution should be obtained that incorporates variability associated with age of the population, exposure levels associated with different activity patterns and micro-environments, and the susceptibility within the population to a specific effect.

Column K of the framework (risk to exposed population) is typically expressed as the expected or observed number of cases in the population or in presenting the population distribution for varying levels of risk. Risks estimated can be in a deterministic fashion with a predictive risk equation as described in Figure 15.1. Alternatively, when sufficient human data allows, a statistical analysis of epidemiologic data can be used to obtain a risk estimate in Column K. In the latter case, exposures (Column E) related to sources (Column B) must then be "backward calculated" (going from the right side of the framework to the left side) in order to suggest a cause and effect relationship.

15.1.3 Risk characterization for non-cancer effects

Since many indoor environmental complaints and illnesses involve non-cancer health effects, a modified framework has been developed for non-cancer risk approaches (Fig. 15.3). The mathematical operators are dropped between Columns (C) and (K) since few non-cancer risk applications are expected to use linear operations between data in the various columns.

For non-carcinogenic risks, individual risk may be estimated as the probability of a response (adverse health effect) or the degree to which exposure or dose exceeds a threshold for adverse health effects. The ratio of the exposure level to the threshold dose gives some indication of the likelihood of occurrence of the adverse health effects associated with exposure to the toxic substance. Threshold-based doses are most commonly established for chronic exposures, but may also be established for acute and subchronic exposures.

15.2 THE HUMAN EXPOSURE MODEL

The Human Exposure Model II, or the HEM II, is one of the most advanced exposure models. The HEM II has four components, called supervisors (Fig. 15.4):

1. the breathing air concentration, or BAC supervisor;
2. the population supervisor;
3. the exposure supervisor, and
4. the risk supervisor.

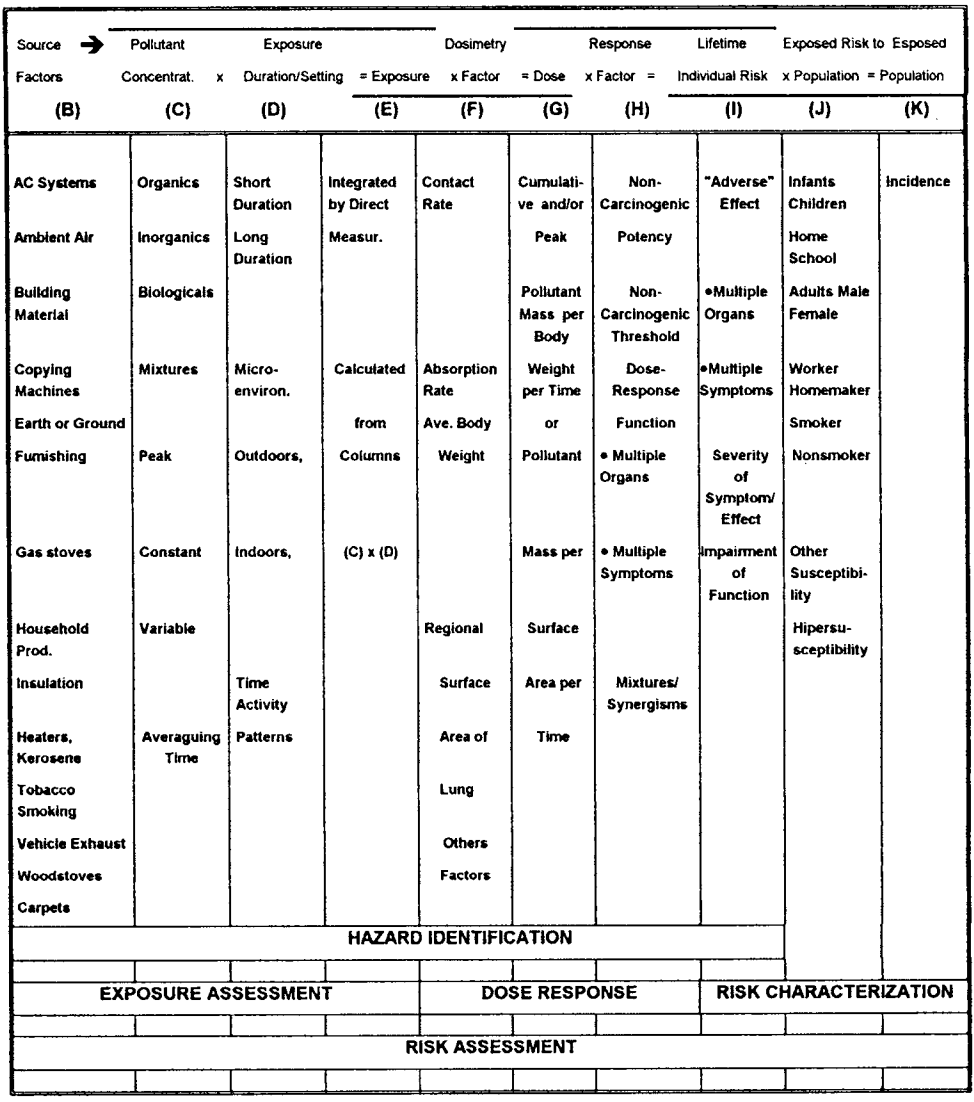


Fig. 15.3: Risk characterization framework (for cancer health effects).

These components are linked with the system supervisor, or central component (see figure), and furnish the system outputs: measures of exposure and risk.

The population supervisor uses census data to determine the exposed population; in addition the model has the ability to determine population growth, and mobility. This supervisor is also able to determine sensitive populations, and to estimate exposed populations in each of several microenvironments. The HEM II characterizes occupancy as a fraction of time spent

NEW HEM II

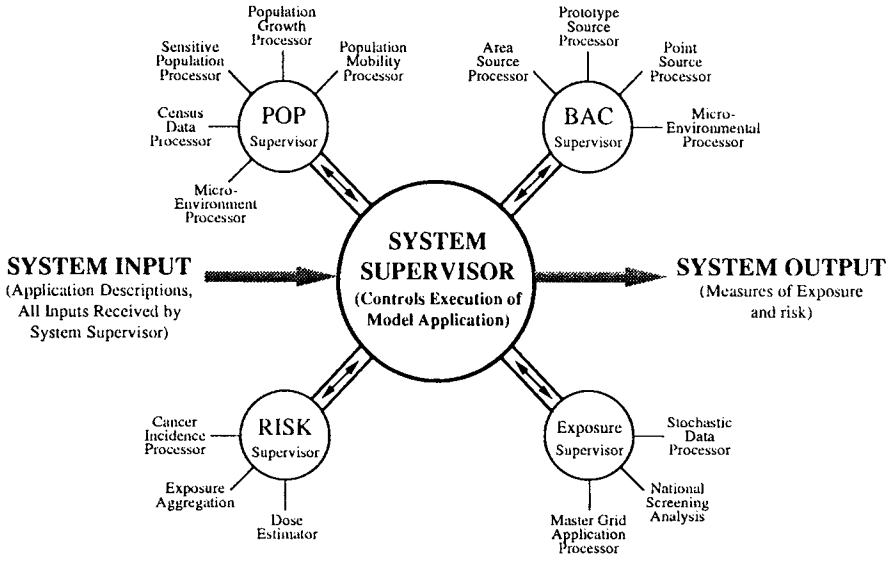


Fig. 15.4: Diagram of the New Human Exposure Model.

in each of the following microenvironments, the model assumes that individuals spent 0.37% of their day at home, 0,33% at the office, 0.05% in their automobile, and 0.25% outdoors.

The Breathing Air Supervisor (BAC) uses an air pollution transport model (the ISC-LT) to estimate pollutant concentrations from area sources, point sources, and line sources. The model requires all input information necessary for pollutant transport models including information on source/receptor terrain, on meteorological conditions, and on source parameters such as stack height, emission rate and exit velocity. The output from this supervisor is the level of the relevant pollutant outdoors as well as in several microenvironments. The model, or this supervisor, can be set to simulate multiple sources and multiple pollutants or risk agents.

The Exposure Supervisor combines information from the BAC supervisor with data from the population supervisor to estimate exposure of the public in the affected area. This component calculates two measures of exposure: the total or aggregate exposure and the maximum exposure. The final component of the HEM II transforms the exposure data to risk estimations by using EPA unit risk factors to estimate possible cancer incidence of the affected, exposed, population.

The model can evaluate many sources together, and is able to provide estimates of the risk attributed to emissions from all sources or to emissions from individual sources. The output of the HEM II can be one or more of the following: total exposure by source, total risk by source, total study-wide exposure, total study-wide risk, maximum exposure, maxim risk, and annual incidence.

15.3 EVALUATING RISKS TO SUSCEPTIBLE POPULATIONS

Research in risk assessment to date has primarily focused on defining the response of the majority of the population to toxic substances. Studies often attempt to define a threshold level below which most of the population does not respond to the agent. Although the slope of a dose–response curve provides an indication of the population variability, dose–response information for most pollutants is often not available for a heterogeneous human population. Because of difficulties associated with studying human populations, current understanding of the range of human sensitivities to environmental agents is still limited. Information on susceptible populations is generally qualitative rather than quantitative, and often little is known about the biological events responsible for differences in susceptibility.

This section reviews the current understanding of the responses of susceptible populations to environmental challenges. In addition, current approaches to assessing risks to susceptible populations are reviewed. The discussion is focused on pollutants and populations relevant to the study of indoor air pollution.

15.3.1 Susceptible populations and indoor air pollutants

Developmental status

The very young and the very old are known to be particularly susceptible to environmental challenge. Biological explanations for this susceptibility include changes in immune response with aging, maturation of enzymatic detoxification systems, decline in organ function with age, and other physiological changes (Calabrese, 1978; Warren and Weinstock, 1988; Weinstock and Beck, 1988). Consideration of the very young and the very old is particularly relevant to discussions of indoor air because these groups are likely to spend more time indoors than the majority of the population.

The ability to metabolize and therefore excrete chemicals varies considerably with developmental stage. Research shows that the rate of metabolism of

various compounds by the human fetal liver is about 35–40% of an adult liver, and some compounds are not metabolized at all by the fetal liver. Adult levels of most enzyme systems are achieved in humans by the age of 2–3 months (Calabrese, 1978). The inability to fully metabolize certain toxins results in the fetus or infant being more susceptible to the toxic effects of certain compounds than adults, although maternal metabolism may detoxify the compound to some extent prior to fetal exposure. On the other hand, in situations where adult metabolism of a compound results in toxic intermediates, a fetus or infant may be less susceptible than an adult (Calabrese, 1978).

In older populations with decreased liver or kidney function, the ability to metabolize compounds may be similarly impaired. The efficiency of other organ systems will also affect the host response to environmental challenge. For example, the inability of the lung to clear inhaled particles is likely to increase lung damage or irritation from exposure to particulate air pollution (Calabrese, 1986).

The immune system is an important component of the physiological response to environmental challenge. Immune response is still developing during infancy and early childhood, and becomes less effective in old age. Variations in immune system function make the elderly and infants particularly susceptible to biological and viral agents, as well as to some carcinogenic agents. For example, infants are developmentally deficient in the immunoglobulin IgA, which is known to play an important role in resisting respiratory tract infections (Calabrese, 1978; Calabrese, 1986).

There are a number of common indoor air pollutants for which developmental status is an important determinant of susceptibility. Children exposed to environmental tobacco smoke (ETS) have been identified as having increased rates of lower respiratory illness, chronic respiratory symptoms, and middle ear disease, as well as decreased lung function. In adults, ETS exposure is associated with an increased risk of lung cancer, but strong associations with other diseases have not been established (Samet, 1987).

Nitrogen dioxide exposure may also cause respiratory symptoms by damaging the lung directly through its oxidant properties, or indirectly by increasing susceptibility to respiratory infections. Neas et al. (1991) found that increases in nitrogen dioxide concentrations in indoor air were associated with an increase in incidence of respiratory symptoms in children aged 7 to 11. Respiratory symptoms included shortness of breath with wheeze, chronic wheeze, chronic cough, chronic phlegm, or bronchitis. Table 15.1 shows the results of this study. Regression analysis showed that a 15 ppb increase in household annual nitrogen dioxide concentration was associated with a statistically significant increase in such symptoms. Other studies have shown that children from homes with gas stoves (which increase nitrogen dioxide levels

TABLE 15.1

Indoor nitrogen dioxide concentrations and respiratory illness in children (adapted from Neas et al., 1991)

Nitrogen dioxide range	Concentration (ppb) mean	Cumulative incidence of respiratory symptoms (%)	Odds ratio
0–4.9	3.7	22.8	1
5–9.9	7.3	24.2	1.06
10–19.9	14.4	27.1	1.36
20–78.9	31	27.8	1.65*

*Statistically significant.

indoors) have a higher incidence of respiratory infection than children from homes with electric stoves (Samet et al., 1987).

Another common indoor air pollutant, carbon monoxide, interferes with oxygen transport by avidly binding to haemoglobin to form carboxyhaemoglobin, reducing oxygen delivery to tissues (Samet et al., 1987). Pregnant women may be susceptible to high concentrations of carbon monoxide because endogenous carbon monoxide production fluctuates considerably during pregnancy. The fetus may be at higher risk as well; carboxyhaemoglobin levels in fetal lambs were higher than in maternal lambs exposed to carbon monoxide. Haemoglobin in the fetus may have greater affinity for carbon monoxide, and endogenous carbon monoxide production differs between the fetus and mother (Beck and Weinstock, 1988).

Pre-existing disease

Persons with chronic respiratory disease, heart disease, emphysema, or other respiratory diseases are generally more susceptible to air pollutants than other individuals. The increased mortality of elderly persons and those with pulmonary disease during the air pollution episodes in Donora, Pennsylvania (October, 1948), and London, England (December, 1952) provides evidence that persons with pulmonary disease are more susceptible to air pollution than healthy individuals. Morrow and Utell (1989) found that volunteers with chronic obstructive pulmonary disease had decreased pulmonary function during exposure to 0.3 ppm nitrogen dioxide in conjunction with light exercise, while non-smoking age-matched controls did not. The extent to which the increased susceptibility is due to differences in particle deposition and clearance or host responsiveness is not clearly understood (Sweeney et al., 1988).

Disorders of the immune system can also increase susceptibility to environmental challenges. As discussed earlier, a weakened immune system can increase susceptibility to bacterial and viral infections and increase cancer incidence (Calabrese, 1978). Genetic disorders which are associated with increased cancer incidence also delineate a population that is susceptible to carcinogens. For example, persons with Bloom's Syndrome and Down's syndrome show chromosome instability or a decreased ability to repair DNA. Persons with these diseases have higher cancer incidence rates than the majority of the population (Calabrese, 1978; Shaikh et al., 1988).

Asthma

Asthmatics are another population which is susceptible to air pollutants. Asthma refers to a poorly defined family of disorders which are characterized by reversible bronchial swelling, airway narrowing, and overproduction of mucus (Solomon, 1990). Asthmatic responses can be triggered by a classic antigen induced allergic reaction mediated by IgE, or by non-antigenic or allergenic stimulus. Bronchial tissues of asthmatics may be sensitive to physical, chemical, immunological, and pharmacological stimulation. Asthmatics may respond to numerous indoor air pollutants, including tobacco smoke, animal dander, biological agents in house dust, nitrogen dioxide, wood smoke, and formaldehyde (Brain et al., 1988).

Behavioural characteristics

Behavioural characteristics such as occupational exposure, smoking habits, and nutritional status also influence susceptibility to environmental challenge. For instance, many work environments contain high concentrations of dusts, animal dander, respiratory irritants, and other chemicals. Continuous exposure to these environments occasionally results in sensitization to the particular compounds or the development of non-specific or allergen-mediated asthma (Brain et al., 1988).

Smokers are at increased risk of chronic airflow obstruction from environmental pollutants that cause airway narrowing. Pollutants which may cause airway pathology include common respiratory irritants (sulphur dioxide, nitrogen dioxide, ozone), specific airway sensitizing agents (common allergens, western red cedar, isocyanates), and miscellaneous dusts and fumes (Calabrese, 1978; Greaves and Schenker, 1988). Smokers are also at substantially increased risk of developing lung cancer from exposure to asbestos, radon, and carcinogenic volatile organic compounds (Collins and Schenker, 1988).

Nutritional status can also influence susceptibility to pollutants. A number of animal experiments have shown that animals exposed to carcinogens in conjunction with vitamin A had a lower tumour incidence than animals that were not given vitamin A. At the same time, vitamin A-deficient animals treated with carcinogenic agents had higher tumour rates than animals with appropriate levels of vitamin A. Vitamin C deficiency may potentiate the effects of several pollutants, including carbon monoxide, sulfates, radioactivity, benzene, and organochlorine insecticides. Guinea pigs exposed chronically to carbon monoxide showed increased consumption of and requirement for vitamin C, and the toxic effects of chronic CO poisoning were prevented by treatment with vitamin C (Calabrese, 1978).

Genetic variation

Genetic variation between humans results in subpopulations with biological differences from the majority of the population. A chemical idiosyncrasy is a genetically determined abnormal reactivity to a chemical (Klaassen et al., 1986). These genetic variations may result in differential susceptibility to environmental challenge. For example, enzyme deficiency may alter chemical metabolism, thereby decreasing excretion rates and potentially increasing tissue damage.

Persons deficient in glucose-6-phosphate dehydrogenase (G-6-PD) may be at higher risk from the haemolytic action of ozone. This population may also be susceptible to other substances known to cause haemolytic changes, including nitrogen dioxide and carbon monoxide.

A different idiosyncratic-type susceptible population are individuals who are deficient in immunoglobulin A (IgA). IgA is believed to act as a protective agent against foreign materials (such as ETS) on secretory surfaces. Studies show direct relationship of patients with recurrent upper respiratory tract infections and the presence of IgA deficiency (Calabrese, 1984).

Studies of the cytochrome P-450 enzyme system have found wide variations in the levels of these enzymes among individuals in the general population. The variation in aryl hydrocarbon hydroxylase (AHH) levels in 30 persons tested was approximately 80-fold. Because AHH is involved in the metabolism of some chemical carcinogens, it has been hypothesized that differences in AHH levels may be another source of inter-individual variation in susceptibility to certain carcinogens (Shaikh et al., 1988).

Sick building syndrome

Over the last two decades, outbreaks of health problems among employees in office buildings have been reported. These outbreaks have been described

as sick building syndrome, tight building syndrome, or building-related illness. Two broad categories of these episodes can be defined: those with a relatively clear clinical picture for which a specific aetiology can be defined, and those in which workers report non-specific symptoms temporarily related to work.

In cases where a causative factor has been identified building-related illness, infectious agents, immunologic sensitizing agents, specific air contaminants, and conditions such as temperature and humidity have been found to cause the outbreak. In cases with non-specific symptoms and no clear aetiology sick building syndrome, typical symptoms include mucous membrane and eye irritation, chest tightness, fatigue, headache, and malaise (Samet et al., 1988).

It is generally thought this syndrome is related to a variety of factors and exposures. Norback et al. (1990) reported that total hydrocarbon concentration seemed to predict chronic symptoms in personnel working in Swedish primary schools. The researchers also found that sick building syndrome was related to the historical presence of wall-to-wall carpeting in the schools and the concentration of respirable dust. In addition, current smoking of personnel, non-specific airway hyperreactivity, and psychosocial factors such as job satisfaction were associated with increased symptoms.

More than 900 different volatile organic compounds (VOC) have been detected in indoor air. Some of the most commonly measured VOC are known or suspected mutagens and/or carcinogens, while many are respiratory irritants or exhibit neurotoxic or hepatotoxic effects. Although the complexity of VOC mixtures prevents unambiguous identification of susceptible populations, these mixtures have been implicated in sick building syndrome based on the supposition that mixtures may produce effects that might not be expected with individual VOC. In addition, many of the VOC which are commonly detected in indoor air could contribute to the eye, mucous membrane, and respiratory tract irritations common in sick building syndrome. Some preliminary experiments support the theory that low levels of VOC mixtures may cause irritation and other effects in some populations (Samet et al., 1988), although the reasons for variation in response are unknown.

Formaldehyde is another common indoor air pollutant that has been implicated in sick building syndrome. There appears to be a wide range of concentrations at which symptoms of irritation occur. In some studies, the range of responsiveness between individuals for eye irritation was 200-fold and for upper airway irritation was 250-fold (Samet et al., 1988). The occupational exposure limit in the USA is 1.0 ppm (Sax and Lewis, 1989), although some individuals experience irritant effects at much lower concentrations. Persons susceptible to sick building syndrome and multiple chemical sensitivities (discussed below) may respond to formaldehyde at particularly low doses, and formaldehyde has been reported to cause asthma, particularly in occupation-

ally exposed workers. Chronic exposure to formaldehyde may be associated with increased incidence of nasal cancer (Samet et al., 1988).

Multiple chemical sensitivities

Multiple chemical sensitivities (MCS) is a poorly understood disorder. It has been defined as follows:

> "multiple chemical sensitivities is an acquired disorder characterized by recurrent symptoms, referable to multiple organ systems, occurring in response to demonstrable exposure to many chemically unrelated compounds at doses far below those established in the general population to cause harmful effects. No single widely accepted test of physiologic function can be shown to correlate with the symptoms." (Cullen, 1987)

Cullen elaborates on the definition by excluding allergic reactions such as those precipitated by occupational exposures to isocyanates or grains. This definition also requires that exposure that elicits symptoms be very low (e.g., less than 1% of established TLVs) but that exposure be demonstrable. Cullen specifically excludes most of the class of illnesses defined by clinical ecologists as Environmental Illness, restricting MCS to patients with a clear relationship between specific episodes of chemical exposures and subsequent symptoms. For further discussion on MCS, see in Chapter 6.

15.3.2 Risk assessment techniques for susceptible populations

Risk assessment for non-carcinogenic substances

Variations in susceptibility to environmental challenge can stem from differences in the relationship between exposure and dose (e.g., breathing volume), the relationship between dose and dose to target tissue, the ability to metabolize, distribute, or excrete a compound, or the responsiveness of the target tissue. In general, population response to environmental challenge often follows a unimodal distribution. This pattern of response reflects variability in exposure–dose relationships and in subject responsiveness; the endpoint is usually consistent across the range of sensitivities. Variation in response in some instances, however, follows a bimodal curve in which a distinct susceptible population has one distribution pattern and the majority of the population has a separate distribution. In these instances, the susceptible population may experience a different endpoint than the majority of the population (Froines et al., 1988).

The reference dose approach: use of uncertainty factors

To characterize the toxicity of non-carcinogenic substances, the U.S. Environmental Protection Agency (EPA) extrapolates a human Reference Dose (RfD), often based on toxicity data from laboratory animals. The extrapolation procedure accommodates expected human intra-species variability by lowering the "safe dose" by an uncertainty factor of ten. This uncertainty factor is expected to compensate for the fact that humans, having a diverse genetic make-up, are apt to experience significantly more interindividual variability than the homogenous laboratory animals. In addition, this uncertainty factor is expected to compensate for specific susceptible human populations who may be particularly sensitive to the effects of a particular chemical. This type of approach is also used by other U.S. regulatory agencies, such as the Food and Drug Administration.

Some research has focused on evaluating the appropriateness of using the uncertainty factor of ten as an indication of human inter-individual variability. Dourson and Stara (1983) analyzed variability in animal experiments of acute lethality (as reported by Weil (1972)) and found that for 92% of the 490 data points, a 10-fold decrease in dose would lower the LD_{50} below the $LD_{0.13}$. For the remaining 8% of the chemicals, a 10-fold decrease in dose would not achieve that reduction in response. Dourson and Stara note that the average variability in the data set was such that only a 2.4-fold reduction in dose would suffice to protect 99.9% of the test population. However, they add that these dose–response values were garnered from laboratory rats, which are expected to be less heterogenous in response to the toxicity of a contaminant than humans. In addition, intra-species variability may differ for endpoints other than lethality.

Dourson and Stara (1983) also report data on human variability (from Krasovskii (1976)) which indicate that there is a general 3 to 5-fold difference in sensitivity between children and adults, and that there is a 6-fold difference in sensitivity to the action of fluorine and nitrates between children. Thus, the overall intra-species variability of humans to the toxicity of some chemicals might be estimated to be as high as 18 and 30, or even higher if sensitivity due to disease and other factors is taken into account.

Hattis et al. (1987) examined human variability of pharmacokinetic parameters such as absorption, metabolism, and excretion rates for 49 chemicals (mostly drugs) in normal human volunteers. They found that for most of the parameters evaluated, the median chemicals had $\log_{10}$ geometric standard deviations of 0.11–0.145. Therefore, a 10-fold difference in these parameters would correspond to between 7 and 9 standard deviations in populations of normal healthy adults. However, for one chemical in the data set, inter-individual

variability for a parameter was considerably higher, such that a tenfold difference in the parameter would correspond to only about 2.5 standard deviations in the average human population. Hattis et al. (1987) found that the 5th percentile and 95th percentile response differed by factors ranging from less than 2 to more than 14. They note that the parameters studied are only components of overall susceptibility, and do not include variability in exposure- and response-determining parameters. Additionally, the study excludes most human variability resulting from age and illness. They conclude that when all sources of variability are included, "it is likely that a tenfold difference will correspond to fewer standard deviations in the overall population." Hattis found that inter-individual variability in specific parameters does not depart markedly from the Weil distribution of total variability in susceptibility to acute lethal effects of chemicals in groups of rats (discussed above).

Calabrese (1986) examined differential susceptibility in animals with respect to age differences. He found that while most age-related differences in susceptibility fall within a factor of 10, there are numerous exceptions to this rule. Calabrese comments that since the safety factor of ten may be exceeded in some cases by age differences alone, the factor may not always be sufficient because it is intended to account for all causes of intra-species variation.

In summary, it is difficult to generalize about inter-individual variability in susceptibility to pollutants. While studies have shown that the range of susceptibility is often less than 10-fold, exceptions to this "rule" are not at all uncommon. Consequently, risk management decisions should be based on chemical-specific toxicity information where possible.

Sensitive populations and the national ambient air quality standards — the probabilistic approach

The Clean Air Act in the USA specifies that primary National Ambient Air Quality Standards (NAAQS) were to be set low enough to protect the health of all susceptible groups within the population. Because of the impossibility of defining a threshold level below which no person would experience adverse effects, Congress provided to EPA such that protection should be afforded to all sensitive groups as a whole, but not necessarily to all members of each group. Thus EPA has attempted to characterize the major populations which are sensitive to each of the primary air pollutants, and then promulgate standards which are protective for all but a certain small percentage (e.g., 1%) of that sensitive group (Frank, 1988). Thus EPA must acquire quantitative information on the range of sensitivities that exists in the general population.

As an example, EPA has determined that exercising asthmatics are the primary susceptible population for sulphur dioxide. The sulphur dioxide

NAAQS is based on clinical and epidemiological studies in which investigators attempted to determine the concentrations of sulphur dioxide that caused airway resistance in exercising asthmatics. Consequently, the variability in response of young, exercising asthmatics to acute clinical sulphur dioxide exposure has been extensively studied. Figure 15.5 shows the distribution of individual airway sensitivity to sulphur dioxide from experiments in mild asthmatic volunteers (U.S. EPA, 1986). It is interesting to note that the dose response curve in the figure shows at least a 30-fold range of susceptibilities within the group characterized as mild asthmatics. One can assume, therefore, that variation in response between non-asthmatics and severe asthmatics is much greater than 30-fold. In this particular case, a factor of 10 would be too low to represent inter-individual variability.

Unfortunately, for many pollutants the dose–response of the majority of the population is not understood well and the response of susceptible individuals may be understood even less. Clinical studies with sulphur dioxide were possible because the observed adverse effects are relatively mild, are reversible, and are observable after a brief period of exposure. For many indoor air

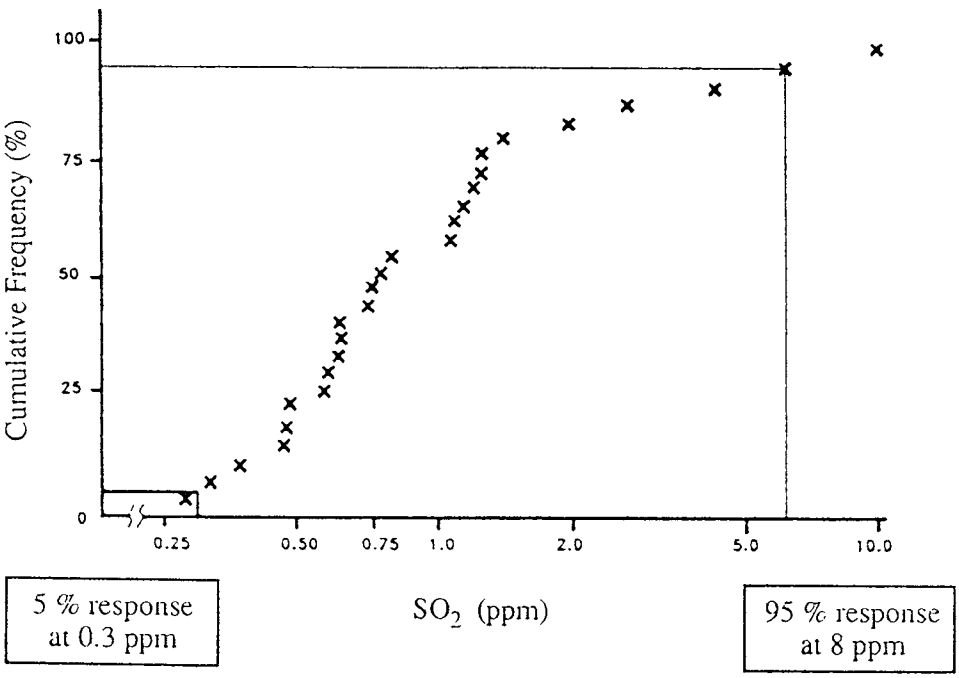


Fig. 15.5: SO_2 concentrations causing 100% increase in airway resistance in mild asthmatics (adapted from U.S. EPA, 1989).

pollutants this may not be the case. The chronic toxicity of pollutants or the effects of pollutants with non-reversible effects cannot be clinically tested in humans. Even in the case of sulphur dioxide, the clinical trials were performed on persons with mild asthma; for ethical reasons, it is not always possible to experimentally characterize the response of the most susceptible members of the susceptible population.

Methods such as physiologically-based pharmacokinetic (PB-PK) modelling may be important in attempting to predict the responses of certain sensitive groups. PB-PK models can address variability in parameters such as ventilation rates and percent body fat as well as differences in innate metabolism. Application of this technique requires substantial information on chemical metabolism and toxicity. Furthermore, it must be noted that significant uncertainties are involved in PB-PK modelling. Hattis et al. (1990) found that differences in data sets used to calibrate PB-PK models resulted in appreciable differences in model predictions.

Risk assessment for carcinogens

The very nature of risk assessment for carcinogens makes identification and assessment of susceptible populations extremely difficult. In a large group of persons exposed to a carcinogen, the risk resulting from the exposure will be characterized by the number of excess cancer incidences observed in the exposed population. Although it can be assumed that some individuals may be innately more susceptible to certain carcinogens, there is no way to differentiate the exposure-response relationship of the susceptible individual from that of the normal individual. In contrast, for toxic effects, the spectrum of susceptibility can be seen in the range of doses which provoke a response (expressed as the percent of the population exhibiting a defined response).

Populations which are susceptible to carcinogens can be identified experimentally when the exposed population can be grouped according to suspected degrees of susceptibility and then incidence rates between groups can be compared. This type of experiment can provide quantitative information on variations in susceptibility. For example, the variable susceptibility of different age groups to certain radiation-induced cancers has been illuminated somewhat through studies of the survivors exposed to radiation from the bombs at Hiroshima and Nagasaki. These studies show that for many types of cancer, the excess incidence 11 to 30 years after exposure increases with age at the time of exposure, suggesting that susceptibility to the carcinogenic effects of radiation increases with age (Warren and Weinstock, 1988). However, very few sources of data exist which are of the magnitude necessary to illustrate such inter-individual variability for humans.

In the case of carcinogens whose pharmacokinetic characteristics in humans are understood, it is possible to identify or predict susceptible populations based on known differences in pharmacokinetic parameters. For example, it has been suggested that variations in the inducibility of cytochrome P-450 enzymes in humans may be associated with susceptibility to certain cancers. Some studies have shown an association between levels of aryl hydrocarbon hydroxylase (AHH) and incidence of cancers of the respiratory tract or oral cavity. Other studies, however, have found no such association (Collins and Schenker, 1988).

Conclusions

It is clear that the range of human susceptibilities for some pollutants is far greater than the range traditionally used in risk assessment to represent inter-individual variability in humans (factor of ten), while for other pollutants, a factor of ten may be too large. Professional judgment based on the best available data must be used when developing health protective toxicity values.

For non-carcinogenic pollutants, it would be useful to inventory where quantitative risk assessment methods are adequate to account for sensitive populations and where they are not. Future research can be prioritized by evaluating the number of people affected by particular pollutants and the severity of the effects. For example, environmental tobacco smoke (ETS) is a prevalent indoor air pollutant with a wide range of potentially serious effects. ETS has been shown to increase rates of respiratory illness and middle ear disease and decrease lung function in children. In addition, it is a suspected human carcinogen. Quantitative information on the effects of ETS on either normal or sensitive populations is limited and considerable research remains to be done before the mechanisms are understood and the factors which make certain individuals more susceptible can be characterized.

Characterizing population responsiveness is in itself a difficult task. Determining the percent of the population that the susceptible population represents is an important step in prioritizing research needs. Quantitative methods for evaluating the severity of effects have also not been fully developed. Furthermore, characterizing bimodal dose–response curves, such as those associated with allergic responses or multiple chemical sensitivities, is an important research priority because these populations exhibit an enormous range of susceptibilities and the mechanisms of these responses are uncertain.

Pharmacokinetic and pharmacodynamic modelling are promising methods for evaluating population variability quantitatively. These methods can incorporate specific information on variation in absorption or metabolism rates

which have the effect of increasing susceptibility to certain pollutants. Models can also be used to determine the effect of other human variations, such as differences in responsiveness or exposure–dose relationships, on individual susceptibility.

In the case of risk assessment for carcinogens, the uncertainties inherent in low dose extrapolation methods are so great that research efforts focused on developing more biologically plausible dose–response models are likely to be more useful than efforts aimed at characterizing inter-individual variability. For example, models for carcinogenesis which allow for threshold effects must be developed before the effects of certain metabolic variations on susceptibility can be assessed. Similarly, pharmacodynamic models of carcinogenesis which incorporate cell proliferation and clonal expansion rates must be developed before the effects of variations in these dynamics can be considered in carcinogen risk assessment. Once methods have been developed for characterizing carcinogenesis in the majority of the population, these methods can be refined to account for variability between individuals.

15.4 LIMITATIONS OF THE RISK ASSESSMENT METHODS

Risk assessment methods are more applicable for some pollutants and health effects than others due primarily to the level of information available on hazard identification, dose–response relationships and exposure or dose profiles for individuals, information deficiencies, and to the methods themselves which have not been fully developed for application to indoor environments.

The methods are most applicable for individual pollutants for which the health effects and dose–response relationships have been well-characterized and exposure measurement data or data for modelling exposure are available. In this regard, risk assessment for certain carcinogens such as radon, asbestos, formaldehyde, benzene and environmental tobacco smoke have been conducted for indoor exposures. Also in this regard, risk assessments for non-carcinogenic effects for individual indoor pollutants such as lead have been conducted. However, the application of risk assessment methods to non-cancer effects are greatly limited by a lack of information on dose–response relationships. Given this situation, the approach taken for characterizing the non-cancer health "risk" attributable to exposure to certain indoor air pollutants has been to modify the methods such that risk are characterizing as the ratio of predicted or measured exposure to a health benchmark such as a Reference Dose or Reference Concentration. A ratio greater than one indicates an exposure level of concern and the higher the ratio, the greater the level of concern.

Risk assessment methods are less applicable for non-cancer health effects associated with exposures to complex mixtures in the indoor environment. There are several very important data limitations in this contest. First, the non-cancer health effects associated with low level exposures to complex mixtures have not been well characterized for any mixture, with the exception of environmental tobacco smoke. Secondly, the relative concentration of each of the pollutants in a mixture will change over time due to different decay rates, sorption coefficients and other factors. This change in relative concentration over time results in difficulty characterizing exposure over various time-averaging periods. Thirdly, dose–response relationships have not been established for non-cancer effects of complex mixtures. The methods have been modified as discussed above. However, this requires that the concentration of each pollutant in the mixture can be determined and a time-weighted exposure profile can be estimated. The ratio of the exposure concentration to a health benchmark for each pollutant can be summed for pollutants effecting the same target organs. This summation process, however, has serious shortcomings with regard to the mechanism of action of each pollutant, synergistic or antagonistic interactions among pollutants, and over all characterization of exposure to the complex mixture.

Risk assessment methods have very limited applicability for many adverse health effects associated with Sick Building Syndrome (SBS), such as headaches, dizziness, respiratory distress and the like. Since risk assessment is a predictive tool, it has no application to settings in which building-related complaints (BRC) or SBS-related symptoms are being reported. However, risk assessment methods may be used to predict whether the air quality inside a building or emissions from a particular source in a building will result in BRC or SBS-related complaints. The principal limitation to such an application at this time is the nearly total lack of dose–response data for individual pollutants and these types of adverse health effects. In addition, there are certain individuals within the general population who are much more susceptible to certain pollutants causing adverse health effects than in the general population. To adequately apply risk assessment methods, susceptible subpopulations need to be identified and their dose–response functions characterized.

Risk assessment methods are not applicable to exposure settings in which the health endpoints of concern might be attributed to other exposure factors such as the physical setting and psychological and social factors.

Risk assessment methods are also not applicable to exposure settings in which sensitization has occurred, resulting in hypersensitivity to certain types of exposure. However, if the dose–response relationship can be characterized for these hypersensitive individuals, then application of the methods might be more feasible.

Principal areas of future inquiry that will broaden the applicability of risk assessment methods to indoor exposures include the following:

- the characterization of adverse health effects of complex mixtures;
- the characterization of dose–response relationships for a variety of non-cancer endpoints associated with SBS; and
- the identification of hypersensitive individuals within the population, with regard to specific pollutants or exposures, and the characterization of the dose–response relationships for such exposures.

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Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 16

Application of Risk Assessment I: Radon[1]

16.1 INTRODUCTION

Natural radiation has always been part of the human environment. Its main components are cosmic and cosmogenic radiation, terrestrial gamma radiation from natural radionuclides in rocks and soil, and natural radioactive substances in our diet and in the air we breathe. Until the end of the 1970s the doses received by the vast majority of the general population from natural radiation were considered to be "background" phenomena of little significance, and the average annual dose was estimated to be about 1 mSv. Here and throughout this account the term dose refers to effective dose (see Section 3.4.1.1).

Special situations were, of course, known to exist. For example, uranium and other underground miners received large doses, and consequently were subject to elevated cancer risks, due to prolonged exposure to high concentrations in air of the radioactive decay products of the natural radioactive gas radon-222 (NRC, 1988). Radon-222 is a naturally occurring radioactive gas with a half-life of 3.82 days (see decay scheme in Figures 3.1a,b). It exists in several isotopic forms. Only two of these isotopes occur in significant concentration in the general environment: radon-222, a member of the radioactive decay chain of uranium-238, and radon-220 (often referred to as thoron), from the decay chain of thorium-232. The contribution made by thoron to the human exposures in indoor environments is usually small compared with that due to radon, due to the much shorter half-life (55 sec), and it will only occasionally be referred to here.

It should be noted that exposure to radon is not a new phenomenon and documentary evidence from as far back as the 16th century indicates that elevated radon exposure was probably responsible for excess lung cancer mortality of miners in some Central European mines, such as the silver mines in Bohemia (see Jacobi, 1993 for historical notes).

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1 A part of the text of this chapter has been derived from ECA (European Concerted Action "Indoor Air Quality and its Impact on Man", COST Project 613), 1994. Radon in indoor air, Report No. 15. Luxembourg: Office for Official Publications of the European Communities.

The view that natural radiation was of little radiological health significance for the general population in most countries changed dramatically with the discovery in the 1970s and 1980s that some homes in a number of countries had indoor radon levels present at concentrations of many hundreds up to some thousands of Bq/m^3 (Gunning and Scott, 1982). For people in such houses the doses are in the range of some tens of mSv per year and the associated risk of lung cancer is estimated to be substantially greater than the general population risk for this disease.

Within European countries national surveys to date have shown that the average indoor radon concentration is in the 20 to 123 Bq/m^3 range (see Table 3.4). Regional average values above this range have been found in some countries. A good example of this is the UK which has a national average value of 21 Bq/m^3 while Cornwall in south-west England has an average value of about 170 Bq/m^3. As far as the maximum indoor radon concentration likely to be present in any country is concerned it is impossible to estimate its value. Concentrations greater than 100 000 Bq/m^3 have already been detected in individual dwellings in some countries. In most situations it appears that elevated indoor radon levels originate from radon in the underlying rocks and soils. While national average indoor radon levels in European dwellings are typically in the 20 to 123 Bq/m^3 range a small percentage are considerably above this range. In keeping with the current Recommendation of the Commission of the European Communities (CEC, 1990), if 400 Bq/m^3 is taken as an "action level" for an existing dwelling (see also Section 34.1) then in many surveys the percentage of dwellings in excess of this level ranges from about 0.5 to 3%.

Radon is one of a very small number of substances which has been established to be a human carcinogen on the basis of human studies. As such it is a Group 1 and Group A carcinogen, according to the classification used by the World Health Organisation (WHO/IARC, 1988) and by the US Environmental Protection Agency (EPA, 1987a), respectively. The principal adverse health effect arising from the inhalation of radon and mainly its decay products is lung cancer. Recent suggestions (Henshaw et al., 1990, 1992) that exposure to elevated levels of indoors radon may be implicated in the occurrence of other cancers such as childhood leukaemia have not yet been scientifically verified. When radon and its short-lived decay products are inhaled the radiation dose to lung tissue is dominated by the alpha particles emitted by the deposited decay products. From a health perspective the daughter products of most significance are the four short-lived ones from polonium-218 to polonium-214 inclusive. The contribution to lung dose arising from the radon gas itself is small in comparison, as very little radon is absorbed: radon gas acts as a "carrier" of radon daughters.

Currently there are different approaches used to estimate the lung cancer risk arising from exposure to radon decay products in indoor air. These are: (a) the dosimetric approach, in which the radiation doses to lung tissues is estimated and from this estimated dose the associated risk is evaluated using currently accepted dose/risk factors for ionising radiations; (b) the mines epidemiology approach, in which risk estimates for underground miners are modified and applied to the general population; (c) the residential epidemiology approach, in which case-control studies of the general population are used to estimate risk factors. The risk factors obtained using these three approaches seem to be reasonably well in agreement (see next sections).

16.2 EXPOSURE EVALUATION FOR RISK ASSESSMENT

In all the three different approaches (see Fig. 16.1) used to estimate the risk due to the exposure of population to radon progeny in indoor air, the first step consists in evaluating the exposure.

The indoor exposure E in the time interval t is evaluated by the following formula:

$$E = C_{Rn} \cdot IOF \cdot t$$

where C_{Rn} is the average indoor radon concentration and IOF is the indoor occupancy factor, i.e. the fraction of time spent indoors.

To assess the risk, a reliable estimation of the indoor C_{Rn} is needed (see also Section 27.3.4). Therefore it is advisable to carry out measurements of long duration (at least one year), so that fluctuations due to climate, meteorological situation and lifestyle in the dwelling can be duly averaged. Short time (i.e. for a few days) measurements, which are usually performed with closed windows and doors, should be avoided as they could give rise to significant overestimates of C_{Rn} (Sextro, 1990). Moreover the measurements have to be carried out with adequately calibrated and reliable instruments, and intercomparison exercises should also be regularly performed.

The occupancy factor cannot in practice be measured, because it would require keeping a rigorous diary of activity for too long a period, but it may be estimated by the responses given to a questionnaire during radon surveys (e.g. Benassai et al., 1990; EPA 1992d), or by following special inquiries (Francis, 1987).

In radon epidemiological studies a major problem with exposure evaluation exists because radon levels in the past, when the subjects received most of their exposure, may differ significantly from those at present in their dwell-

ings. In Sweden, for example, it has been shown that the average indoor radon levels seem to have increased in recent decades probably because of energy conservation practices (Swedjemark and Hubbard, 1993). To address this problem techniques are currently under development, with the support of the CEC Radiation Research Programme, to make retrospective assessment of radon exposure in dwellings, based on the measurement of polonium-210 levels in the surface of glass (Samuelsson, 1988).

As an example of risk assessment calculation (see Fig. 16.1), it will be considered a lifetime (70 years) exposure to 400 Bq/m^3 — the CEC action level (see also Section 34.1) for existing dwellings (CEC, 1990) — with an indoor occupancy factor of 0.8, which is the value adopted by UNSCEAR (UNSCEAR, 1988, 1993) and ICRP (ICRP, 1993).

16.3 RISK ESTIMATION (A): THE DOSIMETRIC APPROACH

One of the three ways to estimate the risk due to the exposure of a population to radon progeny in indoor air is carried out in two stages. In the first one the effective dose to the lung is calculated through complex lung models, which take into account both physical parameters (radon and radon progeny concentration, fraction of progeny attached to aerosols, unattached fraction, aerosol size distribution, etc.) and physiological parameters (characteristics of the respiratory tract, respiratory rate, thickness of bronchial epithelium, location of target cells, etc.). These models (e.g. NEA/OECD, 1983) are continuously evolving. An improved model of the respiratory tract for use in a wide range of circumstances has been recently adopted by ICRP (ICRP, 1994).

It must be emphasised that although the dose is mostly due to inhaled radon progeny (and its characteristics) rather than to radon, some studies (James, 1987; Postendörfer and Reineking 1992; Vanmarke et al. 1989) show that, in domestic ambient air, changes in ventilation rate produce opposite variations in the equilibrium factor F (i.e. the ratio between radon progeny and radon gas concentration) and in the unattached fraction, so that the adsorbed dose (see Section 3.4.1.1) to the lung remains relatively constant at a given radon concentration. After absorbed dose is calculated by models, weighting factors (related to the specific effects of alpha particles and to the lung sensitivity to radiation, as explained in Section 3.4.1.1) have to be applied in order to obtain the "effective dose", which is the quantity considered to be proportional to the health effects (ICRP, 1991).

Different values of the effective-dose/exposure factor have been proposed (see UNSCEAR 1988 and 1993 for a review). Here, for example purposes only,

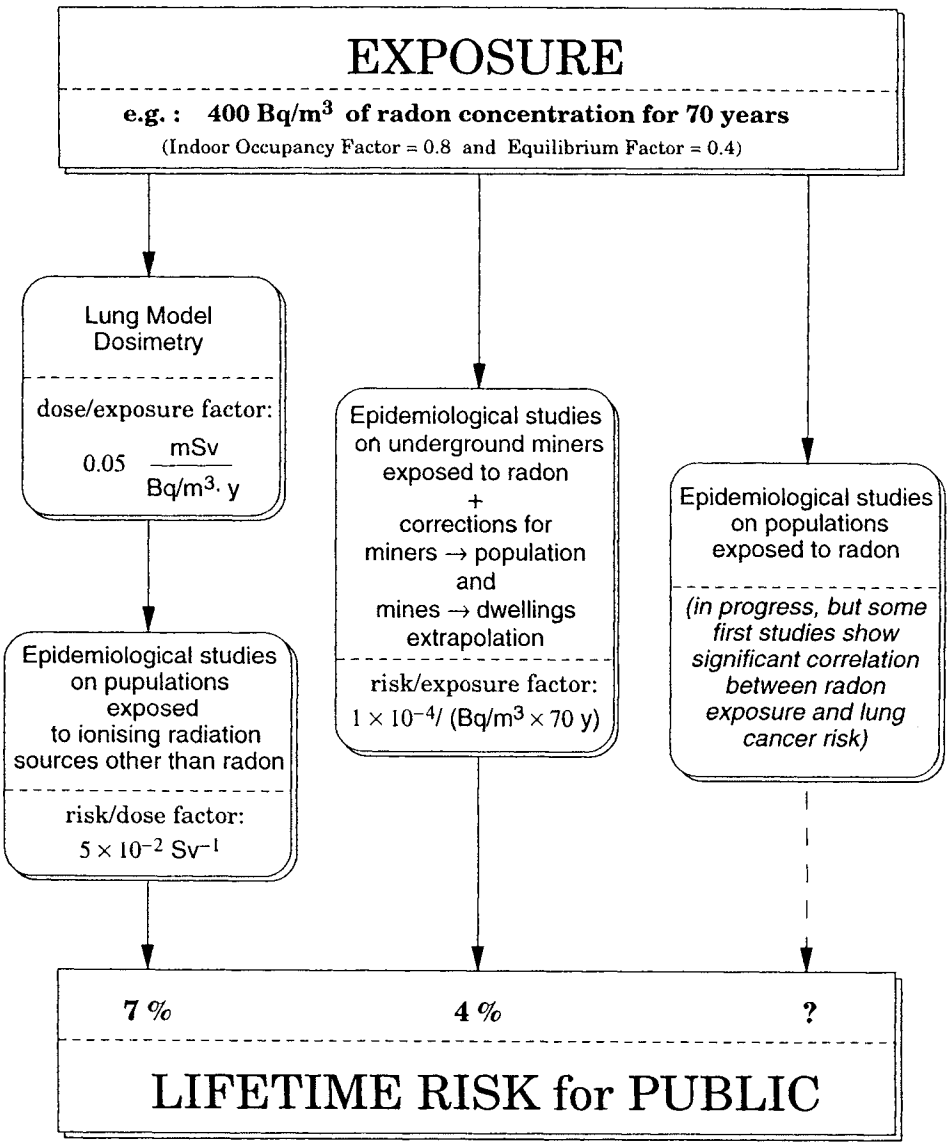

Fig. 16.1: Example of lifetime risk assessment for a chronic exposure (70 years) of general public to radon indoors.

the value adopted by CEC (CEC, 1990) and NRPB (Wrixon et al., 1988) will be used, i.e. 0.05 mSv/y per Bq/m^3 of mean annual radon concentration, with an indoor occupancy factor equal to 0.8 (see Fig. 16.1). Therefore, for 400 Bq/m^3 the effective dose is 20 mSv/y. Another widely used dose factor is 0.025 mSv/y per Bq/m^3, that is adopted by UNSCEAR (1993).

In the second stage of the dosimetric approach the risk connected with the effective dose to the lung is evaluated. This evaluation is made using the results of the epidemiological studies on persons exposed to ionising radiation, mainly studies on survivors of Hiroshima and Nagasaki, although in this case the exposure conditions were very different from those due to residential radon. In particular Hiroshima and Nagasaki survivors were exposed to gamma and neutron radiation, while the main radiation emitted by radon progeny is alpha radiation. In the last Recommendations of the International Commission on Radiological Protection (ICRP, 1991), a review of the most recent studies is made, adopting a risk/dose factor for the general public of 5×10^{-5} probability of fatal cancer per mSv of effective dose. Using this figure, 20 mSv/y would imply a 1×10^{-3} annual risk, corresponding to a 7% lifetime risk for a chronic exposure of 70 years.

The main problems of the dosimetric approach are the appropriate choice of the weighting factors to obtain the effective dose (Birchall and James, 1994) and the use risk/dose factors derived from epidemiological data for persons (such as the Japanese survivors) exposed to radiation other than that from radon progeny. For these and other reasons the present position of ICRP (ICRP, 1993) is to use only the miner epidemiology approach (see below) to estimate the risk. Nevertheless, ICRP introduces a "conventional" dose factor, which is derived so as to obtain the same risk from the dosimetric approach as from the miner epidemiology approach.

16.4 RISK ESTIMATION (B): THE MINER EPIDEMIOLOGY APPROACH

Another approach for the evaluation of risk is the analysis of epidemiological data of persons exposed to radon progeny. However for historical and other reasons, epidemiological studies on underground miners alone have not been available until now, except for some preliminary results of epidemiological studies on general population. In fact, as described in the introduction, it is only in the last 10–15 years that increasing attention has been devoted to radon exposure at home, while knowledge of radon effects on underground miners dates back many years. Moreover, in underground mines, radon concentrations were usually very high[1] — due to the fact that the main radon source is the ground itself — so that the related effects, in terms of excess lung cancers, are more evident. For the principal miner cohorts studied more than 700 excess deaths from lung cancer have been recorded (ICRP, 1993). It is worth noting that this excess is considerably higher than the deaths from all

1 In recent years improved ventilation etc. has been installed in mines to reduce the risk.

cancers that have been attributed to radiation in the lifespan study of the Japanese atom bomb survivors (ICRP, 1991).

Several cohorts of miners have been, and continue to be, analysed over the years. In order to apply the results to radon exposure of the general public in dwellings, the miner data have to be corrected to take into account the differences between miners (strong adult males) and the general public, and between indoor air characteristics in mines and in dwellings. In these studies the risk/exposure factor is usually reported per unit of cumulated exposure to radon progeny, the WLM unit, because radon progeny concentration is the quantity measured in mines. Hereafter this factor will be converted and expressed per unit of radon gas concentration (as described in Section 3.4.1.1), using an equilibrium factor of 0.4 and an indoor occupancy factor of 0.8, which are the values adopted by UNSCEAR (1988) and ICRP (1993). The results of the three major and more recent studies, performed by ICRP and the U.S. Committee on Biological Effects of Ionizing Radiation (BEIR), can therefore be expressed as 0.72×10^{-4} (ICRP, 1987), 1.1×10^{-4} (NRC, 1988) and 0.87×10^{-4} (ICRP, 1993) lifetime risk for a chronic exposure of general population during 70 years to 1 Bq/m^3 of radon gas concentration. These studies, whose results can be considered in good agreement, will not be analysed in depth. It should be noted, however, that different baseline cancer rates are used to convert the results from relative risk to absolute risk: ICRP (1987) refers to a reference population with a baseline cancer rate of 400 cases per year/10^6 persons, NRC (1988) refers to the USA population only, while ICRP (1993) refers to an "average population" composed by populations of United States, Puerto Rico, Japan, the United Kingdom and China. Moreover ICRP (1987) uses a correction factor of 0.8 to extrapolate the miner epidemiology results to the general public in dwellings, while in the other two studies this correction factor is assumed to be equal to one (although different decisions have later been taken, as outlined in a subsequent section). In the example under consideration (see Fig. 16.1), an approximate lifetime-risk/exposure factor of 1×10^{-4} per Bq/m^3 during 70 years will be used, obtaining a lifetime-risk of 4% for a chronic exposure to 400 Bq/m^3 of radon concentration.

It is of interest to note that a possible new miner epidemiological data set for radiation risk estimation of radon exposure has recently become available in Germany (Martignoni et al., 1991). As one of the results of the reunification of Germany, access has now become available to the exposure data on uranium miners who worked at various times during the period 1946 to 1989 in the Soviet controlled "Wismut" uranium mining and milling consortium in the former German Democratic Republic. During the peak production years (early fifties) the work force, including forced labour, amounted to between 100 000 and 150 000 persons. During this period it is roughly estimated that annual

exposures of 30 to 300 WLM were being received by the workers. The exposures reduced in subsequent years. Initial studies indicate that these "new" German uranium miner data are of a similar or probably higher quality than the data from other epidemiological studies concerned with the risk of bronchial cancer in miners. The size of the German miner population is much higher than all other known miner cohorts as is their known number of bronchial cancer cases.

16.5 RISK ESTIMATION (C): THE RESIDENTIAL EPIDEMIOLOGY APPROACH

The difficulties and the uncertainties in extrapolating the epidemiological results from miners to the general public and the growing interest in the radon problem in dwellings have recently induced many countries to undertake epidemiological studies on the general population exposed to radon progeny in dwellings. Many of these studies are coordinated, e.g. the same protocols are used, in order to facilitate the pooling of the results (DOE/CEC, 1989, 1991). These studies take many years to complete and are difficult, thus for most of them, which are under way at present, the final results will not be available in the immediate future. These kinds of studies are expected to produce the most appropriate risk estimation.

A good example of this approach is a study carried out in Sweden (Pershagen et al., 1992). This study included 210 women with lung cancer and 191 hospital and 209 population controls. For the study subjects the radon concentrations measured in their various lifetime residences showed an approximately log-normal distribution with arithmetic and geometric means of 122.2 and 96.0 Bq/m^3 respectively. Even at these slightly elevated indoor radon levels the risk estimates appeared to be within the range expected on the basis of the miner studies. In a more extended study (Pershagen et al., 1994) – which includes 1360 (586 female and 774 male) lung cancer cases and 2847 controls, the risk estimate is lower, but still statistically significant and consistent with the previous study.

16.6 UNCERTAINTIES IN RADON RISK ASSESSMENT

Uncertainties in risk assessment are connected in a number of ways as described above. In the following a brief analysis of the various sources of uncertainty is presented.

Exposure uncertainties

The uncertainties in exposure are due not only to measurement techniques, but also to the procedure used to estimate personal exposure. Actually no "personal dosemeters" are used, but "environment measurements" and occupancy factors are utilised to obtain person exposure, especially for long periods of time. A tentative to compare "personal monitoring" with "environmental monitoring" has been recently published (Litt et al., 1990), but it refers to an exposure of few days. The uncertainties can be summarised as follows:

Measurement technique. Passive track dosemeters — in which alpha particles emitted by radon and radon progeny produce tracks that are subsequently counted — are usually utilised for long-term measurements. The overall measurement uncertainty due both to calibration and reproducibility usually ranges from 10 to 30% (one standard deviation), depending on the actual radon concentration and other factors.

Measurement period. As previously underlined, one-year integrated measurements are usually performed to get good estimates of the mean radon concentration. However the results could differ from year to year, for instance due to strong climate changes. For example, measurements carried out for a 5-year period in 40 residences near the DOE Radon Laboratory of Colorado show a mean coefficient of variation approximately 22% (Martz et al., 1991). Moreover dosemeters continue to measure even when persons are not at home, and this could introduce a little bias in case of significant difference in radon concentration with respect to the period of the day when persons are at home or at work.

Retrospective assessment. As previously mentioned, retrospective assessment of exposure is required in case-control epidemiological studies. Usually this assessment is made measuring at present the radon concentration in all dwellings used in the period under study. This procedure could introduce a high bias, that can be tentatively limited if a strict protocol for case and control selection is used, i.e. excluding all cases and controls who lived in houses that have had any significant structure change that could effect radon concentration. An alternative experimental technique for retrospective assessment of radon exposure based on the build-up of polonium-210 on glass surfaces in dwellings is under development (Samuelsson, 1988). A similar approach is also now under development in which the build up of Po-210 in porous materials (volume trap) in dwellings is measured as an aid to retrospective assessment of radon exposure.

Sampling location. The radon detectors are usually positioned in one room of the house, preferably in bedrooms where most time is spent. This introduces a bias in those cases where the radon concentration varies appreciably from

room to room, as it could happen in multistorey dwellings, where significant variations could exist between the ground and upper floors.

Occupancy factor. As outlined above, the occupancy factor is very difficult to measure in practice. Personal judgment is often the only way for its estimation, especially in case of long periods. This factor is widely variable among persons, as it is strictly linked with age, occupation, state of health, etc. Moreover, it could differ significantly during weekends and holidays. However, when averaged over the general population, it is relatively constant. It still depends on climate, being usually higher in cold climate countries. As mentioned above, most authorities for a first approximation assume an occupancy factor of 0.8, being made up of about 0.6 at home and 0.2 in other indoor situations (ICRP, 1993; UNSCEAR, 1993).

Dosimetric approach uncertainties

(a) Dose / exposure factor

As mentioned above, lung models are continuously evolving, as well our knowledge of the relevant physical and biological–physiological parameters, with an impact on the dose/exposure factor value; in particular a great uncertainty is related to the choice of target cells to be considered (ICRP, 1993; James, 1987; UNSCEAR, 1993) and of the weighting factors used to obtain effective dose from the calculated absorbed dose (Birchall and James, 1994).

Often the most recent models predict higher values than the older ones. For example the factor used by UNSCEAR in its report of 1982 (confirmed also in 1988 and, with some warning, in 1993) is about 50% lower than that one adopted by CEC in 1990. Moreover, the used model results are usually referred to adults only, whereas the dose factor for the age group from 0 to 10 years could be up to 2 times higher. The range of the dose/exposure factor values can be estimated to be within a factor of about 3 (UNSCEAR, 1988).

(b) Risk / dose factor

Radiation risk/dose factors (e.g. ICRP, 1991) generally have been obtained mainly from the epidemiological data of Hiroshima and Nagasaki survivors, whose exposure conditions were very different from those related to radon in dwellings: high dose rates for a relatively short time in contrast to chronic exposure at low dose rates, and neutron and gamma radiation in contrast to alpha radiation. These differences of exposure introduce uncertainties that are difficult to quantify.

Moreover, some uncertainties are related to estimation of the dose received by the exposed persons, and to the confounding factors, such as smoking habits and so on. Another source of uncertainty is due to the fact that not all of the

exposed persons are dead, so that the risk value, calculated on the basis of the number of cancers appeared to date, has to be extrapolated, or projected, to take into account the cancers that will appear in the future. Different risk-projection models produce different lifetime risk factors from the same data. The most recent studies show that the "relative risk" models — where the cancer risk due to radiation exposure is related to the baseline cancer rate of non exposed persons — fit the data better than the "absolute risk" ones — where there is no relation with the baseline cancer rate.

On the basis of the above considerations and of new epidemiologic and dosimetric data, the risk/dose factor adopted by the International Commission on Radiological Protection has changed from 1.3×10^{-2} Sv^{-1} (ICRP, 1978) to 5×10^{-2} Sv^{-1} (ICRP, 1991).

Epidemiological approach uncertainties

Risk factors obtained from the epidemiological studies based on occupationally exposed individuals in underground mines also have many uncertainties. Large uncertainties exist, for example, in estimated cumulated exposure to radon progeny, especially for the period ~1950–1960. Moreover, the presence inside the mines of other co-carcinogenic agents — like metal dust, diesel engine fumes, long-lived radionuclides, gamma radiation and, above all, tobacco smoking — make it more difficult to quantitatively correlate lung diseases to radon occurrence.

For epidemiological studies of the general population contemporary radon measurements in their homes or previous homes may be different from those present in the preceding decades of exposure. Some recent work on retrospective assessment of radon exposure is attempting to address this question (Samuelsson, 1992).

Another great source of uncertainty is the extrapolation of the risk from adult male miners in underground mines to the general public in dwellings. This extrapolation is not easy, due to difficulties in quantifying the effects of the differences of the two environments. Over the last decade many different values of an appropriate correction factor were estimated, ranging from 0.65 to 1.4 (for a summary see ICRP, 1993). For example, ICRP adopted at first a correction factor of 0.8 (ICRP, 1987), while now it assumes a value of unity; on the other hand, NRC assumed at first a default value of unity (NRC, 1988), while later a 0.7 value was adopted (NRC, 1991).

Some studies concerning systematic examinations of the full range of the epidemiological data (for a review see Burkart, 1989; ICRP, 1991; ICRP, 1993) give rise to risk estimates which are different within a factor ~3, attributable not only to the above reasons, but mainly to different risk-projection models

and, in case of relative risk models, to different baseline lung cancer rates that are used. The most recent studies, all using the relative risk-projection models, agree to within a factor less than 2 (ICRP, 1987, 1993; NRC, 1988).

16.7 CONCLUSIONS

We tentatively estimate that currently the overall uncertainty of the risk factor of general population due to radon exposure is probably less than a factor 3. It should be noted that the uncertainty of the radon risk factor is higher for specific groups such as smokers or non-smokers, due to a not yet well quantified synergism between radon and smoking. Uncertainties of this magnitude are not uncommon in estimates of the risk due to radiation or other causes, as chemical substances and so on, where often the uncertainties are much higher. It has to be underlined that the risk assessment is made on human data at exposure values in the range of values found in dwellings.

The results of the dosimetric and miner epidemiology approaches could be considered to be reasonably well in agreement. However, ICRP has decided, in 1993, to use only miner epidemiology approach to estimate the risk, at present. It is anticipated that the epidemiological studies on general populations, now in progress, will yield more appropriate risk estimates. To date only a small number of such general population epidemiological studies have been completed (e.g. Pershagen et al., 1992, 1994) and while no major conflict with the miner epidemiology has emerged, it is prudent to await the outcome of the ongoing studies.

Applying the risk/exposure factors discussed in this chapter to the typical average indoor radon concentrations in North American and European countries, a not insignificant fraction (typically of the order of 10%) of total lung cancers can be attributed to radon and its decay products exposure. However, it should be strongly emphasised that the majority total lung cancers are due to smoking. For example, in a country of 50 millions with a lung cancer lifetime risk of 3% (which is the value assumed by ICRP for its "reference" population), we can estimate that 3 persons per 1000, that is about 2000 each year, may die because of lung cancer due to radon exposure. It has to be underlined that these figures are not precise, and there is an associated uncertainty discussed in the previous sections.

Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 17

Application of Risk Assessment II: Respiratory Health Effects of Environmental Tobacco Smoke[1]

17.1 INTRODUCTION

Tobacco smoking has long been recognized (U.S. DEHEW, 1964) as a major cause of mortality and morbidity, responsible for an estimated 434,000 deaths per year in the United States (CDC, 1991a). Tobacco use is known to cause cancer at various sites, in particular the lung (U.S. DHHS, 1982; IARC, 1986). Smoking can also cause respiratory diseases (U.S. DHHS, 1983). In recent years there has been concern that nonsmokers may also be at risk for some of these health effects as a result of their exposure ("passive smoking") to the tobacco smoke that occurs in various environments occupied by smokers. Although this environmental tobacco smoke (ETS) is dilute compared to the mainstream smoke (MS) inhaled by active smokers, it is chemically similar, containing many of the same carcinogenic and toxic agents.

In 1986, the National Research Council (NRC) and the Surgeon General of the U.S. Public Health Service independently assessed the health effects of exposure to ETS (NRC, 1986; U.S. DHHS, 1986). Both of the 1986 reports conclude that ETS can cause lung cancer in adult nonsmokers and that children of parents who smoke have increased frequency of respiratory symptoms and acute lower respiratory tract infections, as well as evidence of reduced lung function.

More recent epidemiologic studies of the potential associations between ETS and lung cancer in nonsmoking adults and between ETS and non-cancer respiratory effects more than double the size of the database available for analysis from that of the 1986 reports. This chapter critically reviews the current database on the respiratory health effects of passive smoking, and these data are utilized to develop a hazard identification for ETS and to make

1 A part of the text of this chapter has been derived from EPA, 1992 Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders. United States Environmental Protection Agency, Office of Research and Development RD-689, EPA/600/6-90/006F.

quantitative estimates of the public health impacts of ETS for lung cancer and various other respiratory diseases.

The weight-of-evidence analysis for the lung cancer hazard identification is developed in accordance with U.S. EPA's Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1986a) and established principles for evaluating epidemiologic studies. The analysis considers animal bioassays and genotoxicity studies, as well as biological measurements of human uptake of tobacco smoke components and epidemiologic data on active and passive smoking. The availability of abundant and consistent human data, and especially human data at actual environmental levels of exposure to the specific agent (mixture) of concern, allow a hazard identification to be made with a high degree of certainty. The conclusive evidence of the dose-related lung carcinogenicity of mainstream smoke (MS) in active smokers, coupled with information of the chemical similarities of MS and ETS and evidence of ETS uptake in nonsmokers, is sufficient by itself to establish ETS as a known human lung carcinogen, or "Group A" carcinogen. In addition, this paper concludes that the overall results of 30 epidemiologic studies on lung cancer and passive smoking, using spousal smoking as a surrogate of ETS exposure for female never-smokers, similarly justify a Group A classification.

The weight-of-evidence analyses for non-cancer respiratory effects are based primarily on a review of epidemiologic studies. Most of the endpoints examined are respiratory disorders in children, where parental smoking is used as a surrogate of ETS exposure. For the non-cancer respiratory effects in nonsmoking adults, most studies used spousal smoking as an exposure surrogate. A causal association is concluded to exist for a number of respiratory disorders where there is sufficient consistent evidence for a biologically-plausible association with ETS that cannot be explained by bias, confounding, or chance. The fact that the database consists of human evidence from actual environmental exposure levels gives a high degree of confidence in this conclusion. Where there is suggestive but inconclusive evidence of causality, as is the case for asthma induction in children, ETS is concluded to be a risk factor for that endpoint. Where data are inconsistent or inadequate for evaluation of an association, as for acute upper respiratory tract infections and acute middle ear infections in children, no conclusions are drawn.

This paper has also attempted to provide estimates of the extent of the public health impact, where appropriate, in terms of numbers of ETS-attributable cases in nonsmoking subpopulations. Unlike for qualitative hazard identification assessments where information from many sources adds to the confidence in a weight-of-evidence conclusion, for quantitative risk assessments the usefulness of studies usually depends on how closely the study population resembles nonsmoking segments of the general population. For

lung cancer estimates among U.S. nonsmokers, the substantial epidemiology database of ETS and lung cancer among U.S. female never-smokers is considered to provide the most appropriate information. From the large number of similarly designed studies, pooled relative risk estimates have been calculated and used in the derivation of the population risk estimates. The large number of studies available, the generally consistent results, and the condition of actual environmental levels of exposure increase the confidence in these estimates. Even with these conditions, however, uncertainties remain, such as in the use of questionnaires and current biomarker measurements to estimate past exposure, assumptions of exposure–response linearity, and extrapolation to male never-smokers and to exsmokers. Still, given the strength of the evidence for the lung carcinogenicity of tobacco smoke and the extensive human database from actual environmental exposure levels, fewer assumptions are necessary than is usual in U.S. EPA quantitative risk assessments and confidence in these estimates is rated medium to high.

Population estimates of ETS health impacts are also made for certain non-cancer respiratory endpoints in children, specifically lower respiratory tract infections (LRIs, i.e. pneumonia, bronchitis, and bronchiolitis) and episodes and severity of attacks of asthma. Estimates of ETS-attributable cases of LRI in infants and young children are thought to have a high degree of confidence because of the consistent study findings and the appropriateness of parental smoking as a surrogate measure of exposure in very young children. Estimates of the number of asthmatic children whose condition is aggravated by exposure to ETS are less certain than those for LRIs because of different measures of outcome in various studies and because of increased extraparental exposure to ETS in older children. Estimates of the number of new cases of asthma in previously asymptomatic children also have less confidence because at this time the weight-of-evidence for asthma induction, while suggestive of a causal association, is not conclusive.

Most of the ETS population impact estimates are presented in terms of ranges, which are thought to reflect assumptions about the estimates of parameters and variables required for the extrapolation models. The validity of the ranges is also dependent on the appropriateness of the extrapolation models themselves.

While this chapter focuses only on the respiratory health effects of passive smoking, there may also be other health effects of concern. Recent analyses of more than a dozen epidemiology and toxicology studies (Steenland, 1992; NIOSH, 1991) suggest that ETS exposure may be a risk factor for cardiovascular disease. In addition, there are a few studies in the literature linking ETS exposure to cancers of other sites; at this time, that database appears inadequate for any conclusion.

17.2 PRIMARY FINDINGS

A. Lung cancer in nonsmoking adults

1. Passive smoking is causally associated with lung cancer in adults, and ETS, by the total weight-of-evidence, belongs in the category of compounds classified by EPA as Group A (known human) carcinogens.
2. An estimated range of 2500–3300 lung cancer deaths per year among nonsmokers (never-smokers and former smokers) of both sexes are attributable to ETS in the United States. The confidence in this range is medium to high with approximately 3000 annual lung cancer deaths representing the best estimate (about 1×10^{-5}/h on the whole population).

B. Non-cancer respiratory diseases and disorders

1. Exposure of children to ETS from parental smoking is causally associated with:
   (a) increased prevalence of respiratory symptoms of irritation (cough, sputum, and wheeze),
   (b) increased prevalence of middle ear effusion (a sign of middle ear disease), and
   (c) a small but statistically significant reduction in lung function as tested by objective measures of lung capacity.
2. ETS exposure of young children and particularly infants from parental (and especially mother's) smoking is causally associated with an increased risk of lower respiratory tract infections (pneumonia, bronchitis, and bronchiolitis). It is estimated that exposure to ETS contributes 150,000 to 300,000 lower respiratory tract infections annually in infants and children less than 18 months of age, resulting in 7500 to 15,000 hospitalizations in the United States. These higher risks continue at a decreasing rate for children until about age 3, but no estimates are derived for children over 18 months.
3. (a) Exposure to ETS is causally associated with additional episodes and increased severity of asthma in children who already have the disease. It is estimated that ETS exposure exacerbates symptoms in approximately 20% of the USA's 2 million to 5 million asthmatic children and is a major aggravating factor in approximately 10%.
   (b) In addition, the epidemiologic evidence is suggestive but not conclusive that ETS exposure increases the number of new cases of asthma in children who have not previously exhibited symptoms. Based on this evidence and the known ETS effects on both the immune system and lungs (e.g. atopy and airway hyperresponsiveness), ETS is a risk factor for the induction of

asthma in previously asymptomatic children. Data suggest that relatively high levels of exposure are required to induce new cases of asthma in children. It is estimated that previously asymptomatic children exposed to ETS from mothers who smoke at least 10 cigarettes per day will exhibit a probable range of 8000 to 26,000 new cases of asthma annually. The confidence in this range is medium and is dependent on the conclusion that ETS is a risk factor for asthma induction.

4. Passive smoking has subtle but significant effects on the respiratory health of nonsmoking adults, including coughing, phlegm, chest discomfort, and reduced lung function.

It has also examined the relationship of maternal smoking to sudden infant death syndrome (SIDS), which is thought to involve some unknown respiratory pathogenesis. While there is strong evidence that infants whose mothers smoke are at an increased risk of dying from SIDS, available studies do not allow us to differentiate whether and to what extent this increase is related to *in utero* versus post-natal exposure to tobacco smoke products. Consequently, at this time we are unable to assert whether or not ETS exposure by itself is a risk factor for SIDS independent of smoking during pregnancy. Post-natal exposure may potentiate effects of *in utero* tobacco smoke exposure, or it may not have any additional effect.

Regarding an association of parental smoking with either upper respiratory tract infections (colds and sore throats) or acute middle ear infections in children, the evidence is inconclusive.

17.3 ETS AND LUNG CANCER

17.3.1 Hazard identification

The Surgeon General (U.S. DHHS, 1989) estimated that smoking was responsible for more than one of every six deaths in the United States and that it accounted for about 90% of the lung cancer deaths in males and about 80% in females in 1985. Smokers, however, are not the only ones exposed to tobacco smoke. The sidestream smoke (SS) emitted from a smouldering cigarette between puffs (the main component of ETS) has been documented to contain many of the same carcinogenic compounds (known and suspected human and animal carcinogens) that have been identified in the mainstream smoke (MS) inhaled by smokers. Exposure concentrations of these carcinogens to passive smokers are variable but much lower than for active smokers. An excess cancer risk from passive smoking, however, is biologically plausible.

Based on the firmly established causal association of lung cancer with active smoking with a dose–response relationship down to low doses, passive

smoking is considered likely to affect the lung similarly. The widespread presence of ETS in both home and workplace and its absorption by nonsmokers in the general population have been well documented by air sampling and by body measurement of biomarkers such as nicotine and cotinine. This raises the question of whether any direct evidence exists for the relationship between ETS exposure and lung cancer in the general population and what its implications may be for public health. This report addresses that there are more than 30 epidemiologic studies of effects from normally occurring environmental levels of ETS. Because there is widespread exposure and it is difficult to construct a truly unexposed subgroup of the general population, these studies compare individuals with higher ETS exposure to those with lower exposure. Typically, female never-smokers who are married to a smoker are compared with female never-smokers who are married to a nonsmoker. Some studies also consider ETS exposure of other subjects (i.e., male never-smokers and long-term former smokers of either sex) and from other sources (e.g., workplace and home exposure during childhood), but these studies are fewer and represent fewer cases, and they are generally excluded from the analysis presented here. Use of the female never-smoker studies provides the largest, most homogeneous database for analysis to determine whether an ETS effect on lung cancer is present. We assume that the results for female never-smokers are generalizable to all nonsmokers.

Given that ETS exposures are at actual environmental levels and that the comparison groups are both exposed to appreciable background (i.e., nonspousal) ETS, any excess risk for lung cancer from exposure to spousal smoke would be expected to be small. Furthermore, the risk of lung cancer is relatively low in nonsmokers, and most studies have a small sample size, resulting in a very low statistical power (probability of detecting a real effect if it exists). Besides small sample size and low incremental exposure, other problems inherent in several of the studies may also limit their ability to detect a possible effect.

Results obtained after considering all the limits strongly support a causal association between lung cancer and ETS exposure. The overall proportion of individual studies found to show an association between lung cancer and ETS exposure is unlikely to occur by chance ($p < 0.005$). Similarly, the proportion showing a statistically significant dose–response trend ($p < 10^{-9}$) is highly supportive of a causal association. Combined results by country showed statistically significant associations for Greece (Kalandidi et al., 1987), Hong Kong (Chan and Fung, 1982; Koo, 1988; T.H. Lam et al., 1987; W.K. Lam, 1985), Japan (Akiba et al., 1986; Hirayama, 1984; Inoue and Hirayama, 1988; Shimizu et al., 1988; Sobue et al., 1990), and the United States (Brown and Chu, 1987; Buffler et al., 1984; Correa et al., 1984; Fontham et al., 1991; Garfinkel and

Silverberg, 1991; Humble et al., 1985; Janerich et al., 1990; Kabat and Wynder, 1984; Wu et al., 1985), and in that order of strength of relative risk. Pooled results of the four Western European studies (Hole et al., 1989; Pershagen et al., 1987) actually showed a slightly stronger association than that of the United States, but it was not statistically significant, probably due to the smaller sample size. The combined results of the Chinese studies (Gao et al., 1987; Geng et al., 1988; Liu et al., 1991; Wu-Williams and Samet, 1990) do not show an association between ETS and lung cancer, however, two of the four Chinese studies were designed mainly to determine the lung cancer effects of high levels of other indoor air pollutants indigenous to those areas, which would obscure a smaller ETS effect. These two Chinese studies do, however, provide very strong evidence on the lung carcinogenicity of these other indoor air pollutants, which contain many of the same components as ETS. When results are combined only for the other two Chinese studies, they demonstrate a statistically significant association for ETS and lung cancer.

The relative risks for Greece and Japan of 2.00 and 1.44, respectively, are probably the best estimates, because both female smoking prevalence and non-tobacco-related lung cancer risks, which tend to dilute the estimates of ETS effects, are low in these two countries. Also, for the time period for which ETS exposure was of interest, spousal smoking is considered to be a better surrogate for ETS exposure in these societies than in Western countries, where other sources of ETS exposure (work, public places, and other non-home environments) are generally higher.

Based on these analyses and following the U.S. EPA *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 1986a), EPA concludes that environmental tobacco smoke is a Group A (known human) carcinogen. This conclusion is based on a total weight-of-evidence, principally (U.S. EPA, 1992):

- Biological plausibility. ETS is taken up by the lungs, and components are distributed throughout the body. The presence of the same carcinogens in ETS in mainstream smoke, along with the established causal relationship between lung cancer and active smoking with the dose–response relationships exhibited down to low doses, make it reasonable to conclude that ETS is also a lung carcinogen.
- Supporting evidence from animal bioassays and genotoxicity experiments. The carcinogenicity of tobacco smoke has been established in lifetime inhalation studies in the hamster, intrapulmonary implantations in the rat, and skin painting in the mouse. There are no lifetime animal inhalation studies of ETS; however, the carcinogenicity of ETS condensates has been demonstrated in intrapulmonary implantations and skin painting experiments. Positive results of genotoxicity testing for both MS and ETS provide corroborative evidence for their carcinogenic potential.

- Consistency of response. All 4 of the cohort studies and 20 of the 26 case-control studies observed a higher risk of lung cancer among the female never-smokers classified as exposed to ETS. On the 17 studies judged to be of higher utility based on study design, execution, and analysis, 15 observed higher risks, and 6 of these increases were statistically significant, despite most having low statistical power. Evaluation of the total study evidence from several perspectives leads to the conclusion that the observed association between ETS exposure and increased lung cancer occurrence is not attributable to chance.
- Broad-based evidence. These 26 case-control and 4 prospective studies provide data from 8 different countries, employ a wide variety of study designs and protocols, and are conducted by many different research teams. Results from all countries, with the possible exception of two areas of China where high levels of other indoor air lung carcinogens were present, show small to modest increases in lung cancer associated with spousal ETS exposure. No alternative explanatory variables for the observed association between ETS and lung cancer have been indicated that would be broadly applicable across studies.
- Upward trend in dose–response. Both the largest of the cohort studies, the Japanese study of Hirayama — 200 lung cancer cases, and the largest of the case-control studies, the U.S. study by Fontham and associates (1991) — 420 lung cancer cases and two sets of controls, demonstrate a strong dose-related statistical association between passive smoking and lung cancer. This upward trend is well supported by the preponderance of epidemiology studies. Of the total of 17 studies in which data are classified by exposure level, 11 were statistically significant for the trend despite most having low statistical power.
- Detectable association at environmental exposure levels. Within the population of married women who are lifelong nonsmokers, the excess lung cancer risk from exposure to their smoking husbands' ETS is large enough to be observed.
- Carcinogenic responses are usually detectable only in high- exposure circumstances, such as occupational settings, or in experimental animals receiving very high doses. In addition, effects are harder to observe when there is substantial background exposure in the comparison groups, as is the case here.
- Effects remain after adjustment for potential bias. Current and ex-smokers may be misreported as never-smokers, thus inflating the apparent cancer risk for ETS exposure. The evidence remains statistically significant and conclusive, however, after adjustments for smoker misclassification. For the USA, the summary estimate of relative risk from nine

case-control plus two cohort studies is 1.19 (90% confidence interval (C.I.) = 1.04–1.35) after adjustment for misclassification ($p < 0.05$). For Greece, 2.00 (1.42,2.83), Hong Kong 1.61 (1.25,2.06) and Japan, 1.44 (1.13,1.85), the estimated relative risks are higher than those of the United States and more highly significant after adjusting for the potential bias.

- Confounding cannot explain the association. The broad-based evidence for an association found by independent investigators across several countries, as well as the positive dose–response trends observed in most of the studies that analyzed for them, make any single confounder highly unlikely as an explanation for the results. The examination of potential confounding factors (history of lung disease, home heat sources, diet, occupation) leads to conclude that none of these factors could account for the observed association between lung cancer and ETS.

17.3.2 Estimation of population risk

The individual risk of lung cancer from exposure to ETS does not have to be very large to translate into a significant health hazard to the population because of the large number of smokers and the widespread presence of ETS. Current smokers comprise approximately 26% of the U.S. adult population and consume more than one-half trillion cigarettes annually (1.5 packs per day, on average), causing nearly universal exposure to at least some ETS. As a biomarker of tobacco smoke uptake, cotinine, a metabolite of the tobacco-specific compound nicotine, is detectable in the blood, saliva, and urine of persons recently exposed to tobacco smoke. Cotinine has typically been detected in 50–75% of reported nonsmokers tested (50% equates to 63 million U.S. nonsmokers aged 18 or above).

The best estimate of approximately 3000 lung cancer deaths per year in U.S. nonsmokers age 35 and over attributable to ETS is based on data pooled from all 11 U.S. epidemiologic studies of never-smoking women married to smoking spouses. Use of U.S. studies should increase the confidence in these estimates. Some mathematical modelling is required to adjust for expected bias from misclassification of smoking status and to account for ETS exposure from sources other than spousal smoking. Assumptions are also needed to relate responses in female never-smokers to those in male never-smokers and ex-smokers of both sexes, and to estimate the proportion of the nonsmoking population exposed to various levels of ETS. Overall, however, the assumptions necessary for estimating risk add far less uncertainty than other quantitative risk assessments. This is because for ETS the extrapolation is based on a large database of human studies, all at levels actually expected to be encountered by much of the population.

The components of the 3000 lung cancer deaths figure include approximately 1500 female never-smokers, 500 male never-smokers, and 1000 former smokers of both sexes. More females are estimated to be affected because there are more female than male nonsmokers. These component estimates have varying degrees of confidence; the estimate of 1500 deaths for female never-smokers has the highest confidence because of the extensive database. The estimate of 500 for male never-smokers is less certain because it is based on the female never-smoker response and is thought to be low because males are generally subject to higher background ETS exposures than females. Adjustment for this higher background exposure would lead to higher risk estimates. The estimate of 1000 lung cancer deaths for former smokers of both sexes is considered to have the lowest confidence, and the assumptions included are thought to make this estimate low as well.

Workplace ETS levels are generally comparable to home ETS levels, and studies using body cotinine measures as biomarkers demonstrate that non-home exposures to ETS are often greater than exposure from spousal smoking. Thus, an alternative breakdown can be presented of the estimated 3000 ETS-attributable lung cancer deaths between spousal and non-home exposures. By extension of the results from spousal smoking studies, coupled with biological measurements of exposure, more lung cancer deaths are estimated to be attributable to ETS from combined non-home exposures — 2200 of both sexes — than from spousal exposure — 800 of both sexes. This home-versus-other-sources partitioning depends on current exposure estimates that may or may not be applicable to the exposure period of interest. Thus, this breakdown contains this element of uncertainty in addition to those discussed above with respect to the previous breakdown.

An alternative analysis, based on the large Fontham et al. (1991) study, which is the only study that provides biomarker estimates of both relative risk and ETS exposure, yields population risk point estimates of 2700 and 3600. These population risk estimates are highly consistent with the estimate of 3000 based on the combined U.S. studies.

While there is statistical variance around all of the parameters used in the quantitative assessment, the two largest areas of uncertainty are probably associated with the relative risk estimate for spousal ETS exposure and the parameter estimate for the background ETS exposure adjustment. A sensitivity analysis that independently varies these two estimates yields population risk estimates as low as 400 and as high as 7000. These extremes, however, are considered unlikely; the more probable range is narrower, and the generally conservative assumptions employed suggest that the actual population risk number may be greater than 3000. Overall, considering the multitude, consistency, and quality of all these studies, the weight-of-evidence conclusion

that ETS is a known human lung carcinogen, and the limited amount of extrapolation necessary, the confidence in the estimate of approximately 3000 lung cancer deaths is medium to high.

17.4 ETS AND NON-CANCER RESPIRATORY DISORDERS

Exposure to ETS from parental smoking has been previously linked with increased respiratory disorders in children, particularly in infants. Several studies have confirmed the exposure and uptake of ETS in children by assaying saliva, serum, or urine for cotinine. These cotinine concentrations were highly correlated with smoking (especially by the mother) in the child's presence. Nine million to twelve million American children under 5 years of age, or one-half to two-thirds of all children in this age group, may be exposed to cigarette smoke in the home (American Academy of Pediatrics, 1986).

With regard to the non-cancer respiratory effects of passive smoking, epidemiologic evidence appearing since the two major reports of 1986 (NRC and U.S. DHHS), bears on the potential association of parental smoking with detrimental respiratory effects in their children. These effects include symptoms of respiratory irritation (cough, sputum, or wheeze); acute diseases of the lower respiratory tract (pneumonia, bronchitis, and bronchiolitis); acute middle ear infections and indications of chronic middle ear infections (predominantly middle ear effusion); reduced lung function (from forced expiratory volume and flow-rate measurements); incidence and prevalence of asthma and exacerbation of symptoms in asthmatics; and acute upper respiratory tract infections (colds and sore throats). The more than 50 recently published studies reviewed in the 1992 EPA's report (U.S. EPA, 1992) essentially corroborate the previous conclusions of the NRC and Surgeon General regarding respiratory symptoms, respiratory illnesses, and pulmonary function, and they strengthen support for those conclusions by the additional weight-of-evidence. For example, new data on middle ear effusion strengthen previous evidence to warrant the stronger conclusion of a causal association with parental smoking (Willatt, 1986; Fleming et al., 1987; Tainio et al., 1988; Reed and Lutz, 1988; Hinton, 1989; Teele et al., 1989; Corbo et al., 1989; Strachan et al., 1989; Takasaka, 1990; Etzel et al., 1992).

Furthermore, recent studies establish associations between parental smoking and increased incidence of childhood asthma (Burchfield et al., 1986; D. Evans et al., 1987; O'Connor et al., 1987; Murray and Morrison, 1989; Kryzanowski et al., 1990; Sherman et al., 1990; Weitzman et al., 1990; Oldigs et al., 1991; Martinez et al., 1992; Ehrlich et al., 1992). Additional research also supports the hypotheses that in utero exposure to mother's smoke and postnatal exposure to ETS alter lung function and structure, increase bronchial responsiveness,

and enhance the process of allergic sensitization, changes that are known to predispose children to early respiratory illness. Early respiratory illness can lead to long-term pulmonary effects (reduced lung function and increased risk of chronic obstructive lung disease).

It also deserves a discussion of the evidence for an association between parental smoking and sudden infant death syndrome (SIDS), which was not addressed in the 1986 NRC or Surgeon General reports. SIDS is the most common cause of death in infants ages 1 month to 1 year. The cause (or causes) of SIDS is unknown; however, it is widely believed that some form of respiratory pathogenesis is generally involved. The current evidence strongly suggests that infants whose mothers smoke are at an increased risk of dying of SIDS; independent of other known risk factors for SIDS, including low birth weight and low gestational age, which are specifically associated with active smoking during pregnancy. However, available studies do not allow us to conclude whether that increased risk is related to in utero versus postnatal exposure to tobacco smoke products, or to both (Steele and Langworth, 1966; Naeye et al., 1976; Bergman and Wiesner, 1976; Lewak et al., 1979; Malloy et al., 1988; Hoffman et al., 1988; Haglund and Cnattingius, 1990; Mitchell et al., 1991).

The 1986 NRC and Surgeon General reports conclude that both prevalence of respiratory symptoms of irritation and the incidence of lower respiratory tract infections are higher in children of smoking parents. In the 18 studies of respiratory symptoms subsequent to the 2 reports (U.S. EPA, 1992), increased symptoms (cough, phlegm, and wheezing) were observed in a range of ages from birth to mid-teens, particularly in infants and preschool children. In addition to the studies on symptoms of respiratory irritation, nine new studies have addressed the topic of parental smoking and acute lower respiratory tract illness in children, and eight have reported statistically significant associations. The cumulative evidence indicates strongly that parental smoking, especially the mother's, causes an increased incidence of respiratory illnesses from birth up to the first 18 months to 3 years of life, particularly for bronchitis, bronchiolitis, and pneumonia. Overall, the evidence confirms the previous conclusions of the NRC and Surgeon General.

Recent studies also solidify the evidence for the conclusion of a causal association between parental smoking and increased middle ear effusion in young children. Middle ear effusion is the most common reason for hospitalization of young children for an operation.

At the time of the Surgeon General's report on passive smoking (U.S. DHHS, 1986) data were sufficient only to conclude that maternal smoking may influence the severity of asthma in children. Recent studies strengthen and confirm these exacerbation effects. In addition, the new evidence is conclusive that ETS exposure increases the number of episodes of asthma in children who

already have the disease. It is also suggestive that ETS exposure increases the number of new cases of asthma in children who have not previously exhibited symptoms, although the results are statistically significant only with children whose mothers smoke 10 or more cigarettes per day. While the evidence for new cases of asthma itself is not conclusive of a causal association, the consistent strong associations of ETS with both increased frequency and severity of the asthmatic symptoms and the established ETS effects on both the immune system and airway hyperresponsiveness lead to the conclusion that ETS is a risk factor for induction of asthma in previously asymptomatic children.

Regarding the effects of passive smoking on lung function in children, the 1986 Surgeon General and NRC reports both conclude that children of parents who smoke have small decreases in tests of pulmonary output function of both the larger and smaller air passage when compared with the children of nonsmokers. As noted in the NRC report, if ETS exposure is the cause of the observed decrease in lung function, the effect could be due to the direct action of agents in ETS or an indirect consequence of increased occurrence of acute respiratory illness related to ETS.

Results from eight studies on ETS and lung function in children that have appeared since those reports add some additional confirmatory evidence suggesting a causal rather than an indirect relationship (O'Connor et al., 1987; Lebowitz et al., 1987; Tsimoyianis et al., 1987; Kauffmann et al., 1989b; Chan et al., 1989b; Dijkstra et al., 1990; Strachan et al., 1990; Martinez et al., 1992). For the population as a whole, the reductions are small relative to the interindividual variability of each lung function parameter. However, groups of particularly susceptible or heavily exposed subjects have shown larger decrements. The more recent studies suggest that a continuum of exposures to tobacco products starting in fetal life may contribute to the decrements in lung function found in older children. Exposure to tobacco smoke products inhaled by the mother during pregnancy may contribute significantly to these changes, but there is strong evidence indicating that postnatal exposure to ETS in an important part of the causal pathway.

With respect to lung function effects in adults exposed to ETS, the 1986 NRC and Surgeon General reports found the data at that time inconclusive, due to high interindividual variability and the existence of a large number of other risk factors, but compatible with subtle deficits in lung function. Recent studies confirm the association of passive smoking with small reductions in lung function. Furthermore, new evidence also has emerged suggesting a subtle association between exposure to ETS and increased respiratory symptoms in adults (Svendsen et al., 1987; Kalandidi et al., 1987; Masi et al., 1988; Kauffmann et al., 1989a; Hole et al., 1989; Schwartz and Zeger, 1990).

There is some evidence suggesting that the incidence of acute upper respi-

ratory tract illnesses and acute middle ear infections may be more common in children exposed to ETS. However, several studies failed to find any effect. In addition, the possible role of confounding factors, the lack of studies showing clear dose–response relationships, and the absence of a plausible biological mechanism preclude more definitive conclusions.

In reviewing the available evidence indicating an association (or lack thereof) between ETS exposure and the different non-cancer respiratory disorders, the possible role of several potential confounding factors was considered. These include other indoor air pollutants, socioeconomic status, effect of parental symptoms, and characteristics of the exposed child, such as low birth weight or active smoking. No single or combined confounding factors can explain the observed respiratory effects of passive smoking in children.

For diseases for which ETS has been either causally associated (lower respiratory tract infections) or indicated as a risk factor (asthma cases in previously asymptomatic children), estimates of population attributable risk can be calculated. A population risk assessment in USA provides a probable range of estimates that 8000 to 26,000 cases of childhood asthma per year are attributable to ETS exposure from mother who smoke 10 or more cigarettes per day. The confidence in this range of estimates is medium and is dependent on the suggestive evidence of the database. While the data show an effect only for children of these heavily smoking mothers, additional cases due to lesser ETS exposure are also a possibility. If the effect of this lesser exposure is considered, the range of estimates of new cases presented above increase to 13,000 to 60,000. Furthermore, the additional public health impact of ETS on asthmatic children *may* include over 200,000 children whose symptoms are significantly aggravated and as many as 1,000,000 children who are affected to some degree.

In the USA it is estimated that ETS exposure contributes 150,000 to 300,000 cases annually of lower respiratory tract illness in infants and children younger than 18 months of age and that 7500 to 15,000 of these will require hospitalization. The strong evidence linking ETS exposure to increased incidence of bronchitis, bronchiolitis, and pneumonia in young children gives these estimates a high degree of confidence. There is also evidence suggesting a smaller ETS effect on children between ages 18 months and 3 years.

In the USA, more than 5000 infants die of SIDS annually. It is the major cause of death in infants between the ages of 1 month and 1 year and the linkage with maternal smoking is well established. The Surgeon General and World Health Organization estimate that more than 700 U.S. infant deaths per year from SIDS are attributable to maternal smoking (U.S. CDC, 1991a). However, at present there is not enough direct evidence supporting the contribution of ETS exposure to declare it a risk factor or to estimate its population impact on SIDS.

Indoor Air Quality
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Chapter 18

Application of Risk Assessment III: Carpets[1]

Increased use of carpeting as a flooring material in homes and in commercial and public buildings, combined with changes in methods of installation have increased the number of complaints about adverse effects on comfort and health associated with the use of carpet. Over a period of a few decades there has been a rapid increase in the percentage of the total amount of flooring using carpets in many European countries as well as in the United States. In the following, the factors determining these risks during three phases of the useful life of carpets is discussed and the application of risk assessment methods to evaluate exposures to volatile organics off-gassing from new carpets is presented.

18.1 RISKS ASSOCIATED WITH THE USE OF CARPETS

In the course of the manufacture and lifetime of a carpet, different phases can be distinguished:

Phase 1 consists of the manufacture, storage and shipping of the carpets and includes the choice of raw materials, the manufacturing process itself and any controls and surveillance placed on these factors prior to delivery at an installation site. From an exposure and risk assessment point of view, the focus is on workers and the workplace environment.

Phase 2 consists of the installation of carpets and the period of initial off-gassing from the carpets themselves and from adhesives or other materials used in the installation. This phase ends in a quasi-steady state of off-gassing after a period of the order of one week to one month. Emissions during this phase decline from a high level during the actual installation to a lower level.

Phase 3 covers the risk factors which dominate during the remainder of the useful life of a carpet installation. The primary emission of contaminants from the carpet and its associated materials will be low, but there will be an

1 A part of the text of this chapter has been derived from R.G. Hetes, D.S. Womack, T.K. Pierson and D.F. Naugle "Evaluation of Exposures to Volatile Organics Offgassing from New Carpets", 1992. Ed. M. Berry, RTI Report Number 4479-001/12-F.

increasing emission of odorant substances, e.g. from maintenance procedures, as well as biologically active and allergenic materials which have developed or have been accumulated in the carpet.

For each of these phases the carpet related factors which help contribute to the risk potential are somewhat different, and they are embedded in the total of environmental factors which determine the overall risk of carpet-associated adverse effects on perception, comfort and health. A number of factors modify the risk associated with carpets in any particular situation. These simultaneously operating factors include the effective ventilation rate, the rate of occupancy, the type and intensity of the maintenance efforts and the materials used in maintenance, among others. The following risk assessment will consider the exposure and risk associated with the off-gassing of volatile organics from new carpets.

18.2 SOURCE CHARACTERIZATION

Chamber studies were conducted on 19 different carpet products. These chamber studies were used to identify off-gassing volatile organics and to estimate emission rates for major identified constituents and for total volatile organic compounds (TVOC). The carpets tested are generally representative of the types of products available in the United States.

18.2.1 Identification of off-gassing volatile organic compounds

A total of 69 chemical compounds were identified in the emissions from these 19 products. Emission rates were estimated for these compounds. The number of compounds found in individual product samples ranged from 2 to 23 with an average of 13.6 compounds per sample. Thirty-five of the 69 compounds were observed in only a single carpet product. Styrene and 4-phenylcyclohexene (4-PCH) were observed in all 19 carpet products. Compounds in the majority of samples include 4-vinylcyclohexene (4-VCH) (16 products), undecane (13), propylbenzene (12), and decane (11).

18.2.2 Emission rate and decay constant estimation

Emission rates and decay constants for individual constituents were estimated by Black (1991) based on data from the chamber studies. These studies provided constituent concentrations in the chambers after 1 h, 24 h, and approximately 140 h. Average emission rates for each constituents were calculated for 1 h, 24 h, and approximately 140 h, respectively, for all products

TABLE 18.1

Composition of "composite carpet"

Compound	CAS Number
Styrene	100-42-5
4-Phenylcyclohexene (4-PCH)	4994-16-5
4-Vinylcyclohexene (4-VCH)	100-40-3
Undecane	1120-21-4
Propylbenzene	103-65-1
Decane	124-18-5
Ethylbenzene	100-41-4
2-Butoxyethanol	111-76-2
Cumene (isopropyl benzene)	98-82-5
m-Ethyltoluene	620-14-4
Toluene	108-88-3
p-Xylene	1330-20-7

tested. For compounds identified in a sample but below the quantitation limit, Black provided no emission rates. Thus, an emission rate was estimated based on a concentration equal to one-half of the quantitation limit (QL/2). The quantitation limit for these studies was reported to be 1 $\mu g/m^3$ (Black, 1991). Therefore, it was assumed that a constituent concentration present below the quantitation limit was equal to 0.5 $\mu g/m^3$. The corresponding emission rate to be used in the analyses for this set of compounds was estimated to be 0.0006 ($mg/m^2/h$).

A mean emission rate (from $n = 19$ carpet products) was calculated for each constituent at 1, 24, and 140 h. The method of least squares (log mean emission rate vs. time) was then used to estimate decay constant for each constituent. The average emission rate and estimated decay rates were used as inputs to the exposure modelling as discussed below.

18.2.3 Selection of "composite" carpets

To simplify the analysis and to make it generalizable across many carpet products, a "composite" carpet was developed based on the most frequently encountered compounds. The use of a composite carpet allows for a reasonable assessment of the health effects associated with the compounds occurring most frequently in emissions from carpets. The 12 most commonly observed compounds were selected and are identified in Table 18.1.

18.2.4 Selection of worst-case carpets

The use of a composite carpet for analysis represents an "average" carpet and "average" emissions from carpets. However, there is a high degree of variability between carpets in the number of compounds emitted and the rates at which they are emitted. Therefore, analyses were also conducted using worst case emissions data. To illustrate such analyses, two approaches were pursued; the use of a worst case composite carpet and the use of the two highest emitting carpet products. A worst case composite carpet assumes that each of the twelve compounds is present with the highest emission rate observed in any of the 19 tested products. Two individual carpet products were also chosen to represent a more realistic worst case. One of the products (9) was chosen as it was the highest emitter of styrene (nearly twice that of the next highest), and emitted 11 of the 12 compounds. The other product (19) was selected as it was the highest emitter of a 4-PCH, 4-VCH, undecane, xylene, propylbenzene, ethylbenzene, and emitted 10 of the 12 compounds.

18.3 TOXICITY AND HEALTH EFFECT ANALYSIS

There is much variability in the type and quantity of health effects data of the volatile organics off-gassed from carpets. Some compounds (e.g., toluene) are well studied with a range of data available, while others (e.g., 4-PCH) have little or no data available. Computer data bases (e.g., MEDLARS, the National Library of Medicine computer database) were used to identify relevant toxicity data and health benchmarks. Typical health benchmarks that were investigated include Reference Concentrations (RfC), Reference Doses (RfD), Threshold Limit Values (TLV), and odour thresholds. Toxicity data identified include 50th percentile lethal dose and concentration (LD_{50} and LC_{50}) and lowest toxic (TD_{10}, TC_{10}) and/or lethal dose and concentration (LD_{10} and LC_{10}). Attempts were made to identify information on mechanisms of action, target organs, and the types of effects which have been reported in humans.

The Reference Concentrations (RfC) is the prospective health benchmark used by the EPA for exposure to non-carcinogens. The EPA defines the RfC as an estimate (with uncertainty spanning perhaps in order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime of exposure (Barnes and Dourson, 1988). The RfC is based on a single identified threshold level, the highest no observed adverse effects level (NOAEL) for a critical effect. Critical effects may be any effect associated with any target organ and may be reversible or irreversible. The designation of an

effect as adverse is left to the professional judgment of those within the EPA who establish and verify RfCs.

The RfC is determined by the use of the following equation:

$$RfC = NOAEL/(UFxMF)$$

where: NOAEL = no observed adverse effects level; UF = uncertainty factor; and MF = modifying factor.

The NOAEL, as described above, is the lowest (human equivalent) dose for which no adverse health effects have been observed. If a NOAEL is not available, the lowest observed adverse effects level (LOAEL) can be used along with an additional uncertainty factor. The uncertainty factors are intended to conservatively compensate for scientific uncertainty associated with necessary extrapolations. The magnitude of the uncertainty factor is determined by the extent of extrapolation and may range from 10 to 10,000. A factor of 10 is used for each of the following that apply: (1) interhuman variability (always applied to protect sensitive individuals within a population), species extrapolation, (2) interspecies variability, species extrapolation, (3) extrapolation from subchronic to chronic, (4) use of a LOAEL instead of a NOAEL, and (5) incompleteness of the database. The overall uncertainty factor is the product of all individual factors that apply. The modifying factor is an additional uncertainty factor which allows consideration of scientific uncertainties not explicitly treated in the uncertainty factors, such as study design or the number of animals used in the critical study. The value of the modifying factor must be greater than zero and less than or equal to 10 with a default value of 1. The actual value is assigned by professional judgment after review of the entire database.

The RfC is not a direct or absolute estimator of risk, but rather a reference point to gauge the potential effects. Doses at or below the RfC are not likely to be associated with any adverse health effects, and are of little regulatory concern. However, exceeding the RfC does not imply that an adverse health effect would necessarily occur. As the amount and frequency of exposures exceeding the RfC increases, the probability that adverse effects may be observed in the human population also increases. While doses below the RfC are all acceptable, all doses above the RfC are not categorically unsafe.

Threshold Limit Values (TLVs) are widely used in occupational settings to establish worker exposure protective levels. There are two types of TLVs, TLV-time-weighted average (TWA) and TLV-short-term exposure limit (STEL). The TLV-TWA is the time-weighted average concentration for a normal 8-h working day and 40-h working week to which nearly all workers may be exposed day after day without adverse effects. The TLV-STEL is

defined as a 15-min time-weighted average exposure that should not be exceeded at any time during the working day and should not be repeated more than four times a day (ACGIH, 1992). The TLV also assumes a 46-year working life. There are several limitations to the use of TLVs as health benchmarks for exposures in the general population. TLVs are intended to be protective for occupational exposures, in healthy workers with a recovery period. TLVs are not applicable to continuous exposures and sensitive subpopulations. They are, however, often used as a crude index of toxicity since better indices are often not available for a great number of air toxic constituents.

In assessing the human health effects associated with exposure to a chemical, the study results from actual human exposures are the most useful. Lacking good epidemiology studies, toxicology study results, preferably from a chronic study, yield the most useful information on the range of target organs affected and possible health effects. However, these data are not always available and less informative toxicity measures must be used in health assessments. They include chronic and subchronic studies designed to characterize effects that take longer to appear than those in a short-term study, and acute studies including the median lethal dose (LD_{50}) or concentration (LC_{50}) or low lethal or toxic dose or concentration (TD_{10}, TC_{10}, LD_{10}, and LC_{10}).

For the purposes of health effects analysis, RfCs are the most useful toxicity benchmark. Exposure below the RfC is assumed not to result in adverse health effects. If RfCs are not available, TLVs or chronic toxicity data such as NOAELs and LOAELs can be used. If these are not available, crude toxicity measures such as LD_{50} or LC_{50} are the last resort as indicators of relative toxicity.

Table 18.2 summarizes the available health benchmarks associated with the constituents of the composite carpet. Four of the compounds (ethylbenzene, cumene, toluene, and xylene) have RfCs that have been verified by EPA; four compounds (styrene, ethylbenzene, cumene, and toluene) have RfDs; and six (styrene, ethylbenzene, 2-butoxyethanol, cumene, toluene, and xylene) have TLVs. Very few toxicity data were available for the remaining compounds though most are believed to be of relatively low toxicity compared to the other compounds. However, it must be noted that the lack of toxicity data for compounds such as 4-PCH does not mean the chemical is not toxic, rather that the toxicity of the chemical is not well characterized.

18.4 EXPOSURE ASSESSMENT MODEL

Computer modelling was used to estimate indoor concentrations of the constituents and exposure profiles based on emission rate data for specific constituents. Several indoor air quality computer models were reviewed and

TABLE 18.2

Summary of existing data or information on "composite carpet" compounds

Compound	LC_{50} (mg/m^3)	NOAEL (mg/m^3)	LOAEL (mg/m^3)	RfC (mg/m^3)	TLV (mg/m^3)
Styrene		425 (h)		215	
4-Phenylcyclohexene		320 (r)			
4-vinylcyclohexene	27,000				
Undecane					
Propylbenzene	20,000				
Decane	72,300				
Ethylbenzene		434 (r)		1.0	435
Ethanol, 2-butoxy	3,360		480 (h) 960 (h)		120
Benzene, isopropyl (cumene)			92 (r)	0.009 (c) 0.09 (s)	245
m-Ethyltoluene					
Toluene		151 (h)		2.0	375
Xylene, para		27 (h)		0.3	435

EPA's EXPOSURE computer model was selected as the most applicable to this project. EXPOSURE, developed by the EPA's Air and Energy Engineering Research Laboratory (AEERL), was selected for the following reasons:

- can be applied to decaying emission sources (i.e., allows input of emission rate and decay constant);
- can be applied to products with an application emission rate (i.e., allows input of application emission rate and decay rate);
- can calculate exposure levels to individuals (up to 3) through the use of time-activity patterns;
- can specify varying activity pattern and breathing rate for each individual;
- can specify adsorption rates and re-emission rates; and
- can estimate sink effects and specify times when sinks (up to three) are on and off.

18.4.1 Model parameters

A residential exposure scenario was developed based on the floor plan of the EPA test house in Cary, North Carolina with the following assumptions or model parameter values:

- home has 7 rooms with carpet in all rooms excluding baths and kitchen;
- air flows in the home were defined based on EXPOSURE model test house example; alternative flows are possible but the EPA test house was selected to simplify initial modelling efforts;
- air exchange rate (ACH) was set at 0.35 which is based on actual measured data from the Cary test house (measured values ranged from 0.3 to 0.5);
- HVAC was assumed to be continuous. EXPOSURE does allow for alternative HVAC patterns with time settings for HVAC to be either on or off;
- number of consecutive days modeled was 5 (120 h). This was chosen as it represents a typical work week for which time-activity patterns could be designated, modelling of longer periods required sequential model runs. Concentration profiles were estimated for longer periods for styrene and 4-PCH;
- outdoor pollutant concentration was assumed to be zero;
- sinks in carpeted rooms were walls and ceilings and in uncarpeted rooms were walls, ceilings, and floors.

The emission rates and decay constants discussed previously were used for the residential scenarios.

18.4.2 Estimated concentration profiles

Concentration profiles were estimated for each of the 12 compounds in the composite carpet for the residential scenario. For purposes of illustration, the residential scenario concentration profile for styrene is shown in Figure 18.1. Styrene is used in this illustration as it had the highest peak concentration of any of the 12 compounds. Similar profiles were developed for all other compounds. Table 18.3 presents the peak concentrations estimated for each of the compounds, and the range of estimated concentrations over the 120-h model period. A summary concentration profile for all compounds in the test house (in the first bedroom) is presented in Figures 18.2 and 18.3.

Most of the rooms have equivalent profiles due to the fact that most rooms were carpeted. Concentrations in the bathrooms were lower but followed the same general profile due to the well mixed assumption used by EXPOSURE that the constituents are evenly distributed throughout a room. In general, peak concentrations did not occur immediately following installation but rather occurred after a delay allowing sufficient emissions to occur. Peak concentrations were estimated to occur between about 3 h and 63 h after installation. With the exception of 4-PCH, all constituents experienced peaks simultaneously, between about 9 and 12.5 h after installation. The peak for 4-PCH was somewhat delayed relative to the others starting at about 18 h

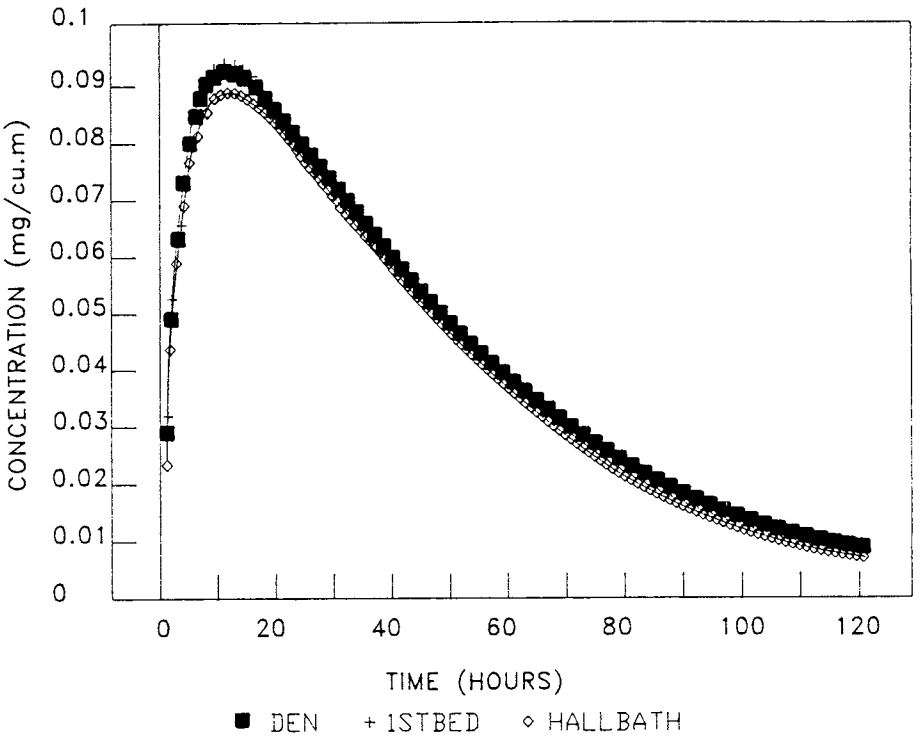

Fig. 18.1: Predicted concentration profile for styrene in test house composite carpet.

TABLE 18.3

Summary of estimated concentration peak and ranges for compounds: composite carpet

	Initial	Maximum	at 120 hours
Styrene	0.015	0.092	0.009
4-Phenylcyclohexene	0.007	0.057	0.032
4-Vinylcyclohexene	0.002	0.012	0.002
Undecane	0.001	0.004	0.001
Propylbenzene	0.001	0.006	0.001
Decane	0.001	0.005	0.001
Ethylbenzene	0.001	0.007	0.001
2-Butoxyethanol	0.007	0.004	0.006
Cumene	0.001	0.005	0.001
m-Ethyltoluene	0.000	0.002	0.001
Toluene	0.001	0.004	0.001
Xylene	0.001	0.005	0.001

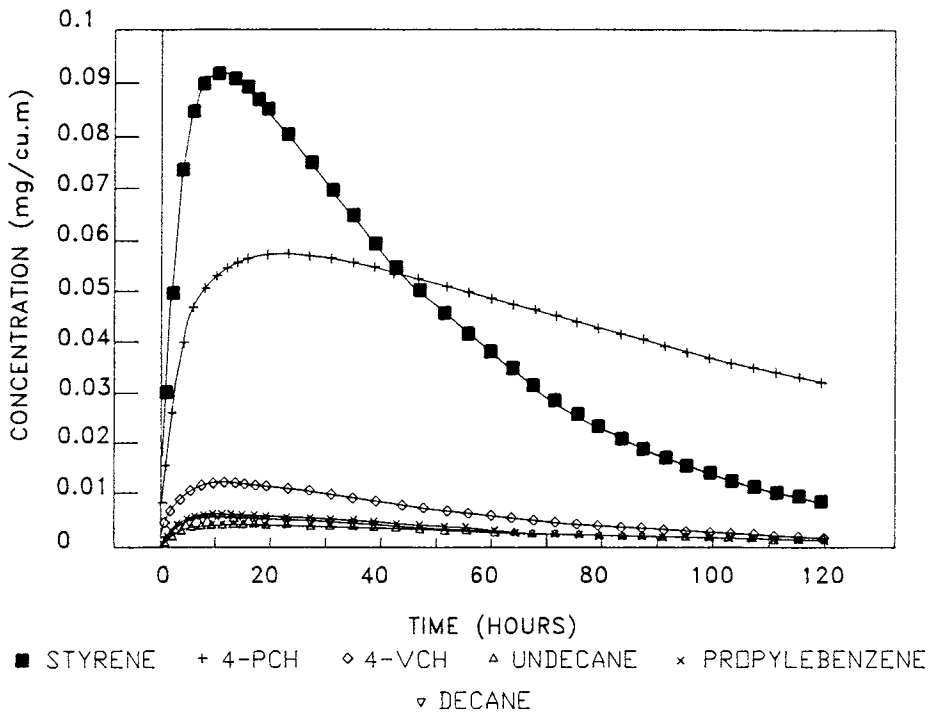

Fig. 18.2: Predicted concentration profile summary for all compounds: test house composite carpet.

after installation but still coincided with the peaks of many of the compounds. Peak concentrations from Table 18.3 were selected for initial health effects analysis.

For the composite carpet in the test house, concentrations of styrene, 4-PCH, and 2-butoxyethanol were the major contributors to the TVOC off-gassed from carpet in the indoor environment with peak concentrations of 0.092, 0.057, and 0.044 mg/m^3 respectively, and to a lesser degree 4-VCH is also a contributor with a concentration of 0.012 mg/m^3. Likewise styrene, 4-PCH, and 4-VCH were major contributors in terms of frequency, having been observed in all or most carpet products tested. The remaining compounds are not major contributors with peak concentrations ranging from 0.002 to 0.007 mg/m^3.

Concentration profiles were also estimated for the worst case composite carpet and carpet products 9 and 19. Table 18.4 presents a comparison of estimated peak concentrations for each of the four carpets analyzed: composite, worst case composite, carpet products 9 and 19. The peak concentrations presented in Table 18.4 were used in an initial health effects analysis.

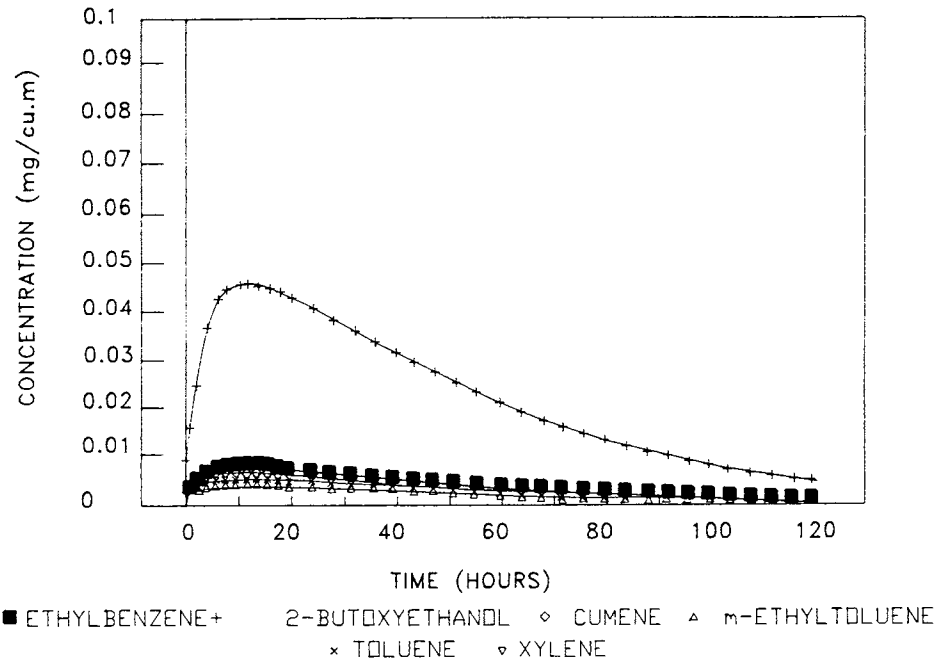

Fig. 18.3: Predicted concentration profile summary for all compounds: test house composite carpet.

TABLE 18.4

Summary of estimated peak concentrations: test house (mg/m^3)

Compound	Worst-case			
	Composite	Composite	Carpet 9	Carpet 19
Styrene	0.092	0.420	0.420	0.121
4-Phenyl	0.92	0.420	0.420	0.121
4-Phenylcyclohexene	0.057	0.123	0.038	0.123
4-Vinylcyclohexene	0.012	0.065	0.024	0.065
Undecane	0.004	0.014	0.002	0.013
Propylbenzene	0.006	0.021	0.021	0.018
Decane	0.005	0.039	0.004	—
Ethylbenzene	0.007	0.039	0.010	0.039
2-Butoxyethanol	0.044	0.326	0.006	—
Cumene	0.005	0.022	0.014	0.018
m-Ethyltoluene	0.002	0.028	—	—
Toluene	0.004	0.022	0.004	0.07
Xylene	0.005	0.030	0.012	0.030

18.4.3 Time activity patterns

Using these estimated concentration profiles in conjunction with the time activity patterns, actual exposure profiles were estimated. Standard time activity patterns were used in initial modelling runs (with 0 = 12 noon) (EPA, 1985). Three individual time activity patterns were modeled and are described below.

Adult/Home	Child/Home	Adult/Working Outside
0–5 den	0–4 den	0–7 outdoors
5–6 outdoors	4–5 outdoors	7–11 den
6–11 den	5–9 den	11–20 master bed
11–19 master bed	9–19 1st bed	20–24 outdoors
19–22 den	19–22 den	
22–23 outdoors	22–23 outdoors	
23–24 den	23–24 den	

Breaths/h and volume/breath values used in model to estimate inhalation exposure were based on reported volumetric rates (EPA, 1987b). As EXPOSURE allows only a single breathing rate to be entered into the model, the breathing rates are weighted averages for the entire day.

Adult/Home	Child/Home	Adult/Working Outside
1.1 m^3/h	1.1 m^3/h	0.95 m^3/h

18.4.4 Exposure profiles

EXPOSURE was run for each constituent using mean emission rates (ER) at h = 1 as the initial emission rate, standard time-activity patterns and inhalation parameters. Exposure profiles were generated for each constituent under each scenario and incorporates concentration and contact. For demonstration purposes, the exposure profile for styrene emitted from the composite carpet is shown in Figure 18.3. Exposure profiles closely follow the concentration profile with peak exposures occurring around 7–15 h after installation. Inhalation exposure profiles were also estimated assuming the breathing rates shown above. The exposure profiles of the adult at home and the child at home are essentially equivalent while the adult who works outside of the home is lower. The exposure profiles from the adult and child at home were essentially the same, as the child's higher respiratory rate compensated for a lower tidal volume.

18.5 RISK CHARACTERIZATION/HEALTH EFFECT ANALYSIS

18.5.1 Non-cancer health effects analysis for individual constituents

As an initial analysis, the peak concentrations for each compound were compared to relevant health benchmarks. As described above, RfCs represent the best health benchmark for analysis as exposures below the RfC are assumed to be without adverse health effects. Where possible, comparisons of peak exposure levels were made with RfCs. In the absence of RfCs other health benchmarks were used including TLVs, TCs, or LCs.

Compounds with RfCs

Four of the compounds have verified RfCs: ethylbenzene, cumene, toluene, and xylene. A graphical summary of estimated peak concentrations and relevant health benchmarks (including the RfC) for these four compounds is presented in Figure 18.4. The subchronic and chronic RfC for ethylbenzene is 1.0 mg/m^3 and is based on fetal developmental toxicity in rats. The uncertainty factor used for estimation of the RfC was 300 for both subchronic and chronic. The estimated peak concentration for ethylbenzene from the composite carpet is 0.007 mg/m^3, about 0.7% of the RfC (1.0 mg/m^3). The estimated peak concentration for ethylbenzene in the worst case composite carpet and carpet sample 19 is 0.039 mg/m^3, about 3.9% of the RfC; and peak exposure level for Carpet 9 was estimated at 0.010 mg/m^3 or 1% of the RfC.

Another common measure used in non-cancer risk assessment is called the margin of exposure (MOE) which is defined as the ratio of the NOAEL in the study species to the human exposure level. It can be interpreted as margin of safety, the degree below adverse effects level. In all cases the MOE should be greater than the uncertainty factor used in CfC derivation. For ethylbenzene emitted from the composite carpet, the MOE is 62,000, that is the NOAEL of 434 mg/m^3 is 62,000 times greater than the estimated peak concentration of 0.007 mg/m^3. This can be interpreted as peak exposure levels are 62,000 times below exposure levels thought to be indicative of adverse effects in rats (NOAEL). For the worst-case composite and Carpet 19 the MOE was 11,000 and, for Carpet 9, it was 43,000.

The RfC for cumene (isopropyl benzene) is based on a LOAEL of central nervous system (CNS) changes and nasal irritation in rats. The chronic RfC is 0.009 mg/m^3 while that for subchronic exposures is 0.09 mg/m^3 with uncertainty factors of 10,000 and 1000 respectively. Peak exposure for the composite carpet is estimated at 0.005 mg/m^3, which is below both the subchronic and

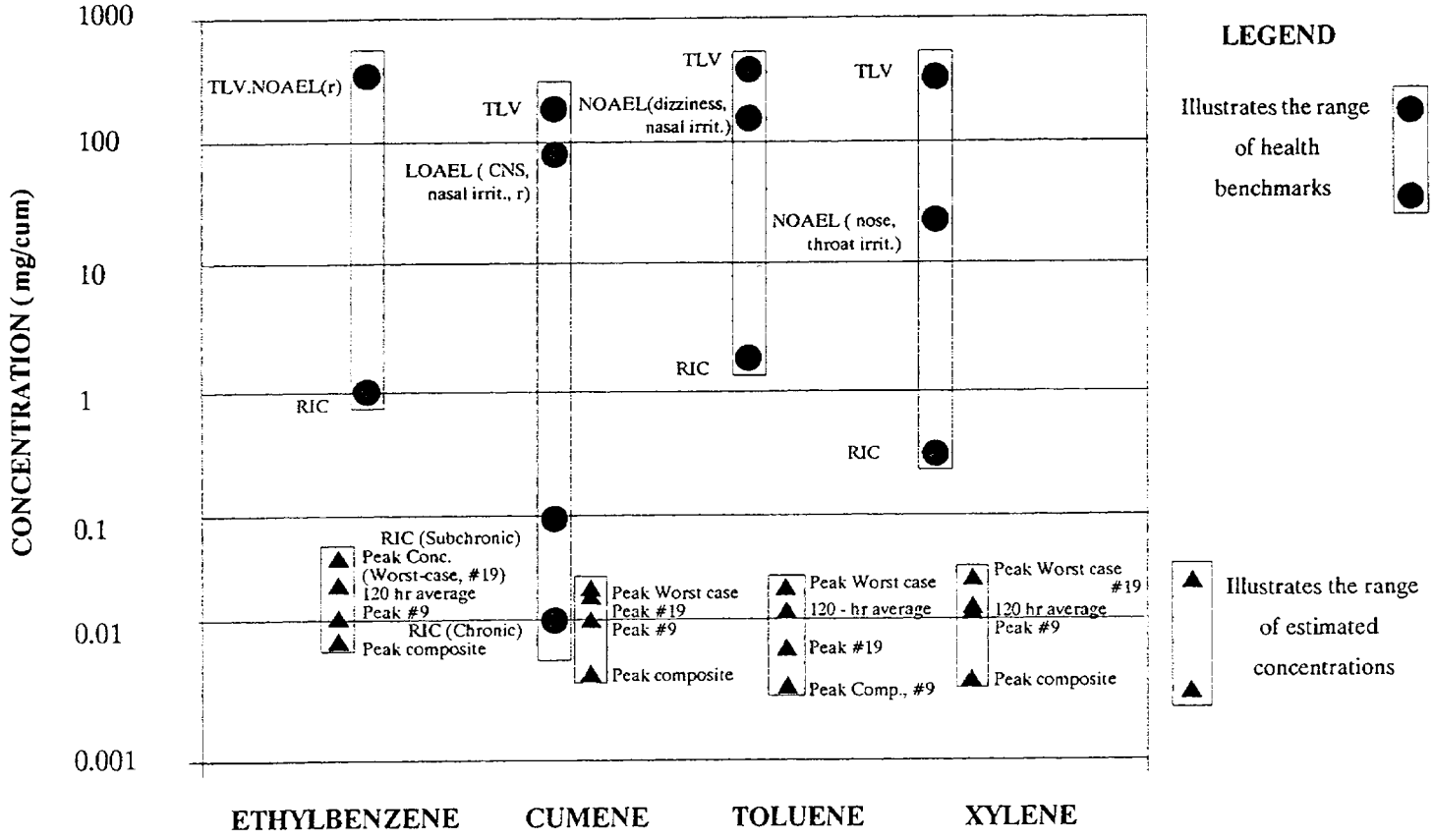

Fig. 18.4: Comparison of concentrations to relevant health benchmarks.

chronic RfC values and would not be anticipated to result in adverse effects. The chronic RfC value (0.009 mg/m^3) is exceeded for about the first 60 h after installation of carpet and, is exceeded for about the first 50 h for Carpet 19. In no case was the subchronic RfC value exceeded, as the peak concentration from the worst case composite reached 0.022 mg/m^3, or 22% of the subchronic RfC. Carpet products 9 and 19 had estimated peak concentrations of 14% and 18% of the subchronic RfC, respectively. The MOE for cumene is 18,400 for the composite carpet; 4180 for worst case composite; 6600 for Carpet 9; and 5100 for Carpet 19. Given that the subchronic RfC was never exceeded, peaks were relatively short (< 6000 h) and further decreases in concentration would occur after the peak period, it is not likely that adverse health effects would occur from exposure to cumene.

The RfC for toluene is based on CNS effects and nasal irritation in humans. The chronic and subchronic RfCs are equivalent at 2.0 mg/m^3 with an uncertainty factor of 100. The estimated peak concentration for toluene from composite carpet of 0.004 mg/m^3 is about 0.2% of the RfC indicating that adverse effects are not likely. The estimated peak concentration from worst-case composite is about 1.1% of the RfC. The NOAEL used in the RfC estimation was 151 mg/m^3 and the peak exposure from composite carpet was 0.004 mg/m^3 yielding a MOE of 37,750. The MOE was 6860 for worst-case composite, 37,000 for Carpet 9 and 22,000 for Carpet 19.

The RfC for xylene is based on CNS effects and nose and throat irritation in humans. The chronic and subchronic RfCs are equivalent at 0.3 mg/m^3 with an uncertainty factor of 100. Confidence in the RfC is medium. The estimated peak concentration for xylene of 0.005 mg/m^3 below the RfC indicating that adverse effects are not likely. Peak exposure from the composite carpet (0.005 mg/m^3) is about 1.7% of the RfC for worst-case composite and Carpet 19 about 10 percent of RfC, and for Carpet 9 about 4% of the RfC. The NOAEL used in the RfC estimation was 27 mg/m^3, and the peak exposure from the composite carpet was 0.005 mg/m^3 yielding a MOE of 5400. The MOE for the worst-case composite and Carpet 19 was 900, and 2250 for Carpet 9.

Compounds without RfCs

In the absence of an RfC, other health benchmarks indicative of thresholds for adverse health effects would be preferable including the No Observed Adverse Effects Level (NOAEL) and Lowest Observed Adverse Effects Level (LOAEL) from toxicity studies. TLV and other indices may also be used, with caution, as a health benchmark. TLV are intended for occupational exposures, for health workers, and for non-continuous exposures. Some states have used TLVs to establish protective ambient concentrations for air toxics by dividing

the TLV by either a time scaling factor and/or a safety factor. Typical safety and scaling factors range from 10 to 300. In the absence of other data, crude toxicity measures can be used. Low toxic concentration (TC_{10}) or dose (TD_{10}) are the best of such measures. In some circumstances of very limited data, only lethality data may be available. Lethality data (e.g., LC_{50}, LD_{50}) are only crude indicators of relative toxicity. For less severe effects, it is possible that the relative toxicities of compounds may differ from that indicated by lethality data.

A graphical comparison of estimated styrene concentrations and relevant health benchmarks for styrene is shown in Figure 18.5. The 8-h time-weighted average TLV for styrene is 215 mg/m^3. Estimated peak exposure levels of 0.092 mg/m^3 are well below the TLV, well below the typical safety and scaling factors used in converting TLVs to protective ambient exposure levels. Peak concentration from worst-case composite is 0.420 mg/m^3 more than 500 times below the TLV. For further comparison, mild irritation of the eyes and throat have been observed in humans at levels of 425 mg/m^3 and above. Peak concentrations from composite carpet are about 4600 times below this level associated with mild irritation and 1000 below this level for the worst-case composite and Carpet 9.

A graphical comparison of estimated 2-butoxyethanol concentrations and relevant health benchmarks is included in Figure 18.6. The 8-h time weighted average TLV for 2-butoxyethanol is 120 mg/m^3. The estimated peak exposures from the composite carpet of 0.044 mg/m^3 (about 2700 times below the TLV) and from the worst-case composite carpet of 0.326 mg/m^3 (about 370 times below the TLV), are well below the safety factors used in converting TLVs to protective ambient concentrations. In addition, the observed lowest toxic concentration is 480 mg/m^3, at which there were changes observed in the CNS and respiratory irritation indicated by dyspnea. Nausea and vomiting have been observed at levels of 936 mg/m^3 and above. Again the estimated peak concentration is well below these health benchmarks.

There are very few data available for the remaining six compounds (propylbenzene, decane, undecane, 4-PCH, 4-VCH, *m*-ethyltoluene) but all are believed to be of low toxicity. Available data are typically limited to acute toxicity data (e.g., TD_{10},TC_{10}, LD_{10}, and LC_{10}). These measures are not useful directly in assessing risks at low exposure concentrations but can be used in a comparison with other compounds. For example, for 2-butoxyethanol the lowest LC_{50} value reported for inhalation is 3360 mg/m^3 for mice. By comparison, the inhalation LC_{10} in mice is 27,000 mg/m^3 for 4-VCH, 20,000 mg/m^3 for propylbenzene, and 72,300 mg/m^3 for decane (NIOSH, 1991). Therefore, using lethality data as an indicator of overall relative toxicity, these three compounds can be considered much less toxic than 2-butoxyethanol. As described above,

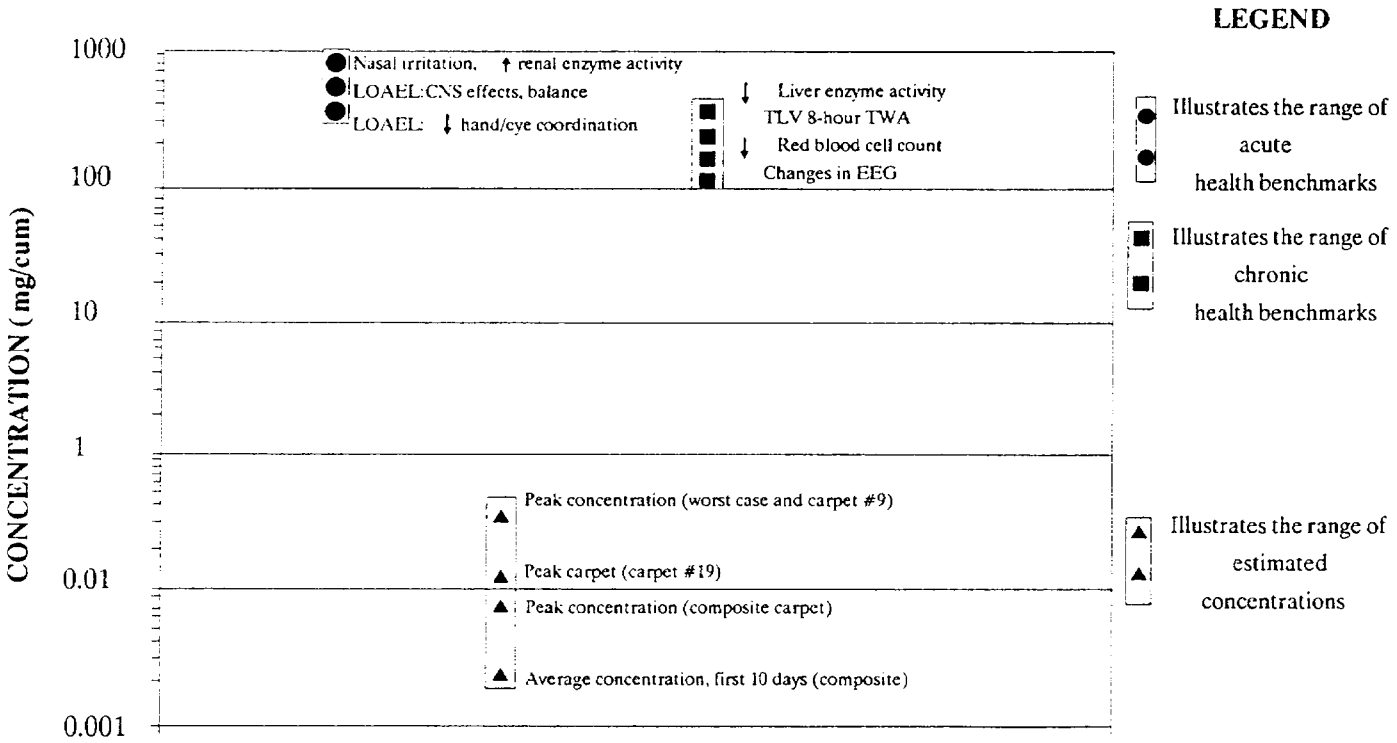

Fig. 18.5: Comparison of styrene concentrations levels to relevant health benchmarks.

adverse effects were not predicted for exposure to 2-butoxyethanol. Based on lower relative toxicity of these three compounds and lower peak concentrations (0.006–0.012 mg/m^3 versus 0.044 mg/m^3), it is unlikely that adverse effects would occur from exposure to either 4-VCH, propylbenzene, or decane.

Undecane is moderately toxic by the intravenous route, but there are no data available on the toxicity by the inhalation route. Given the low peak concentration from composite carpet of 0.004 mg/m^3 and relatively low toxicity it is unlikely adverse effects would occur.

No toxicity data were available on *m*-ethyltoluene but it is believed to be of low toxicity. Related isomers (*o*-ethyltoluene and *p*-ethyltoluene) have some limited data and have been found to be of low toxicity with LD_{10} values of 5000 mg/kg. As a result, this compound is believed to be less toxic than propylbenzene (which has a LD_{50} of 6.40 mg/kg). There have been several toxicity studies conducted for the Office of Toxic Substances on the C9 fraction from petroleum refining which includes (about 7%) *m*-ethylbenzene and 23% other ethyltoluene isomers.

Toxicity studies on the C9 fraction include general inhalation toxicology, inhalation neurotoxicity, and inhalation development toxicity studies. Each of these tests showed the C9 fraction to be of relatively low toxicity. By inference *m*-ethyltoluene can also be considered to be of low toxicity and given the low peak concentration (0.002 mg/m^3) it is unlikely that adverse health effects would be expected.

There are very few published data in the toxicity of 4-PCH, though recent studies have shown 4-PCH to be of low toxicity (Nitschke et al., 1991). Nitschke et al. (1991) conducted studies to determine the potential for dermal sensitization and inhalation toxicological potential of 4-PCH. Dermal sensitization studies in guinea pigs did not reveal any evidence of delayed contact hypersensitization. Rats were exposed to 4-PCH of up to 320 mg/m^3 for two weeks and showed no clinical or pathologic evidence of eye, skin, nasal, or respiratory tract irritation. No exposure related effects were found in any of the parameters analyzed. The relatively low toxicity of 4-PCH is also supported by an observed oral LD_{50} greater than 2000 mg/kg (Nitschke et al., 1991). Therefore, while a quantitative measure of toxicity cannot be made, the relatively low toxicity and low peak concentrations (0.057 mg/m^3) indicate that adverse effects are not predicted by exposures to 4-PCH.

18.5.2 Cancer health effects analysis for individual constituents

Styrene is designated as a B2 Probable Human Carcinogen by the U.S. Environmental Protection Agency (EPA). The unit risk factor for styrene is 5.7×10^{-7}) $(\mu g/m^3)^{-1}$. This unit risk factor is used to calculate the probability of

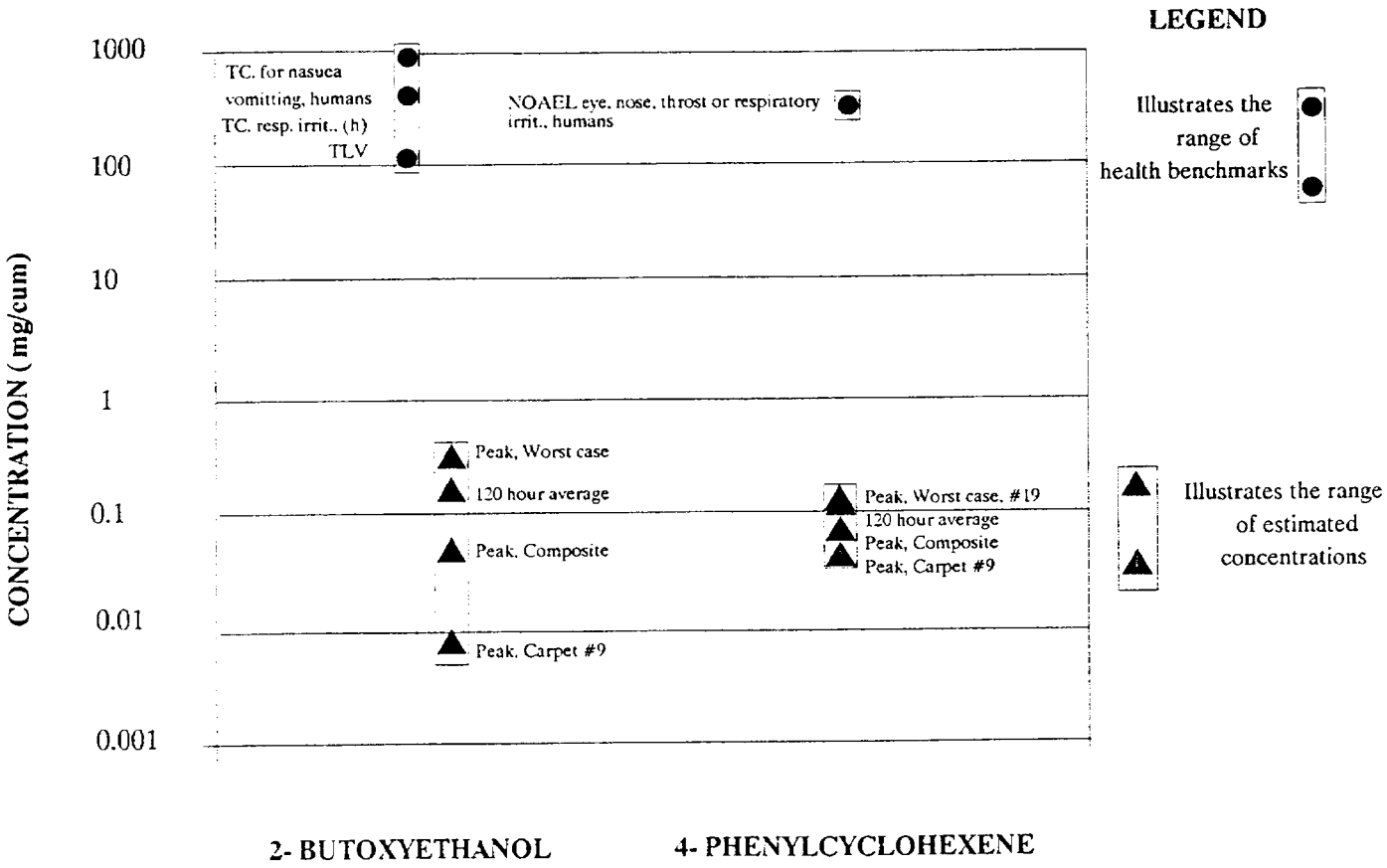

Fig. 18.6: Comparison of concentrations with relevant health benchmarks.

developing cancer during one's lifetime due to exposure to styrene. For example, a lifetime average exposure level of 1.75 g/m^3 corresponds to a one in a million lifetime risk of cancer.

The estimated peak (about 2.5 h) concentration for styrene from composite carpet is 92 μg/m^3 (0.092 mg/m^3). Indoor concentration decay with time and after 120 h the styrene concentration has been reduced to 9 μg/m^3. Further decreases in indoor levels of styrene are anticipated and it is estimated that indoor concentrations would approach 0 μg/m^3 at 236 h after installation. Therefore, exposure to styrene due to off-gassing from carpets would be anticipated to be limited to the first 10 days (240 h). The average exposure level during the first 10 days is 22.9 μg/m^3 which equates to a lifetime (70 year) average exposure level of 0.009 μg/m^3. Based on this average exposure level and the cancer potency factor, the lifetime cancer risk associated with styrene from carpets is estimated at 5.1×10^{-9}. Lifetime cancer risk is associated with exposure to new carpet, if an individual were to be exposed to newly installed carpet several times during a lifetime, the lifetime cancer risk would then be 5.1×10^{-9} times the number of carpet installations.

The estimated peak concentration for styrene from the worst case composite and Carpet 9 is 420 μg/m^3 (0.420 mg/m^3) with concentrations after 120 h reduced to 48 μg/m^3 and would approach 0 μg/m^3 at 320 h after installation. Therefore, exposure to styrene from worst-case composite due to off-gassing from carpets would be anticipated to be limited to the first 14 days (336 h). The average exposure level during the first 14 days is 80.1 μg/m^3 which equates to a lifetime (70 year) exposure level of 0.044 μg/m^3 for each carpet installation. Based on this average lifetime exposure level and the cancer potency factor, the lifetime cancer risk associated with styrene off-gassing from new carpets is estimated at 2.5×10^{-8}, or 25 cancer cases in 1 billion persons exposed.

For comparison the EPA uses cancer risk levels of between 10^{-4} (one in 10,000) and 10^{-7} (one in 10 million) as being protective of human health. The risk associated with carpets is well below this protective range. Therefore, it is not anticipated that styrene exposures from new carpets represent a significant risk. However, it should be noted that assessing peak exposures for cancer risks is difficult in that most models utilize potency factors based on an average lifetime exposure level and do not consider such issues as timing of exposure or age at exposure. No other compound in the composite carpet is a verified or designated carcinogen by the EPA.

18.5.3 Health effects analysis of the mixture

Exposure to individual constituents do not appear to predict any adverse effects. However, the mixture as a whole also needs to be assessed. While

individual constituents may not result in adverse health effects, it may be possible that the mixture, of many compounds may result in some adverse effects. Two methods of evaluating the mixture have been applied to the compounds in the composite carpet, the hazard index and margin of exposure approaches. Both of these methods utilize the No Observed Adverse Effects Level (LOAEL) from either animal toxicology studies or human epidemiologic data. The margin of exposure (MOE) approach extends the individual MOEi, as described above, to a mixture and sums individual MOEi. The MOEi for an individual substance (MOE) is defined as the ratio of NOAEL to exposure of a compound (EPA, 1989). The MOE is not a direct measure of risk, as the MOE approaches the value of 1 the level of concern for possible effects is increased. It is interpreted as the extent to which human exposures are below the observed NOAEL in the study species. For a mixture, the MOE is the sum of the MOEi for the individual constituents for a given target organ. Individual MOEi are summed only for the same target organ system.

Compounds with RfCs

Four compounds of the composite carpet had verified inhalation RfCs. MOEis were calculated for these compounds and are presented for the composite carpet in Table 18.5. Three of the 4 compounds having RfCs have identified NOAELs for the central nervous system. Therefore, a mixture MOE for the central nervous system (MOE_{cns}) can be calculated by using the MOE_{cns} equation shown on the bottom of each of these Tables. The MOE_{cns} was calculated as 3760 for the composite carpet. This is interpreted as the exposures are about 3760 times below the "threshold value for the mixture" with regard to central nervous system effects.

The mixture MOE approach was also applied to the worst case carpets. The MOE_{cns} ranged from 670 for the worst case composite to 1600 for Carpet 9. These values are similar to the uncertainty factor of 1000 used in the calculation of the subchronic RfC for cumene alone. This indicates some concern over possible adverse effects. However, these MOE values are calculated using peak concentrations, which last for short periods of time. Concentrations (and exposures) would decrease over time and, therefore, the likelihood of adverse health effects is also decreased.

The Hazard Index (HI) approach is the most common method used for non-cancer effects from mixtures and is suggested in the EPA Guidelines on Complex Mixtures (EPA, 1987b). It is the accepted practice in Superfund risk assessments. The hazard index is the sum of the hazard index for individual compounds (HIi) for a given target organ. The hazard index for individual compounds is the ratio of exposure of a compound to the RfC. Similar to the

MOE approach, as HIi values approach 1 the level of concern for possible health effects increases. However, for the Hazard Index approach, values exceeding 1 indicate that the RfC has been exceeded. The HIi values calculated for those 4 compounds having verified RfCs vary from 0.007 for ethylbenzene to 0.555 (chronic) or 0.055 (subchronic) for cumene.

HIi values can be summed for a specific target organ to obtain an overall mixture Hazard Index. RfCs for three compounds, cumene, toluene, and xylene, are based on central nervous system; (HIcns) was calculated for the composite carpet as 0.574 which indicates that the exposure is less than the estimated "mixture RfC" for CNS effects. When the subchronic RfC for cumene is used the HIcns was calculated as 0.074. Therefore, adverse health effects are not anticipated from exposure to the mixture of cumene, toluene, and xylene. While there is significant uncertainty in the RfC, this is a useful method for assessing some health (CNS) effects for the example mixture.

The HI approach also was applied to the worst case carpets. The HI_{cns} for worst case carpets ranged from 1.60 (Carpet 9) to 2.555 (worst case composite) when the chronic RfC for cumene was used. These values all exceed one, which indicates that the potential for adverse effects may exist. However, when the subchronic RfC value for cumene is used — a more appropriate measure given the short duration of exposure — the HI_{cns} for each carpet was below 1 ranging in values from 0.198 (Carpet 9) to 0.355 (worst case composite). Given that values of HIcns are below using peak exposure levels, it is not likely that adverse health effects would be expected from exposure to carpet.

18.6 CONCLUSIONS

The use of volatile organic emission rate and decay constant data proved useful in estimating concentration profiles, and to simulate exposures associated with new products. In the past, sampling occurred some time after installation and was incapable of quantifying initial peak concentrations which may be responsible for increases in adverse health complaints. Coupling time-activity patterns with concentration profiles allows for the construction of exposure profiles. This technique for the estimation of exposures is particularly useful in evaluating indoor pollutant sources with high initial emissions which experience reductions in organic emission over time and the potential for health effects from these sources.

Based on this analysis, exposure to volatile organics from newly installed carpets does not result in adverse health effects. Estimated peak concentrations for individual compounds were below relevant and available health benchmarks. Concentration profiles were estimated for each of the 12 constitu-

ents in the composite carpet. Peak concentrations ranged from a high of 0.092 mg/m^3 for styrene to a low of 0.002 mg/m^3 for *m*-ethyltoluene. The major contributors to indoor concentrations of volatile organics were styrene, 4-PCH, 2-butoxyethanol and, to a lesser degree, 4-VCH. The remaining compounds were relatively minor contributors to total volatile organics. Peak concentrations for individual constituents were used as a worst case initial analysis. However, actual concentrations would decrease over time and, exposure would be limited by activity patterns. In all cases, the peak concentrations for individual constituents do not exceed any relevant non-cancer health benchmarks that are currently available. Therefore, adverse health effects are not anticipated to result from exposure to newly installed composite carpet. This conclusion was further supported by the worst case analyses using a worst case composite carpet and the two highest emitting individual carpet products. Using peak concentration levels from these sources also did not result in predicting adverse health effects.

Exposure to styrene from installation of new carpets occurs almost entirely within the first ten days. From the data used in this study the amount of this styrene exposure equates to a lifetime cancer risk of 5 cancer deaths in one billion persons exposed. This represents the cancer risk associated with a new carpet installation, if carpets were to be installed more than once in a lifetime, the lifetime cancer risk would be 5×10^9 times the number of carpet installations in a lifetime. The worst case composite data resulted in cancer risk of 25 cancer deaths in one billion persons exposed to carpets. These cancer risk associated with styrene exposure from carpet emissions are well below the levels assumed by the EPA to be protective of human health.

This study should be viewed as only an initial phase in the work needed to address exposures and risks associated with organic emissions from carpets, carpet padding, consumer products, building materials and building renovations. There are several limitations which prevent this from being a comprehensive indoor air quality study. Only a single source (carpets) was tested without associated materials such as padding and adhesives. Volatile organic emissions from new carpet installation may actually be higher when other materials (i.e., pads or adhesives) are also used.

This study did not include other materials used in building renovations (such as partitions, paints, particle-boards). The health effects analysis for the individual components was limited by available data on individual compounds, many of which had little or no available data. Analysis for other compounds was not done due to the unavailability of data on potential interactions (synergism and/or antagonisms) between individual constituents.

PART III — REFERENCES

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PART IV
Investigation, Diagnosis and Management of Illnesses and Complaints Related to Buildings

This part deals with medical aspects of illnesses and complaints related to buildings. After defining building-related illnesses and complaints, their epidemiology and the use of questionnaires in building investigation are presented and discussed. The different diseases observed are described and guidance is provided for medical diagnosis and treatment. Finally, general aspects of medical management of building-related diseases are discussed.

Chapters 19, 20 and 22 have been prepared by F. Levy; Chapter 21 by A. Pickering, with contributions from J. Day. The contents of this part derive from presentations made at the NATO/CCMS workshop "Epidemiology and Medical Management of Building-Related Complaints and Illnesses" held in Olso, in 1991, the proceedings of which are available.

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Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 19

Epidemiology of Principal Building-Related Illnesses and Complaints[1]

19.1 INTRODUCTION

People in the industrialized world spend about 90% of their lives indoors (Moschandreas, 1981). Chemical measurements have shown that for many organic compounds, indoor concentrations are higher than the outdoor ambient concentrations (Jarke et al., 1981; Yocom, 1982; Berglund et al., 1982b). Thus, for the majority of the population, not exposed to chemicals in polluted industrial environments, the indoor environment may contribute to a large proportion of their life dose of volatile organic compounds.

During recent decades, concern has increased about more subtle health effects resulting from low level exposure to complex mixtures of chemicals and bioaerosols. One such new area involves the possible health effects of poor indoor air quality in non industrial work environments (e.g. schools, offices, hospitals, day care centres).

Human reactions to the indoor environment are divisible into three main categories:

1. Certain building-related diseases with known aetiologies such as allergic alveolitis (hypersensitivity pneumonitis), humidifier fever, building related asthma, legionellosis and radon-induced lung cancer.
2. Complaint reactions to a poor indoor environment. In such cases, unpleasant stimuli are perceived by the subject, who externalizes the cause to the environment. Examples of such complaints are thermal discomfort, complaints of stuffy air, dry air, static electricity and malodours.
3. Medical symptoms with an unclear aetiology, but with a possible relationship to the indoor environment. The term "sick building syndrome" has been used to describe such symptoms. This syndrome was defined

1 A part of the text of this chapter has been derived from D. Norbäck "Epidemiology of Principal Building-Related Diseases" in: Report on a Meeting "Epidemiology and Medical Management of Building-Related Complaints and Illnesses", pp. 27–46, Oslo, Norway, 19–21 August 1991. Eds. F. Levy and M. Maroni. NATO/CCMS Pilot Study on Indoor Air Quality.

by a working group of the World Health Organization, and involves various nonspecific symptoms such as eye, skin and upper airway irritation, headache and fatigue (Akimenko et al., 1986).

19.2 DEFINITIONS

Building-related illness (BRI)

BRI is a term referring to illness brought on by exposure to the building air, where symptoms of a diagnosable illness are identified and can be directly attributed to environmental agents in the air.

Sick building syndrome (SBS)

From most countries in the industrialised western world there are reports about sick buildings or of syndromes such as "tight building syndrome", "stuffy office syndrome", "office illness", "day care centre illness", "illness due to work with carbonless copy paper", "VDU-workers syndrome" and the like. With small alterations they are described in the same way, with the same type of symptoms.

A working group within WHO was the first to propose a description of SBS in 1982: "The symptoms reported are of a broad spectrum but have many features in common such as:

- eye, nose and throat irritation
- sensation of dry mucous membranes and skin
- erythema
- mental fatigue
- headaches, high frequency of airway infections and cough
- hoarseness, wheezing, itching and nonspecific hypersensitivity
- nausea, dizziness".

In 1982 the WHO working group stated that "symptoms found in sick buildings can fall into four groups:

- sensory irritation of skin and upper airways, along with headache and abnormal taste
- complaints about odour
- general symptoms such as fatigue, dizziness and nausea; and
- lower airways and gastrointestinal symptoms".

It indicated that the last group is not generally found in the sick building syndrome, and that psychogenic symptoms may also be present in some cases, and may require further investigation.

19.3 EPIDEMIOLOGICAL INVESTIGATION OF PRINCIPAL BUILDING-RELATED ILLNESSES AND COMPLAINTS

Epidemiological investigations on building-related illness may be performed at different levels to answer different questions. Descriptive investigations may be performed to determine prevalence and incidence of illness and its possible relationship to the indoor environment. In analytical epidemiology, the relationship between various indoor exposures and other factors in such illness is investigated. Finally, in longitudinal analytical studies, it may be possible to calculate how much of a disease is attributable to indoor exposure (aetiological fraction). Until recently, the literature on building-related illness mainly consisted of case reports. During recent years, however, analytical epidemiological studies on some types of building-related illness have been published.

19.3.1 Building-related illnesses with a known aetiology

Extrinsic allergic alveolitis or hypersensitivity pneumonitis

This disease has been observed mainly among farmers that have been exposed to high concentrations of mouldy organic dust ("farmers' lung") (Rask-Andersen, 1988). Allergic alveolitis is a rare disease; among farmers the incidence rate is estimated to be 2–30 cases/10,000 farmers (von Essen et al., 1990). There are also, however, some exposures in the home environment that may cause allergic alveolitis. Allergic alveolitis due to indoor moulds has been reported (Belin et al., 1989). In Japan, the most prevalent form of allergic alveolitis is summer hypersensitivity pneumonitis which is caused by indoor mould growth (Ando et al., 1991). Bird fancier's lung is another form of allergic alveolitis that may occur among those handling birds (Ando et al., 1991; Belin et al., 1989). Allergic alveolitis due to exposure to microorganisms from air conditioners or humidifiers has also been described (Ando et al., 1991; Finnegan and Pickering 1986).

Organic dust toxic syndrome and humidifier fever

Organic dust toxic syndrome (ODTS) is a term recently coined to describe a noninfectious, febrile illness associated with chills, malaise, myalgia, a dry cough, dyspnea, headache and nausea that occurs after heavy organic dust exposure. This syndrome is thought to be an acute febrile reaction to massive organic dust exposure distinct from allergic alveolitis. Among farmers, ODTS is reported to be much more common than allergic alveolitis (Rask-Andersen, 1988), the incidence being 10–190 cases/10,000 farmers (Von Essen et al., 1990). An outbreak of ODTS caused by residential exposure to mouldy organic

material has also been reported (Brinton et al., 1987). Similar febrile reactions have occurred after taking sauna baths or showers with tap water that was found heavily contaminated by moulds and coliform bacteria (Muittari et al., 1982).

Humidifier fever is the commonest febrile reaction related to the indoor environment. It was first described in 1959 by Pestalozzi, who described an outbreak of severe tiredness of the limbs, 4–8 h after exposure to humidifiers, fever with shivering, 8–12 h after exposure, and shortness of breath and coughing (Pestalozzi, 1959). Subsequently there have been a number of publications describing similar outbreaks but there is no epidemiological data available on the prevalence or incidence of ODTS or humidifier fever in relation to indoor exposures.

Building-related asthma

Residential allergen sources include plants, dogs, cats, rodents, birds, and cockroaches (Samet, 1990). House dust mites (Andersen and Korsgaard, 1986), storage mites (Iversen and Dahl, 1990) also produce allergens that may cause bronchial asthma, and such allergy is often related to damp housing conditions. There is, however, minimal documentation on asthma related to other building-related exposures. In case reports, asthma provoked by contaminated humidifiers has been described (Burge et al., 1985; Finnegan and Pickering 1984; Solomon 1970). A case-referent study on 72 asthmatics, and 72 controls, showed that asthmatics were more exposed to indoor moulds than controls, especially Penicillium (Burr et al., 1988). Home dampness and moulds were shown to be likely risk factors for respiratory disease in a Canadian population in a recent epidemiological survey (Dales et al., 1991).

Vapours of volatile organic compounds (VOC) commonly contaminate indoor air, and a relationship between indoor VOC concentration and upper airway symptoms has earlier been demonstrated in epidemiological studies (Norbäck et al., 1990a,b). The relationship between indoor VOCs and asthma has not yet been investigated in epidemiological studies. In a recent exposure chamber study, however, a decline of FEV1 was registered among asthmatic subjects after a 90- min exposure to 25 mg/m^3 of a mixture of VOCs (Harving et al., 1991). The authors conclude that VOCs can be of importance as bronchial irritants in the work environment and perhaps even in the home environment and that other VOCs may be of greater importance as indoor irritants than formaldehyde.

Other building-related allergic reactions

The incidence or prevalence of building-related allergic rhinitis is not known, and is documented in the literature only in sporadic case reports

(Robertson and Burge, 1985). Allergic reactions to bioaerosols emitted from air-conditioners seem to be of importance. In a German study among 150 patients who suffered from various symptoms in air-conditioned rooms, 135 patients showed allergic symptoms in response to skin or challenge tests. Most positive results were produced by Penicillium species (Schata et al., 1989). A case report of various types of symptoms and antibodies to toluene diisocyanate (TDI) in a redecorated office has recently been published (Thrasher et al., 1989).

Building-related infections: legionellosis and Pontiac fever

The epidemiology on legionellosis and Pontiac fever consists of sporadic case reports, and there seem to exist no systematic studies on the prevalence of these diseases in the general population. Various case series, however, suggest that between 1 and 27% of community-acquired pneumonia may be due to legionella (Kreiss, 1989).

Other types of building-related infections

Legionellosis and Pontiac fever are examples of infectious diseases in which the ventilation system may both amplify and transmit legionella. Influenza, common colds, measles, rubella, tuberculosis, and Q fever are other examples of infections whose transmission can be facilitated by indoor air circulation and by the ventilation system (Burge, 1989; LaForce, 1986). In a recent epidemiological study, the rate of acute febrile respiratory disease was higher among army trainees in modern barracks as compared to old barracks, suggesting that airborne respiratory pathogens are more easily spread in modern energy-efficient buildings (Brundage et al., 1988).

Cancer and the indoor environment

Indoor air may contain many different chemical compounds, usually in low concentrations (Jarke et al., 1981). Some of these compounds are suspected human carcinogens (Mølhave 1982; WHO, 1989). One common indoor pollutant is tobacco smoke, and passive smoking may affect the health of both adults and children in dwellings and workplaces. The weight and consistency of the epidemiological literature favours a link between passive smoking and serious disease, especially lung cancer (Woodward, 1991). Recent studies have showed a relative risk of 1.1–2.3 on lung cancer and passive smoking (Pershagen, 1990), and calculations on the aetiological fraction attributable to passive smoking suggest that about 1% of all lung cancers can be explained by passive smoking (Edling, 1990). One recent case-referent study, however, concludes that as much as 17% of all lung cancers among nonsmokers can be attributed to passive smoking during childhood or adolescence (Janerich et al., 1990).

Radon, and radon daughters, are other indoor carcinogens that have been studied in epidemiologic investigations. The carcinogenicity of radon is well documented both in experimental studies on animals and in studies on miners. During the last decade the potential hazards of radon exposure in dwellings have also been evaluated. Swedish case-referent studies have shown a relative risk of 1.6–2.0 for lung cancer (Axelson et al., 1979, 1988; Edling et al., 1984, 1986; Svensson et al., 1987, 1989). Aetiological fractions calculated from these studies ranged from 25 to 29% and suggest that up to 30% of all lung cancers in the studied populations might be attributable to radon in dwellings. Recently, relationships between radon exposure in homes and acute myeloid leukaemia (Henshaw et al., 1990), childhood leukaemia and other childhood cancer (Collman et al., 1991) have also been demonstrated.

Epidemiological studies on non-industrial exposure to indoor carcinogens other than radon and tobacco smoke, are sparse in the literature. There are, however, theoretical estimates of the expected carcinogenic risk of some indoor volatile organic compounds. The World Health Organization has estimated that less than 2% of the total incidence of leukaemia would be attributable to indoor exposure to benzene (WHO, 1989). In another publication, the carcinogenic risks for 53 common indoor chemicals were estimated, using a linear multi-stage procedure (Tancrede et al., 1987). They conclude that none of the mean estimates of risk for particular chemicals, nor the total mean estimate of risk, reached the risk from exposure to average levels of radon in homes (10^{-4}/year). The upper 98th percentile of the cancer risk was, however, greater than 10^{-4} per lifetime for some compounds such as styrene, formaldehyde and some chlorinated hydrocarbons (Tancrede et al., 1987).

19.3.2 Complaints of poor indoor air quality

As for the sick building syndrome, complaints of poor indoor air quality have mainly been investigated among office workers. The Danish Town Hall Study has demonstrated that complaints of poor indoor air quality are common among office workers in Copenhagen, and usually women reported more complaints than men (Skov et al., 1987) (Table 19.1).

All complaints listed in Table 19.1, except complaints of high temperature, differed significantly between men and women ($p < 0.05$).

In a telephone interview investigation among 600 randomly selected office workers in USA, even higher prevalences of complaints were reported: 47% complained of poor temperature control; 41% were disturbed by noise; 39% perceived poor indoor air quality; and 32% complained of inadequate lighting. Moreover, 20% of the office workers perceived their performance to be hampered by poor indoor air quality (Woods et al., 1987).

TABLE 19.1

Prevalence of work-related complaints during winter in the Danish Town Hall Study (Skov et al., 1987)

	Men (n = 1100)	Women (n = 2328)
Draught	18%	35%
Noise in the office	15%	25%
Poor lighting	9%	16%
Static electricity	11%	16%
High temperature	18%	19%
Low temperature	15%	23%
Stuffy air	24%	41%
Dry air	30%	52%
Dusty air	17%	28%
Odour	6%	12%

TABLE 19.2

Complaints on the indoor climate among office workers in Northern Sweden (Stenberg et al., 1990)

	Men	Women
High temperature	7%	9%
Noise in the office	7%	10%
Stuffy air	15%	28%
Dry air	17%	43%

In a questionnaire study in 6000 randomly selected office workers in northern Sweden, complaints of poor indoor air quality were common, and more common among women than among men (Stenberg et al., 1990) (Table 19.2).

Besides investigations among office workers, there are two Scandinavian population based investigations on indoor air quality. In a Norwegian interview investigation, 2197 randomly selected adults were interviewed during winter time. Here, 14% reported poor indoor air quality at the workplace, while only 1% reported poor air quality in the dwelling (Saetre, 1990). In a Swedish questionnaire study in a random sample of 466 adults in Middle Sweden, 6% of the non-industrial population reported symptoms related to poor indoor air quality at work, while only 0.6% had symptoms related to poor indoor air quality at home (Norbäck, 1991).

19.3.3 Sick building syndrome

Until recently, the literature on SBS mainly consisted of case reports on sick buildings (Kreiss, 1989). In recent years, however, a few larger cross-sectional studies using multivariate analysis have been published. Most of the studies use self-administered questionnaires as a cost-effective means of gathering information on symptoms (Burge et al., 1987; Skov et al., 1987; Hedge et al., 1989; Norbäck, 1990). In a few studies, objective signs of eye irritation (Franck, 1986; Franck and Skov, 1989) and dermatosis (Stenberg, 1989) have been studied in order to establish the relationship between such signs and symptoms.

Personal factors

In most studies, females report a higher prevalence of symptoms (Burge et al., 1987; Hedge 1984, 1989; Norbäck and Edling, 1991; Taylor et al., 1984; Valbjörn and Kousgård 1984), the odds ratio being 1.6 for mucosal irritation and 1.8 for general symptoms (Skov et al., 1989). The relation between age and SBS appears more complex, and there seems to be no consistent relation between age and SBS symptoms (Norbäck, 1990).

Atopy, allergy to nickel, non-specific hyperreactivity and infection proneness have been shown to be related to SBS (Norbäck and Edling, 1991). In the Danish Town Hall study, individuals with a history of hay fever had a slightly higher prevalence of work-related mucosal irritation (OR = 1.6), but no excess of general symptoms (Skov et al., 1989). Epidemiological studies on the relation between SBS and other personal factors are sparse in the literature. Office workers suffering from migraine report a higher prevalence of work-related general symptoms (headache or fatigue) than office workers without migraine (OR = 1.8), but no excess mucosal irritation (Skov et al., 1989).

Tobacco smoking

SBS symptoms are more common among smokers than non-smokers (Rogier et al., 1989; Skov et al., 1989). Rogier et al. (1989) found an OR of 1.8 for SBS symptoms among smokers as compared to non-smokers. Skov et al. (1989) found an OR of 1.3 for general symptoms and no significant excess of mucosal irritation among smokers.

In two Swedish studies, smoking among adults was related to an increased prevalence of symptoms in sick buildings (Norbäck et al., 1990a), but not in the general population (Norbäck and Edling, 1991). In another longitudinal study, smoking increased the incidence but not the prevalence of SBS symptoms (Norbäck et al., 1990b).

Residential factors

On average, the population spends about 5 times more time at home than at work (Moschandreas, 1981). Despite this fact, the SBS literature has mainly studied the effect of nonresidential exposure. In the Town Hall study (Skov et al., 1989), some residential factors were studied and a relation between indoor climate problems in the residence and general symptoms was found (OR = 1.6). In a preliminary report from another Danish study (Valbjörn and Kousgård, 1984), smoking in the dwelling, high population density, flats, terraced houses, and building moisture or moulds at home were related to a higher prevalence of headache or mucosal irritation. In a Swedish study in the general population, current urban residency, fresh paint, and pre-school children in the dwelling were related to an increased prevalence of SBS symptoms (Norbäck and Edling, 1991).

Occupational factors

Most of the SBS studies are restricted to office workers, and information on prevalence of such symptoms among employees from other occupational settings is sparse. Specific office tasks such as photocopying (Skov et al., 1989; Taylor et al., 1984), handling of carbonless copying paper (Norbäck et al., 1983; Skov et al., 1989) and work with video display units (Norbäck and Edling, 1991; Skov et al., 1989) have been demonstrated to be related to SBS symptoms. In addition, an association between psychosocial factors at work and SBS symptoms, particularly general symptoms, have been demonstrated (Michel et al., 1989; Norbäck et al., 1990a,b; Norbäck and Edling, 1991; Skov et al., 1989). Crowdedness (Taylor et al., 1984; Valbjörn and Kousgård, 1984), office size (Skov et al., 1989), poor environmental control (Hedge et al., 1989) and architecture of the workplace (Hedge, 1984) may also influence the prevalence of SBS symptoms. A higher prevalence of symptoms among office workers in the public sector as compared to such workers in the private sector has also been demonstrated (Hedge et al., 1989).

Childhood environmental exposure

Exposure to common allergens during the first months of life has been shown to be associated with an increased risk of developing atopy, particularly among children with allergic parents (Björksten and Kjellman, 1987; Croner and Kjellman, 1986). Maternal smoking during pregnancy leads to increased IgE levels in cord blood and increases the risk of subsequent allergy (Magnusson, 1986). Exposure to environmental tobacco smoke during childhood has also been shown to influence various childhood health parameters such as prevalence of bronchial responsiveness (Andrae et al., 1988; Martinez et al., 1988), respiratory disease (Schenker et al., 1983), atopy (Björksten and Kjell-

man, 1987), and otitis media (Pukander et al., 1985; Stählberg et al., 1986). Outdoor environmental pollution (Andrae et al., 1988; Dales et al., 1991), moisture in the dwelling (Andrae et al., 1988), and wood-burning stoves (Honicky et al., 1985) may also affect the prevalence of respiratory illness among children.

Most of the studies on childhood environmental exposures have only evaluated the health effect on children. In one Swedish population study on adults, however, data on childhood exposure were gathered retrospectively. Childhood exposure to tobacco smoke from smoking mothers doubled the prevalence of both atopy and nickel allergy in the adult population. A relation between sick building syndrome and childhood exposure to tobacco smoke and the urban environment was also demonstrated (Norbäck and Edling, 1991).

Outdoor environmental exposure

Urban residency may lead to a higher exposure to outdoor pollutants, because of traffic or other pollution sources. In addition, urban residency may be related to a more unhealthy lifestyle than rural residency. Many SBS investigators do not mention if the buildings studied are situated in rural or urban areas. It appears, however, that most of the studies in this field have been performed in large cities such as Copenhagen (Skov et al., 1989), Helsinki (Jaakkola et al., 1989), Stockholm (Göthe et al., 1989), New York (Taylor et al., 1984) and San Francisco (Turiel et al., 1983). In one Swedish study the effect of urban residency on SBS symptoms in the general population was studied. Here, a significant relationship between urban residency and airway symptoms was demonstrated (Norbäck and Edling, 1991). This indicates that urban residency should be controlled for in epidemiological investigations on the sick building syndrome.

Outdoor air rate

The rate of outdoor air supply is used as the criterion of most ventilation standards (ASHRAE, 1989; Sundell, 1982; Yaglou, 1936). Since humans emit carbon dioxide, there is a relationship between carbon dioxide concentrations and outdoor air supply. As early as 1858 it was shown that CO_2 could be used as indicator of human emissions (body odour), and at levels above 1000 ppm, non-adapted visitors found the indoor air quality unacceptable (Pettenkofer, 1858). Today, similar ventilation standards are used, and 800 ppm has been suggested as a control limit value for CO_2 (Berglund et al., 1984b). Few investigations, however, have been able to demonstrate a relationship between the outdoor air supply and the prevalence of sick building syndrome. In a preliminary report from a study among 150 office workers from 5 offices, a relationship between CO_2 levels and SBS symptoms was demonstrated (Hodg-

son et al., 1989). Other studies have not found any relationship between CO_2 levels and symptoms (Norbäck et al., 1990a,b; Skov et al., 1989). In particular buildings however, improved ventilation in combination with other improvements can decrease the number of SBS symptoms (Turiel et al., 1983; Hansen 1989; Helsing et al., 1989). The lack of relationship between outdoor air rate and SBS symptoms may be due to the fact that non-human factors such as building materials or the ventilation system may be the dominant source of air pollution in the modern indoor environment (Fanger, 1987).

Type of ventilation system

Both Danish and British studies of office workers have indicated a relationship between the type of ventilation system and the prevalence of SBS symptoms. In one study, a higher prevalence of mucosal irritation, general symptoms and skin symptoms was found in six mechanically ventilated buildings, whether or not air was recirculated, in comparison with three naturally ventilated buildings (Finnegan et al., 1984). The Danish Town Hall study demonstrated that office buildings with supply air systems had a higher prevalence of mucosal irritation than buildings with exhaust ventilation only (Skov et al., 1990). The last and largest study evaluated 4373 office workers in 42 office buildings and 47 ventilation conditions (Burge et al., 1987). Symptoms were most common in buildings in which the air was chilled or humidified. Naturally or mechanically ventilated buildings without air chilling or humidification had the lowest prevalence of symptoms. The authors conclude that it is air conditioning rather than the mechanical ventilation as such that has an influence on SBS, the possible cause being microbial growth in humidifiers or chilling units.

Microorganisms and bioaerosols

Bacteria and moulds may grow in building material when moisture content is high. Fungal or bacterial growth has also been found in some office buildings with sick building complaints (Morey, 1984). In a Swedish survey, 50% of the sick buildings known by occupational health care centres were reported to be affected by either mould growth or water damage (Widström and Norbäck, 1988). In dwellings, a relationship between damp houses and a higher prevalence of respiratory symptoms (Waegemaekers et al., 1989) and hyperreactivity symptoms (Andrae et al., 1988) among children has been demonstrated. It is well known that thermophilic fungi, e.g. Aspergillus species, may survive and grow on human mucous membranes among individuals with impaired function of the immunological system (La Force, 1986). It has been suggested that thermophilic fungi may be of significance for the sick building syndrome even among normal subjects. This hypothesis was supported by a study among

33 subjects in Swedish dwellings, where aspergillus spore concentrations above 50 cfu/m^3 were associated with a higher prevalence of SBS symptoms (Holmberg, 1987).

Fleecy material and wall-to-wall carpets

In the Danish Town Hall study, a relationship between the amount of fleecy material, the amount of shelves, wall-to-wall carpets and SBS symptoms was demonstrated (Skov et al., 1990). The conclusion from this study was that fleecy surfaces, paper and cardboard may accumulate various pollutants that can be emitted due either to activity or changes in temperature and humidity. A relationship between SBS and wall-to-wall carpets has been demonstrated also in another study. In addition, it was shown that removal of carpets can significantly reduce the prevalence of SBS symptoms (Norbäck and Torgen, 1989). Wall-to-wall carpets are one special type of fleecy indoor material, which could act as a depot for various pollutants. Several studies have shown an accumulation of bacteria, mould and total amount of proteins in wall-to-wall carpets as compared to hard floor covering (Anderson et al., 1982; Gravesen et al., 1983; Gravesen et al., 1986). Dry carpet shampoos used to clean wall-to-wall carpets may also cause allergic contact dermatitis (Taylor and Hindson, 1982) and airway irritation (Kreiss et al., 1982). In a Danish case report, positive prick tests and precipitating antibodies were demonstrated against extracts of dust from poorly cleaned wall-to-wall carpets (Nexö et al., 1983).

Volatile organic compounds

The term volatile organic compounds (VOC) is used to describe the sum of organic chemicals within a defined boiling range, usually 50–260°C. The term very volatile organic compounds (VVOC) has sometimes been used for organic compounds of lower boiling range (<0–100°C) and the term semi-volatile organic compounds (SVOC) has been used to describe compounds of a higher boiling range (240–400°C) (WHO, 1989). The need for such summary terms for describing chemical exposure arises from the observation that indoor air may contain many different organic compounds, usually all in very low concentrations (Berglund et al., 1982b, 1984a; Jarke et al., 1981; Johansson, 1978).

Formaldehyde in indoor environments has been shown to correlate with symptoms related to the sick building syndrome (Main and Hogan, 1983; Olsen and Dössing, 1982). There are also both cross-sectional and longitudinal studies available demonstrating a relationship between indoor VOC concentrations and SBS symptoms (Norbäck et al., 1990a,b). In another longitudinal investigation in a suspected sick library building, a relation was found be-

tween SBS symptoms and the concentration of VOC (Berglund et al., 1990). In addition, a relationship between newly painted surfaces in dwellings, and upper airway symptoms has also been demonstrated (Norbäck and Edling, 1991).

There are also experimental studies demonstrating health effects even at exposures to low levels of VOC. In a Danish exposure chamber study, the effect of 2-h exposures to a mixture of 20 common indoor hydrocarbons was studied (Mølhave et al., 1986). The result shows that a total hydrocarbon concentration of 5 mg/m^3 could induce irritation in the eyes, nose and throat. The effect was acute and showed no signs of adaptation. In addition, performance on a digit span test deteriorated during exposure. Chemical stimulation of the trigeminal or olfactory nerves has been suggested as an explanation of the sick building syndrome (Berglund and Lindvall, 1986). There are, however, some indications that inflammatory reactions to VOCs could also be of importance in some cases. In an experimental study, it was demonstrated that a 4-h exposure of humans to 25 mg/m^3 of the same hydrocarbon mixture (Mølhave et al., 1986) induced an inflammatory response in the upper airways (Koren et al., 1990).

Aerosols and settled dust

Aerosols may rapidly be removed from the air by sedimentation and impaction. Such settled dust might be released again to the air at certain activities especially from fleecy material (Skov et al., 1990). In the Danish Town Hall Study, both airborne dust and settled dust were measured. Here, a relationship between SBS and the amount of macromolecular organic part of the floor dust, but not total concentration of airborne dust could be demonstrated (Skov et al., 1990). In other studies, however, a relationship between respirable dust in air and SBS symptoms has been demonstrated in schools (Norbäck et al., 1990b), offices (Hodgson et al., 1989) and in dwellings (Quackenboss et al., 1989).

Environmental tobacco smoke and other combustion products

There are some indications that environmental tobacco smoke (ETS) might be of importance for the prevalence of SBS symptoms. In a Finnish office investigation, a relationship between complaints on ETS and the prevalence of SBS symptoms was seen (Jaakkola et al., 1989). Recently, the British study of 4373 office workers (Burge et al., 1987) has been reanalysed. Here a relation between ETS and SBS symptoms could be demonstrated (Robertson et al., 1988). On the other hand a review of a large series of problematic buildings investigated by NIOSH suggested that only about 2% of the problems were attributable primarily to environmental tobacco smoke (Melius et al., 1984).

Besides tobacco smoking, possible sources of indoor combustion pollutants

can be vehicles, gas ranges, pilot lights, kerosene heaters, wood and coal stoves, and candle lights (Lambert and Samet, 1989). Most of these sources seem to occur in dwellings rather than in non-industrial workplaces, at least in the Scandinavian countries. There are a few case reports on problem buildings where mucosal irritation or general symptoms could be related to exposure to combustion gases from underground garages or boiler stack gases (Lambert and Samet, 1989). However, we have not found any epidemiological investigations on the significance of combustion sources for the prevalence of SBS in non-industrial workplaces.

Building age and specific building material

Most of the problem buildings ("sick buildings") seem to be new buildings, built after the energy crisis in 1974 (Widström and Norbäck, 1988). It is well known that there is a decay of the emission of volatile chemicals from building materials over time, and the highest chemical exposure is found in new buildings (Berglund et al., 1982a). In addition, building technology has changed over time and more sophisticated ventilation systems and new building materials have been introduced during the last decades. Thus, an inverse relationship between building age and the prevalence of SBS symptoms could be expected. Such a relationship has also been demonstrated among office workers in the Town Hall study (Skov et al., 1987). It has also been shown that moving from old to new dwellings resulted in an increase in SBS symptoms (Norbäck et al., 1990b).

There are 25,000 types of building materials registered in Sweden (HIM Group, 1987), and there are no indications that the range of building material is narrower in other industrialized countries. The emission rate of chemicals could differ with one or two orders of magnitude between different building materials (Levin, 1989). In addition, the rate of emission from different brands of the same type of building material could differ substantially (Levin, 1989). Therefore, it is not surprising that there are few epidemiological studies relating specific building materials to the prevalence of SBS symptoms.

Chip board and foam insulation have been identified as important sources of formaldehyde emissions, and formaldehyde emissions have also been shown to be related to an increased prevalence of SBS symptoms (Olsen and Dössing, 1982). There has also been a concern about man-made mineral fibres (MMMF) as a cause of SBS symptoms (Rindel et al., 1987). Moist mineral wool may emit malodour and chemicals such as higher aliphatic aldehydes (*n*-hexanal to *n*-decanal) (Van der Waal et al., 1989), and malodorous bacteria may grow in such thermal insulation (Ström et al., 1990). However, we have not found any epidemiological investigations demonstrating any clear relation between MMMF and SBS symptoms.

Building moisture and water damage

There are indications that the water content in building material may have an effect on the emission of VOC by several mechanisms. Microorganisms may emit odorous chemical compounds such as geosmin (Gerber, 1968) and 1-octen-3-ol (Kaminski et al., 1974). There may also be an interaction between building moisture and the emission of chemicals from building material even without microbial growth. One such example is the hydrolysis of phthalates in PVC floor material (HIM Group, 1987), and emission of ammonia and amines (Lundholm et al., 1990) from a particular brand of casein-containing self-levelling mortar in the presence of building moisture.

In a Swedish survey, 50% of the problem buildings ("sick buildings") known by occupational health care centres were affected by either mould growth or water damage (Widström and Norbäck, 1988). In a study of Dutch dwellings, a higher prevalence of respiratory symptoms was found among children or females living in damp houses in comparison with those living in dry homes (Waegemaekers et al., 1989). Similar results were found in a Swedish study, where a relationship between damp houses and hyperreactivity symptoms among children was demonstrated (Andrae et al., 1988). A relationship between SBS symptoms and dwellings with moist casein-containing self-levelling mortar has also been demonstrated. It was also shown that removal of such contaminated mortar resulted in a decrease of SBS symptoms (Andersson et al., 1990).

Other indoor environmental factors

A relationship between enhanced room temperature (above 22–23°C) and both mucosal irritation and general symptoms has been demonstrated (Jaakkola et al., 1989; Norbäck et al., 1990b; Skov et al., 1990). This effect could partly be due to an increased emission of VOC at higher room temperatures (Norbäck et al., 1990b).

The relationship between air humidity and SBS symptoms is more complex. Experimental studies have failed to demonstrate any relationship between the measured and perceived air humidity among normal subjects (Andersen et al., 1973, 1974). In two longitudinal field studies, however, the prevalence of SBS symptoms was shown to decrease at higher air humidities (Menzies et al., 1990; Reinikainen et al., 1990). In addition, individuals wearing contact lenses report more eye irritation at lower air humidity (Breunis et al., 1987). Case reports also indicate that low air humidity may result in dermal symptoms (Rycroft, 1980). On the other hand, there are also negative health effects of high air humidity, including an increased tendency to bronchoconstriction in asthmatics (Aitken et al., 1988) and enhancement of microbial and house dust mite growth.

Experimental studies have shown that infrasounds could induce a drowsy state with EGG changes as well as elevated blood pressure (Landström et al., 1982). In addition, noise may induce annoyance and response may be affected by the attitude to the noise. Few investigators, however, have evaluated the significance of noise or vibrations for the SBS syndrome. In one case report, it is suggested that low-frequency vibration could induce SBS symptoms (Hodgson et al., 1987).

There is some evidence that illumination could play a role in the SBS syndrome. In an office building, a combination of change in type of fluorescent lighting and ventilation improvements resulted in a decrease of eye irritation (Sterling and Sterling, 1983). In two other studies, poor lighting control and lack of daylight illumination were found to be related to a higher prevalence of SBS symptoms (Hedge et al., 1989; Robertson et al., 1989).

Both wall-to-wall carpets (Göthe et al., 1989) and VDU work (Olsen, 1981) may increase the exposure to electrostatic fields, and such fields may increase dust accumulation (Olsen, 1981). Concerning the possible relationship between electrostatic charge and SBS, there is conflicting information in the literature. In some studies, subjects reporting exposure to static electricity also had an increased prevalence of SBS symptoms (Michel et al., 1989; Norbäck and Torgen 1989; Norbäck and Edling, 1991). On the other hand, in one study where the electrostatic potential differences were measured, no such relation was found (Göthe et al., 1989).

Some investigations suggest that light air ions may result in physiological effects, with negative ions associated with well-being (Yates et al., 1986). When evaluated in a blind fashion, however, negative air ions have not been shown to correlate with SBS symptoms (Robertson et al., 1985a; Finnegan et al., 1987b).

19.3.4 General summary and conclusions

Lung cancer from radon and passive smoking are two examples of building-related diseases where both the aetiology and the aetiological fraction are known. For other types of building-related illness with known aetiology, the literature mainly consists of case reports and there is a lack of both descriptive and longitudinal epidemiological investigations.

Available studies, however, show that a fairly large proportion of the population perceives inadequate indoor air quality, particularly in the office environment and among females. There are also indications that poor indoor air quality may affect the productivity of office workers. There are few investigations on the relationships between measured and perceived indoor air quality. It is therefore, difficult to give recommendations on how to create an

indoor environment that is perceived as good. It seems reasonable that epidemiological experience from investigations on the sick building syndrome could be applied also to complaint reactions of poor indoor environment.

Several studies have demonstrated that sick building syndrome is common in the population and is related to several causes. Although recent investigations have shown a relationship between SBS symptoms and the indoor environment, there is still some uncertainty as regards the aetiology of SBS. Despite this uncertainty, the total concentration of volatile hydrocarbons seems to be a simple and useful exposure measure, since it has been empirically shown to be a predictor of symptoms. The finding concerning SBS and dust or carpeting suggests that fleecy materials could act as a depot of organic macromolecular dust that may induce SBS symptoms by its release to the air under certain conditions. Exposure to microorganisms, which can grow in damp houses or be spread by air conditioning systems, appears also to be of importance. Finally, there seems also to be a relation between SBS symptoms and ETS exposure during childhood, the urban environment, work with video display units, and the psychosocial climate of the workplace.

From a preventive point of view, the results suggest that wall-to-wall carpets should be avoided in the indoor environment and the amount of fleecy materials kept at a minimum. Furthermore, building materials, building construction and indoor activities should be selected on the principle that the emission of volatile hydrocarbons should be as low as reasonably achievable. Building moisture should be minimized in order to avoid microbial growth and enhanced chemical emissions. In addition, smoking — and maternal smoking in particular — should be minimized. General ventilation, preferably expressed as CO_2 concentration, should be kept at a reasonable level. Return air should be avoided since it enhances the indoor level of volatile hydrocarbons and other pollutants. In suspected sick buildings, identification and elimination of the sources of air pollution should be preferable to improvements of the general ventilation, which has a limited capacity to reduce unusually high emissions of volatile hydrocarbons. A sound psychosocial climate of the workplace would also have a preventive effect on sick building syndrome.

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Indoor Air Quality
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Chapter 20

Questionnaires on Exposure and Effect Assessment in Buildings

20.1 INTRODUCTION

Perceptual and comfort aspects are essential in building climate control. They involve a complex of thermal, air quality, visual, and auditory sensations. Thus, sensory reactions play a decisive role. These reactions are a synthesis and integration of complex environmental exposures. The measuring device is mainly the sensory systems. Evaluations may be based on measuring the excess incidence or prevalence rates of subjective observations, symptoms, and complaints, or of medically more or less well-defined disorders.

The typical tool for assessing sensory effects in occupied buildings is the questionnaire survey from which dose–response relationships may be constructed. Two main classes of perceptions are at focus. The first and obvious class includes perceptions which the observer attributes to the surrounding physical environment (environmental perceptions). Examples are draught and odour. The second and less obvious class includes perceptions of events inside the body or taking place on the body surface (body perceptions). Examples are perceived eye irritation or dry skin. The observer may or may not be able to attribute the body perceptions causatively to the surrounding physical environment. Commonly, it is much easier to establish dose–response relationships for environmental perceptions than for body perceptions. The latter are to a larger extent determined by unknown observer-related factors.

In a survey of sensory effects usually a representative group of persons is presented questions on perceptions and symptoms with several well-defined response categories. Two principal approaches are common: (a) a battery of questions constituting an attitude scale to obtain, for example, scores of annoyance and discomfort or (b) self-rating questions to obtain direct estimates of perceptions and symptoms. The results of the self-rating questions are treated in different ways depending on whether the aim is to measure the

extensity of effect (e.g., prevalence) or the mean degree of the effect (e.g., symptom strength) in the population. When self-rating questions are chosen, certain general requirements should be satisfied. First, one should openly display the assumptions behind the method of effect assessment. Second, the allowances and constraints of the measurement method should be clarified before it is used for comparing dose–response relationships. Finally, a calibration procedure should be attempted in the measurement.

Surveys of sensory effects are mostly conducted in occupant populations who are chronically exposed to the indoor environment. This circumstance always limits the generalization of results. The responses from exposed individuals may be biased due to the exposure itself, differences in attitudes, and other so-called extra expositional factors. Their response criteria may differ from the criteria of the non-exposed individuals. Similarly, the response criteria for populations exposed, but to a varying degree, may differ. To obtain a comparison between differently exposed populations, the variation of the response criteria must be known and if necessary corrected for.

As mentioned above, questionnaire techniques are often used for measuring both indoor air quality variables (environmental perceptions) and reported symptoms (body perceptions) among occupants in buildings. Usually, a pattern of indoor air perceptions and a pattern of symptoms are desired. A pattern can differ between indoor chemistry and odours: the presence or absence of a chemical substance has been shown to be uncorrelated with the presence or absence of perceived odour. A similar point can be made for the relationship between chemistry and symptom reports: the pattern of presence or absence of chemical substances may exert a joint effect on the health of the occupants. In addition, concentration may be irrelevant for both chemistry and symptom patterns. For instance, substances available in very weak concentrations may nonetheless produce strong odours and, conversely, substances available in strong concentrations may not be sensed at all. A single spike of chemical concentration may be a powerful stimulus for a sensory reaction whereas long-term health effects may result from the continued exposure to low concentrations of toxic substances in the indoor air.

It is clear that pattern representations should be examined together with the concentration for single substances. This means that recognition of patterns (e.g., between sick and healthy buildings) requires that we match patterns obtained in different buildings, decide on the similarity among patterns, and use similarity measures to decide upon the similarity of conditions within different buildings.

20.2 THE QUESTIONNAIRE

Writing questions for questionnaires is not simply an art; a scientific approach should be taken. Some guidelines have emerged from the collective research tradition, as for example presented by Sudman and Bradburn (1982) and Converse and Presser (1986). The basic core of questions is of course that they are formulated with the aid of language. Thus, there is a need for simplicity, intelligibility and clarity. It is imperative that common language should be used, questions should be short, and confusions should be avoided (Sheatsley, 1983).

If the respondent is faced with a task he cannot manage, the responses have low information value. It is believed that it is easier to answer questions bearing on one's own experience and behaviour ("facts") rather than questions on opinions and attitudes ("evaluations"). The latter are assumed to be more open to the respondent's own definition than the former. Thus, it has been repeatedly pointed out by field researchers that it is the respondents who know the full definition of the question.

20.2.1 Recall of the past

Valid descriptive data are obtained from questionnaires that focus on the current, the specific, and the real (Turner and Martin, 1984). Memory questions in general appear to be difficult for the respondent. Recalling an event or behaviour can be especially difficult in several circumstances: (a) if the decision was made almost mindlessly in the first place, (b) if the event was so trivial that people have hardly given it a second thought since, (c) if questions refer to events that happened long ago, and (d) if recall is required of many separate events. Even important events either fade with time or require specific cues to bring them into focus at the time of the survey. Preferably questions should refer to the respondents' perceptions "right now".

Five techniques have been recommended to improve the validity of reporting on past events (Converse and Presser, 1986). (1) Bounded recall addresses the over-report due to "forward telescoping" outside the requested time range. It may be controlled by establishing the baseline in an initial survey. (2) Narrowing the reference period for survey reporting is a good corrective means. In questions related to the Sick Building Syndrome (SBS), it is recommended that they refer to the conditions "right now". (3) Averaging or questions about typical conditions are better than the "single day" focused questions. (4) Landmark events may be referred to instead of specific dates to anchor the timing of other events. The question could ask about a symptom "since Christmas", instead of "during the last month". (5) Cueing means that

cues are provided to help memorizing. The purpose of cues is to stimulate recall by presenting a variety of associations.

20.2.2 Simplicity is the rule, complexity the exception

There are some cases when the simplicity rule of question design does not apply. For example, Factorial surveys use vignettes of "stories" in the study of judgment, decision making, or attribution processes. Factorial surveys have some attractive features. The respondents see the vignettes of concrete, detailed situations on which to make judgements rather than being asked for abstract generalizations. Even though the questions are hypothetical, vignettes reduce the need for respondents to have insight and be conscious of their own thought process. This vignette technique has been conducted with occupational groups as well as with the general public, for example, in studies of annoyance to community noise.

Ranking Scales have a long history in survey research. Alwin and Krosnick (1985) showed that rankings (rank order is given) do not show the same relationships to predictor variables as Ratings (category scale value is given) even though the same factors were investigated. Magnitude Estimation Scales are examples of a third, more complex, technique of responding. Magnitude scaling of annoyance and attitudes has been "calibrated" through numerical estimation and physical line-length estimation of physical stimuli such as light and sound (Berglund et al., 1975; Lodge, 1981). Although these more complex techniques have considerable interest and potential usefulness in survey research, there is much about them that we do not yet understand.

20.2.3 Some other important issues

Standardized measurement is central to survey research. The idea of standardization is to define the measurement instrument in relation to a subpopulation for which it discriminates, for example, the "normal" population or children or asthmatics. The survey instrument may simply be a questionnaire or a questionnaire combined with psychological and physiological tests thus together constituting a test battery. The standardization of questionnaires is most successful if the respondents are only allowed to respond according to the same principles. Therefore questionnaires with closed questions are the easiest to standardize for a subpopulation. A widespread criticism of closed questions is that they force people to choose among offered alternatives instead of responding in their own words. Closed questions given the same response options are more specific than open questions, and therefore more apt to communicate the same frame of reference to all respondents.

The typical survey question incorporates assumptions not only about the nature of what is to be measured, but also about its very existence. The most basic finding from research on question wording is a double-edged one: even small changes in wording can shift the answers of many respondents, but it is frequently difficult to predict in advance whether or not a wording change will have such an effect.

Large-scale surveys are now common in SBS research. In designing a questionnaire, one should consult these earlier data for they are very likely to save time and effort. However, borrowed questions also need pre-testing because the meaning of questions can be affected by the context of neighbouring questions in the questionnaire. The following should be considered in a pre-test: variation, meaning, task difficulty, respondent interest and attention, flow and naturalness of the questionnaire sections, the order of questions, "skip" patterns or internal drop-outs of responses, timing, respondent interest and overall attention, and respondent well-being with the response task (Converse and Presser, 1986).

What is being measured? In questionnaire and physical-chemical surveys of the indoor climate in buildings, it is imperative that the purpose or research problem of the survey will determine in which "measurement form" the results have to appear. There is a difference between (a) measuring a physical or psychological variable and (b) estimating the risk of an outcome; the social scientists usually focus on the former type of results while the health epidemiologists commonly focus on risk estimation (probability of an event).

20.2.4 Measurements

The measurement concept refers to a system of measuring or of measures as such; the act of measuring is often performed by comparison with a standard. Thus, Last (1983) defines measurement as a "procedure of applying a standard scale to a variable or to a set of values". Measurement scales may be characterized with regard to level of measurement, which will put restrictions on potential data analysis (e.g., Torgerson, 1958). The most common data are in the form of a Nominal Scale only (presence/no presence of an occurrence or a set of qualitatively different classes such as different types of symptoms). Sometimes the responses are assessed on Ordinal (rank order), Interval (distance is constant, relative to a zero-point), or Ratio Scales (ratios are constant, absolute zero-point). Physical-chemical measurements are usually interval or ratio scales in contrast to the questionnaire data with self reports that are commonly on nominal or ordinal scales. The causal relationships sought between SBS and the physical-chemical variables may only pertain to rank order causations.

It seems that there is a great need for improving the level of measurement in questionnaire studies. This is particularly true for applications in intervention studies where it is desirable to draw more refined conclusions. For example, after a remedial action it is appropriate to conclude that the indoor concentrations of total volatile organic compounds (TVOCs) have been reduced to e.g., 50% (ratio), but it is usually only permissible to conclude that SBS related symptoms became less (rank order), not how much less.

The grading of scales may have different resolutions for different types of measuring instruments — 'instrument' here defined as the combination of an individual or group of individuals and a particular questionnaire. For perceptual measurements the resolution is related to discrimination capability along the scale. For example, it is expected that older people have lost some of their discriminatory power for sensory stimulation and therefore the resolution of response scales obtained from old persons may be less than for young persons.

20.2.5 Calibration

The procedure for calibrating scales is tied to the level of measurement. Calibration of scales is performed in order to rectify the grading of quantitative measurements obtained. Ordinal scales are dubious to calibrate. Interval scales may be calibrated with regard to a distance between two scale values and ratio scales may be calibrated with regard to one scale value only, respectively. Calibration procedures are imperative in chemical and physical measurements but are relatively new to psychological scales in environmental questionnaires (Berglund et al., 1975). In order to accomplish comparability between scales in behavioural science, the practice has been to standardize scales by transforming the empirical response distributions to a standard distribution of z scores (e.g., Lord and Novick, 1968) rather than to calibrate the scale as such.

20.2.6 Questionnaire data and symptom measurement

With regard to measurement, it is not quite clear what has been accomplished with commonly used SBS questionnaires. Frequency of persons reporting a specific symptom is often used as a graded effect measure of the symptom. Do such frequencies (or percentages) represent a quantification of symptoms in such a way that symptom frequencies may be added in to some SBS index or score? Theoretically, a prerequisite for such a calculation is that all respondents both define each symptom identically and use identical criteria in reporting each symptom. The practical importance of not fulfilling these prerequisites is not clear but in the worst-case situation the SBS scores are

only rank order or cannot be compared at all on the same scale.

In social surveys, frequencies are considered to represent the extent of a symptom in a target population (often defined geographically) rather than the symptom intensity for groups of individuals. Intensity is defined as an attribute or dimension that may be quantified or measured, while extensity refers to the counting of occurrences reported by individuals. A questionnaire may also be used for assessing the perceived severity of symptoms.

20.2.7 Measurement of annoyance/discomfort

By definition, annoyance and discomfort are psychological phenomena. First, they involve mental processing. Second, annoyance and discomfort are believed to start with a perception. In a way the perception can be regarded as the effective dose on which the individual reacts by cognitive and emotional processing. Third, annoyance and discomfort are largely determined by the perceived characteristics of the environmental stimulus. For instance, perceived intensity is one of the most important determinants of degree of annoyance. Fourth, annoyance and discomfort may arise indirectly, for example, from experience of perceived task interference. Fifth, annoyance and discomfort are also related to anger and frustration as well as to feelings of constraint in territoriality and control. Finally, they are largely modified by personal and contextual factors.

In epidemiological studies of self-reported annoyance or discomfort from environmental exposures, the investigator often assumes that one, and only one, relationship exists between reported and the actually experienced annoyance. However, identical expressions of annoyance may mean different things to different populations and groups.

20.3 PERSONS AS MEASURING INSTRUMENTS

In indoor climate questionnaires, persons are used for measuring body perceptions (symptoms) as well as environmental perceptions. A prerequisite for using persons as "measuring instruments" is that we know something about the nature of the sensory systems and humans as measuring instruments. For example, all persons are able to perceive the thermal sensations in a room but only some persons may be able to perceive eye irritation in that same room. A number of psychological factors will have an influence on questionnaire data because they function as modifiers. Human stress is considered to increase the adverse effect of pollutants. Given that stress can reduce host resistance to biological pathogens (e.g., Dubos, 1965), stress may

also affect reactivity to environmental pollutants (e.g., Evans, 1982). Other psychological factors believed to be modifiers of adverse effects are individual evaluation of threat, possibilities for cognitive coping with the exposure situation, as well as possibilities for individual control of the exposure situation. Such psychological factors will contribute to true measurement variance which should not be confused with error variance of measurement. For example, true differences in host sensitivity may show up as an increased measurement variance but not in error variance. This is a fundamental problem in identifying sensitive groups and thus also in causal analysis.

A critical psychological variable in intervention studies is expectancy. The Elton Mayo's Hawthorne studies (reviewed in Roethlisberger and Dickson, 1939) revealed that environmental changes, whether in positive or negative physical direction, resulted in improved work productivity. One theory is that the attention given the workers by the management was the true causal factor. It would be expected that any environmental change will improve the outcome for psychological reasons, at least from a short-term perspective (cf. Griffith and Raw, 1987). In building intervention studies, the knowledge of change accompanied with expectations will become strong intervening variables which have to be controlled.

20.4 RELIABILITY, VALIDITY AND QUALITY ASSURANCE

One problematic point is that the measurement power of SBS and indoors climate questionnaires seems not yet to have been discussed. Is it possible to quantify symptoms with a questionnaire? Is the instrument, the questionnaire, sensitive and specific enough for the measurement task? These questions relate to the reliability and validity of the questionnaire as well as the quality assurance of questionnaire field studies.

The reliability of a measurement refers to how well it measures what it measures (repeatability). The reliability is usually expressed as a reliability coefficient which is the proportion of obtained variance that is due to true variance in variables being measured. A number of practical methods for determining reliability are used: (a) test-retest method, i.e., the same measuring instrument is applied on two occasions to the same sample of individuals; (b) parallel-forms method, i.e., equivalent forms of a test are administered to the same group of subjects; (c) split-half method, i.e, equivalent forms of a test are administered to the same group of subjects; and (d) internal-consistency methods, i.e., the method requires a knowledge of certain test-item statistics, for example regarding dichotomous scoring.

The validity of the measurement refers to how well it measures what it

intends to measure. There are two kinds of validity. Empirical validity is defined as the degree of association between the measurement and some other observable measurements, for example, eye irritation reports from a questionnaire and clinically diagnosed eye irritation (Franck and Skov, 1991). A second kind of validity is theoretical validity expressed as the correlation of an observed variable (symptom frequency) with some theoretical construct of interest (a specific definition of SBS among a variety of suggestions). The difficulty is that the criterion, the construct (the SBS) is not directly measurable (but the symptoms may be).

The principles of quality assurance have been discussed by the World Health Organization (WHO, 1983) with regard to public health and the scientific community. This concept refers to all the steps that should be taken by the researcher to ensure that the research findings are of good quality; quality assurance is accomplished by adequate self-evaluative and self-corrective strategies. Thus, quality assurance covers the utilization of scientifically and technically sound practices for the collection, transport and storage of samples, the laboratory analysis, as well as the recording, reporting, and interpretation of results. More specifically, for questionnaire studies quality control has two components. One is external quality control which is a system for objectively checking investigation performance by an external group. The other is internal quality control which is a set of procedures used by the staff or the investigating group for continuously assessing results as they are produced in order to decide whether they are of good enough quality to be released.

20.5 QUESTIONNAIRES IN INVESTIGATIONS OF PROBLEM BUILDINGS

As recommended by the World Health Organization (WHO, 1986) and the European Concerted Action 613 (Molina et al., 1989), it is important to conduct investigations of problem buildings in a systematic stepwise way. The reasons behind the stepwise procedure are cost-effectiveness as well as the belief that simple and immediate actions are basic elements in the good management of buildings with SBS problems. Questionnaires are recommended for the first initial step as well as for the final step of such investigations. The first step includes a technical survey and the use of a simple questionnaire. This concerns symptoms and complaints about different factors and is distributed to selected samples (preferably random) of employees. If all intermediate investigation steps fail to give enough information for the solution of the problem, the investigation might eventually in the final step include medical

investigations, specific measurements of suspected components, and a detailed questionnaire. The latter should explore in detail the symptoms reported and should ask questions about the psychosocial conditions at work, the relationships of individuals to their colleagues and superiors, and the type of work they are performing.

Questionnaires are probably the most useful for carrying out epidemiological investigations of indoor-related health problems. A set of core questions could be used in all questionnaires on IAQ, e.g., on occupation, gender, age, descriptors of work environment, smoking history, critical symptoms and working conditions (job satisfaction). An agreement on standardization seems possible, but is not at hand yet. This is also true for the different ways the questions may be asked. For instance in determining work-relatedness of symptoms there are different options:

1. do you think the symptom is due to your work environment? or
2. is the symptom better when you are away from work? or
3. do you have the symptom at work?

There is a general agreement on which are the majority of the principal symptoms related to indoor climate problems. There is discussion on the secondary symptoms and the descriptors. For instance "irritated, stuffy or runny nose" could be divided into questions on "irritated nose", "stuffy nose" (common symptom) and "runny nose" (less common, implies allergic rhinitis or infection). Secondary symptoms such as dizziness are usually not present alone but are adjunct to the major ones of concern.

The frequency of the symptoms should be asked for, but the format is not generally agreed, (e.g. daily, weekly, monthly, or often, sometimes, never). Most studies are designed to obtain a prevalence over a particular period of time. The reference period should be sufficiently short to prevent recall bias, but should be long enough to take into account seasonal differences. One method to account for seasonality would be to repeat the questionnaire at different times in the year.

The choices in questionnaire administration usually considered are: the questionnaires could be self — or interviewer — administered; they could be written, computer-based or mailed. Mailed administration of questionnaires is still the method of choice for large population studies. Response rate is the key to a successful study. Computerized administration with subject direct entry is becoming increasingly popular because of ease of administration, increased speed in response, and no data entry leading to decreased error.

Reference values connected with specific questionnaires have been developed in some countries for the general population and offices. These reference values are unfortunately not transferable across cultures on types of questionnaires. Since reference population data which are comparable between coun-

tries and questionnaire are not available, control groups are essential in most investigations. An example of a commonly used questionnaire is given in the Appendix.

A questionnaire that might be useful to develop, is a building checklist that could be administered to the building maintenance personnel and those responsible for the building. As for all types of questionnaires there is a need for intercalibration between methods and investigators, or, at the least, a standardization of the most commonly used questions and response alternatives.

Recommendations

1. The assessment of the health and comfort problems of a building should be based on either the whole working group or random selection of workers stratified by facing, floor and distance from the window and not on volunteer studies.
2. Simplicity in the questionnaires is essential; interlinked, complex or multiple-part questions should be avoided.
3. Researchers are strongly recommended to use a validated questionnaire before making up new questions.
4. Control groups and control buildings should always be included in the study design.
5. The question of building- or work-relatedness of symptoms should be specifically addressed even though a unique format is not agreed upon.
6. There is a strong need for developing an intercalibration procedure of questionnaire responses, and for a standardization of at least a core of questions typically used and empirically being found usable. As in all other building investigations, procedures for quality assurance should be used regarding design, data collection and data treatment.

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Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 21

Diagnosis of Building-Related Illnesses and "Sick Building Syndrome"[1]

Building-related illnesses fall into the following categories:

- Upper respiratory tract disease
- Asthma
- Hypersensitivity pneumonitis(extrinsic allergic alveolitis)
- Humidifier fever
- Toxic reactions
- Infectious diseases — viral, bacterial, fungal
- Dermatitis
- Sick Building Syndrome
- Nonspecific environmental hypersusceptibility
- Sensory effects and other effects on the nervous system
- Cancer and effects on reproduction.

21.1 INTRODUCTION

The ultimate goal of indoor climate investigations is the optimal health of the occupants, as defined by the WHO. The health consequences of indoor climate may affect an individual or be epidemic among groups of individuals.

The diagnosis of indoor climate health effects should primarily be based on patient history and clinical investigation. The physician should be trained and aware of the possibility that IAQ factors may be the cause for illnesses and complaints, and be aware of the most probable manifestations of different aetiological factors.

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1 A part of the text of this chapter has been derived from NATO/CCMS Pilot Study on Indoor Air Quality 4th Plenary Meeting "Epidemiology and Medical Management of Building-Related Complaints and Illnesses", Eds. F. Levy and M. Maroni, Oslo, Norway, 19–21 August 1991; ECA (European Concerted Action "Indoor Air Quality and Its Impact on Man", COST Project 613), 1989. Sick building syndrome — a practical guide. Report No. 4 (EUR 12294 EN). Luxembourg: Office for Publications of the European Communities; Chemical and Environmental Science Vol. 4 Chemical, Microbiological, Health and Comfort Aspects of Indoor Air Quality — State of the Art in Sick Building Syndrome (1992), Eds.: H. Knöppel and P. Wolkoff.

TABLE 21.1

Factors to address when examining the patient

Complete history and physical examination
Objective findings of a recognized disease pattern — rhinitis, bronchitis, asthma, hypersensitivity pneumonitis, dermatitis
Associated conditions (e.g. mucous membrane irritation), complicating factors (i.e. concomitant problems)
Emphasis on pre-existing conditions (e.g. atopic predisposition, asthma, etc.)
Note exact history of onset of symptoms and relationship with building environment, including medical condition when away from the building, any time pattern that the onset of symptoms exhibit
Personal habits which may influence symptoms (e.g. smoking)
Recognition of psychosocial component of tight building complaints and other complaints involving non-specific symptoms: comments about unpleasant working conditions, job dissatisfaction and other stressors should be recorded

The above may be supported by the use of a questionnaires.

The complete history and physical examination should address the factors listed in Table 21.1.

Table 21.2 shows the clinical tests available for the diagnosis of building related illnesses.

21.2 UPPER RESPIRATORY TRACT DISEASES

The upper respiratory tract is frequently affected by indoor air contaminants or alteration in air quality. This is readily understandable if the anatomy is considered. Nasal and pharyngeal mucosa as well as the larynx may be affected either directly or through an allergic mechanism. Clear anatomical or only functional changes may occur. Rhinitis is a very frequently observed picture. When allergic in origin it is often accompanied by sinusitis and conjunctivitis. In fact patients with allergic rhinitis and sinusitis commonly present with ocular irritation, sinus fullness, headache, nasal congestion, sneezing, watery rhinorrhea, dry throat, non-productive cough, difficulty wearing contact lenses and itching tearing and soreness of the eyes often coupled with gelatinous conjunctival discharge in the mornings, reduced sense of well-being with irritability and fatigue. Allergic rhinitis–sinusitis may be complicated by nasal polyposis and/or mucocyst development in the sinuses. Predisposing factors are personal and family history of atopic diseases.

TABLE 21.2

Clinical tests available for the diagnosis of BRI

Pulmonary tests	Pulmonary function tests
	– lung volumes (FVC)
	– spirometry (FEV1
	– diffusing capacity
	Chest X-rays
Haematological tests	Anaemia (haemoglobin)
	Eosinophilia
	Leukocytosis
	Elevated erythrocyte sedimentation rate
Immunological studies	Direct challenge tests
	– skin prick test, patch test
	– intradermal delayed hypersensitivity skin test
	– inhalation challenge test
	Binder-ligand assays: RAST
	Immunodiffusion
	– double diffusion in agar
	– single radial diffusion
	Antigen-induced leukocyte histamine release assay
	Immunoelectrophoresis
	Agglutination reactions
	Complement fixation
	Tryptase test
Exposure tests	Challenge studies
	– single subject exposure: mask
	– single subject exposure: chamber
	– multiple subjects exposure: chamber
Histologic studies	Microscopically observable features
	Immunofluorescent methods
	Bronchoalveolar lavage
	Nasal washings
Biological tests	See Chapter 3.6

TABLE 21.3

Allergic rhinitis–sinusitis and conjunctivitis

Symptoms	ocular irritation, sinus fullness, headache, nasal congestion, sneezing, watery rhinorrhea, dry throat, non-productive cough, difficulty wearing contact lenses, itching
	allergic rhinitis-sinusitis may be followed by nasal polyposes
	less common complaints: tearing and soreness of eyes coupled with gelatinous conjunctival discharge in the mornings, loss of well-being with irritability, fatigue, depression
	predisposing factors are personal history of other atopic diseases, family history of atopic diseases
Physical findings	Rhinoscopy: pale, bluish edematous nasal turbinates coated with thin, clear secretions, nasal membrane swelling, conjunctival injection and edema
Laboratory tests:	direct skin testing (scratch, prick or intradermal): positive wheal and erythema responses to skin testing with allergen, (evidence of mostly IgE-mediated reaction)
	nasal washing show a prominent increase in eosinophils, and is useful for making a distinction between infectious and non-infectious rhinitis
	total IgE levels frequently elevated, but for etiologic diagnosis antigen specificity for IgE needs to be demonstrated by radioallergosorbent test (RAST), or by enzyme-linked immunosorbent assay (ELISA). To minimise problem of false positive results, diagnosis should rest not only on skin test results or IgE levels but on correlation of clinical history with skin reactivity
	findings on challenge tests: nasal challenge can elicit symptoms
Differential diagnosis:	NARES (non-allergic rhinitis with eosinophilia) — normal serum IgE levels, tendency to respond well only to topical corticosteroid therapy
	non-allergic rhinitis with no eosinophilia (vasomotor rhinitis) — persistent symptoms without correlation to specific allergen exposure: symptoms elicited by temperature or humidity changes, emotions
	rhinitis medicamentosa — rebound effects of adrenergic nose drops or sprays, or side effect of various drugs
	infectious rhinitis — thick purulent nasal discharge contains many neutrophils, as opposed to eosinophils
	many chemicals are mast cell degranulators or histamine liberators, symptoms similar to allergic causes

Differential diagnosis includes non-allergic rhinitis with eosinophilia (NARES) which is characterised by increased eosinophils in nasal secretions, normal IgE levels, and a good response to topical corticosteroid therapy and minimally to antihistamines. Non-allergic rhinitis without eosinophils in the nasal secretions (vasomotor rhinitis) presents with persistent symptoms without correlation to specific allergen exposure: symptoms are usually elicited by

temperature, humidity changes, or emotions and not to allergens. Infectious rhinitis is marked by thick purulent nasal discharge. Many chemicals are mast cell degranulators or histamine liberators, eliciting symptoms similar to those which are allergy induced (Table 21.3).

21.3 ASTHMA

Asthma (see Tables 21.4 and 21.5) can be defined as reversible airway obstruction characterised by bronchial reactivity and associated airways inflammation. In allergic asthma, there may be immediate or late immunological responses of the bronchus while in non-allergic asthma, non-specific bronchial irritability is the basis.

Occupational asthma is rare in office and similar buildings. The source of allergen may be external to the building and drawn in via the ventilation system (Carroll et al., 1976), or in poorly maintained buildings the source of allergen may be from the ventilation system itself — a contaminated cold water spray humidifier (Finnegan and Pickering, 1984) or from building materials (Cernelc, 1982).

The diagnosis is initially suggested by a history of respiratory symptoms (cough, wheeze or breathlessness) improving on days away from work and on holidays. Individual validation of their symptoms can usually be obtained from serial measurements of peak expiratory flow rate. This method is well established in occupational asthma, it is specific and has a sensitivity of 50–80% depending on the amount of treatment that is being taken by the patient at the time. The sensitivity is reduced from 80% taking inhaled bronchodilators alone to 50% when inhaled steroids are introduced. In a study of 15 workers with occupational asthma caused by contaminated humidifiers (Burge et al., 1985) described four clinical patterns of asthma: worst asthma on the first day of the working week, equivalent daily deteriorations, progressive deterioration over the week and worst midweek. The subset of asthmatics with progressive deterioration over the week were found to have a high atopic status and the majority had positive skin prick tests to an extract removed from the humidifier.

Critical to the establishment of occupational asthma and important to indoor air quality causes of asthma is the presence of increased methacholine/histamine responsiveness following a period of time within the suspected environment.

Identification of the causative allergen may be assisted by skin testing or by the identification of specific IgE using a RAST or ELISA technique.

Management involves the removal or control of the allergen source. If the source is microbiological pollution within the building then improved mainte-

TABLE 21.4

Asthma

Definition:	reversible airway obstruction characterized by hyperirritability of the bronchial mucosa, allergic asthma is a manifestation of type I allergy localized in the bronchus, bronchial irritability from non-allergenic causes may be the primary state
Pattern of asthmatic reaction:	immediate, late or dual immediate: develops within minutes of challenge, short duration, readily reversed by bronchodilators, can be prevented by beta-adrenergic stimulants, disodium cromoglycate but not corticosteroids late: develops after 2–3 h of challenge, resolves within 24–36 h, long duration, lesser response to bronchodilators than immediate reaction, can be inhibited by prior administration of corticosteroids, followed by increase in nonspecific bronchial reactivity
Symptoms	dyspnea, cough, wheezing, tightness in the chest, fatigue (due to increase in respiratory work)
Physical findings	depends on severity of asthma moderate attack: expiratory wheezes, scattered, changing on coughing, viscid mucus secretion, on auscultation rhonchi are widespread with severe attack there is use of accessory muscles of expiration, cyanosis, tachypnea, pulsus paradoxus
Laboratory evaluation	haematology: increased eosinophil count in about 50% of asthmatics increased WBC count with infections chest X-ray may be normal, indicate hyperventilation state (radiolucency), or complications e.g. spontaneous pneumothorax, mediastinal emphysema lung function tests: shows airway obstructive disease (drop in FEV1, FVC, MMEFR; TLC may be increased) PEFR is a convenient way of monitoring asthma, and can be done by the patient at home or suspected building environment for diagnosis and management skin testing: positive wheal and erythema reaction to appropriate allergen in allergic subjects reversibility test (bronchodilator response) positive exercise test — positive in about 80% methacholine/histamine test positive (a test of bronchial hyperresponsiveness) challenge test: challenge with appropriate allergen/irritant can reproduce symptoms immunological tests: total serum IgE concentration is frequently elevated but is sometimes normal

TABLE 21.5

Asthma related to work environment

Can be allergic or non-allergic (irritant) in nature.

Certain irritants may also act as allergens to susceptible individuals; therefore underlying mechanism can involve direct irritation and/or immunologic processes, ie. IgE or IgG

Causative agents can be divided into groups according to mechanism of sensitization:

1. *High Molecular Weight (HMW) compounds*: cause production of specific IgE antibodies

– positive immediate skin reaction

– antigen specific IgE antibodies (RAST or ELISA)

– atopy is predisposing factor

Examples:

– biologic enzymes (in detergent enzyme industries)

– products of laboratory animals (animal handlers: laboratory workers, farmers, veterinarians, furriers, breeders, etc.)

– psyllium, antibiotics (health professionals)

– mites (grain and flour handlers, granary workers, bakers)

2. *Low Molecular Weight (LMW) compounds*: mechanism responsible for asthmatic reaction not yet fully understood, proposed mechanisms include the LMW compound acting as a hapten and coupling to proteins of the body

– skin testing using the LMW compound is usually not helpful

– specific IgE antibodies to protein conjugate of the LMW compound found only in small proportion of patients

– presence of eosinophilia

Examples:

– acid anhydrides such as trimellitic anhydride, isocyanates, or organic dusts such as western red cedar and white cedar (plicatic acid)

Differential diagnosis (relevant to IAQ):

Bronchitis (acute and chronic) — cough with expectoration, for chronic bronchitis: on most days, for at least 3 months of the year, for at least 2 consecutive years

– blood eosinophilia not typical

– daily PEFR stable, in asthma it is variable

– minimally reversible, responds minimally to corticosteroids

chronic obstructive pulmonary disease (COPD) — progressive breathlessness

– no blood eosinophilia, unlike in asthma

– diffusing capacity for CO decreased, unlike in asthma

– non-reversible, does not respond well to corticosteroids

nance and cleaning may be required, if the source is from outside the building, for example, from a local factory, then source control may be more difficult.

Early identification of building related or occupational asthma is important since the longer a symptomatic individual is exposed to a causative allergen the more likely he is to remain permanently disabled when the allergen is removed.

21.4 HYPERSENSITIVITY PNEUMONITIS (EXTRINSIC ALLERGIC ALVEOLITIS)

Extrinsic allergic alveolitis is an inflammatory reaction of the lungs featured by alveolar filling and interstitial inflammation resulting from intense or prolonged exposure to finely dispersed organic dusts of appropriate particle size. There are three forms of clinical presentation including acute, subacute and insidious. The acute form is manifest as fever, headache, malaise, lethargy, chills, cough, dyspnoea, sputum production, anorexia and weight loss. There may be a temporal correlation of symptoms with the building environment in which case symptoms will begin 4–6 h after exposure begins, with the patient being asymptomatic between attacks. The subacute form appears over a period of weeks and is characterised by cough and dyspnoea, with intermittent episodes of acute illness. The insidious form is generally one of the gradual progression of symptoms with possible intermittent acute episodes and progressive dyspnoea. The CXR may be normal, reveal reticulonodular changes in the early stages of the disease or patchy pneumonitis. Bronchial challenge tests with the relevant allergens reproduces the disease. Pulmonary function tests demonstrate a restrictive defect and impairment of gas transfer. The pulmonary inflammation may lead to pulmonary fibrosis with permanent lung damage and associated disability.

Extrinsic allergic alveolitis was first associated with an air- conditioning system in 1970 (Banaszak et al., 1970). Four of the twenty-seven office workers developed symptoms of allergic alveolitis, investigations incriminated thermophilic actinomycetes contaminating the building's central air-conditioning system. The diagnosis was confirmed by a combination of bronchial provocation and immunological testing and a lung biopsy. There have been several further reports of allergic alveolitis in the office (Arnow et al., 1978) and the home (Sweet et al., 1971; Fink et al., 1971; Tourville et al., 1972; Hodges et al., 1974; Marinkovitch and Hill, 1975), all associated with various types of ventilation systems. Thermotolerant organisms have been implicated in nearly every instance. The potential scale of the problem is well demonstrated in an epidemiological study of 93 suspect homes in the USA. Positive cultures

for thermophilic actinomycetes were found in 74% of the suspect homes (Banaszak et al., 1974). In the UK, this type of problem is rare, two cases (Robertson et al., 1985b) have been reported due to a contaminated cold-water spray humidifier. In this report the humidifier was operating at 15°C, bronchial provocation studies were negative to thermophilic organisms but positive to a crude extract from the humidifier. The specific cause of the alveolitis was not identified.

Water-based structures within buildings may also become contaminated with organisms and induce an allergic alveolitis. Some examples include a sauna — *Pullularia* (Metzger et al., 1976), a hot-tub room — *Cladosporium cladosporoides* (Jacobs et al., 1986), faulty central heating — Penicillium species (Fergusson et al., 1984), from "water features" in an indoor swimming pool — endotoxin (Rose et al., 1991), tap water (Muittari et al., 1980) and a vacuum ring pump (Friend et al., 1977).

21.5 HUMIDIFIER FEVER

Humidifier fever is an influenzal type illness characterised by headache, myalgia, lethargy, fever and shortness of breath occurring on the first day back at work after a break, usually resolving over 24 h and not recurring despite continued exposure to their polluted environment until after a further period away from work. It was first described in 1959 (Pestalozzi, 1959) in a group of workers in a carpentry shop. Twelve out of 17 workers developed a febrile illness with cough and breathlessness, occurring 8–12 h after starting work and resolving over 24 h. These symptoms only occurred on the first day of the working week with no recurrence of symptoms until after a period away from work such as a weekend or holiday. Mould growth within contaminated humidifiers was considered to be the cause. There were no further reports until 1969 (Her Majesty's Chief Inspector, 1969) when an outbreak of similar symptoms was described in a group of printers in a workshop with contaminated spinning disc humidifiers. Subsequently a number of outbreaks of humidifier fever have been described (Pickering et al., 1976; Parrott and Blythe, 1980; Rylander et al., 1978; Newman Taylor et al., 1978; Campbell et al., 1979; Anderson et al., 1989). Apart from the symptom periodicity, the clinical and physiological findings are similar to an allergic alveolitis but the chest radiograph remains normal. This difference between two essentially similar conditions may reflect exposure to a soluble antigen in humidifier fever and to a particulate antigen in allergic alveolitis. In a number of outbreaks of respiratory disease (Rose et al., 1991; Muittari et al., 1980; Friend et al., 1977) described as examples of an allergic alveolitis, the exposure has been to water

droplets, thermophilic organisms have not been identified and the chest radiographs have been normal.

The specific cause(s) of humidifier fever is/are not known. However since inhalation challenge tests with water from the contaminated source reproduces the disease in affected workers (Friend et al., 1977) the antigen is contained in the water. There are several postulated causes including protozoa (Edwards et al., 1976), *bacillus subtilis* (Parrott and Blythe,1980) and endotoxin (Rylander et al., 1978). These postulated causes have been based on serological testing and circumstantial evidence only. In a study of the sera of 119 workers exposed to air from contaminated humidification systems, 25 had humidifier fever and 6 occupational asthma, no correlation was found between the presence of antibodies to the amoebae, *Acanthamoeba polyphaga* and *Naegleria gruberi* and disease (Finnegan et al., 1987a). Pickering et al. (1976) in the course of an investigation of an outbreak of humidifier fever in a group of printers carried out inhalation challenge tests with extracts of organisms to which the subjects had demonstrable precipitating antibodies. They were unable to produce any systemic symptoms or pulmonary function changes on challenge testing with these extracts. The available evidence suggests that serological testing is not a reliable method of determining the cause of this disease. In a study of a large group of works (Finnegan et al., 1985) exposed to heavily and moderately contaminated humidifiers the two major factors influencing the development of precipitating antibodies were the duration of exposure and the smoking status of the worker. A strong inverse relationship between current smoking and the detection of precipitating antibodies was shown. The effect of smoking appeared to be lost within 3 years of smoking cessation. No relationship between the detection of antibodies and the presence of disease was shown.

In general, outbreaks occur when there is heavy microbial contamination of the water source and poor maintenance and design are usually implicated.

Unlike cases of allergic alveolitis, subjects with humidifier fever appear to make a full recovery when they cease exposure to the source of contamination.

21.6 TOXIC REACTIONS

This is a more poorly understood type of response to microbial contamination of indoor air. Microorganisms produce secondary metabolites, these include mycotoxins and a large number of different types of volatile organic compounds (VOC).

Mycotoxins have a low volatility and they are likely to be inhaled absorbed on dust particles or in spores or mycelia. Little is known of their possible role

in producing symptoms within buildings. Croft et al. (1986) describe the heavy infestation of a Chicago suburban house by *Stachybotrys atra*. The family of five had, over a period of 5 years, experienced a variety of complaints including recurrent colds and flu, sore throats, diarrhoea, headaches, fatigue, dermatitis, focal alopecia and general malaise. After removal of contaminated duct work and ceiling materials the family's symptoms resolved. Some of these symptoms are similar to those which may be experienced in building sickness syndrome. Nexo et al. (1983) describe building related symptoms in a group of 5 of 12 office workers. The symptoms they were experiencing included extreme fatigue and eye and upper respiratory tract irritation. The carpet in their workplace was found to be heavily contaminated with microorganisms. The organisms isolated included massive growths of *Aspergillus* spp., *Penicillium* spp., *Aureobasidium pullulans, Rhodotorula rubra* and yeasts. Peak flow records in 2 subjects demonstrated a work-related decrease in peak flow recordings in one. Both subjects' peak flow recordings improved after cleaning of the carpet had been instituted and all work-related symptoms resolved.

21.7 INFECTIOUS DISEASES

Infections associated with indoor air quality problems can be classified into two categories; those whose transmission is only facilitated by indoor air circulation and those that live and grow under indoor air conditions. The symptoms depend on the type of infection with the most common being respiratory infections.

21.7.1 Viral infections

Commonly individuals in mechanically ventilated buildings complain of the frequency that they experience upper respiratory tract infections. This occurs presumably by person to person contact in large open plan offices.

There is, in addition, evidence that viruses may be circulated by the ventilation system itself. A study of the spread of measles in an elementary school, where the source case infected two children in their own classroom and 26 children in 13 other classrooms, suggested that the mode of spread was via the school's ventilation system (Riley et al., 1978). A more recent investigation (Brundage et al., 1988) of acute febrile respiratory disease amongst army recruits in the USA again implicated the ventilation system in the spread of disease. The army trainees were located in either recently constructed, well-insulated, mechanically ventilated barracks or in older-style naturally ventilated barracks. The living accommodation was similar in both types of barrack

with platoon-sized open bays for up to 55 trainees. The rates of hospital admissions with acute respiratory disease were significantly greater amongst trainees accommodated in the modern barracks compared to those living in the old style barracks adjusted relative risk estimate, 1.51 (95% confidence interval, 1.46 to 1.56). Subsequently the risk of acute respiratory disease amongst trainees in modern barracks was significantly reduced by immunoprophylaxis (adenovirus vaccine). This study suggests that the type of ventilation may have a more important role in the transmission of viral infections than is at present recognised.

21.7.2 Bacterial infections

Another example of transmission via a ventilation system of a bacteria was described in 1965 (Honk et al., 1968). A single sailor amongst a crew of 350 individuals was noted to have converted his tuberculin skin test from negative to positive. His chest x-ray was normal and he was not given anti-tuberculous chemotherapy. He developed respiratory symptoms ten months later which were not investigated for a further six months when he was diagnosed as having sputum smear positive pulmonary tuberculosis. The remainder of the crew were studied and 140 members were found to have converted their tuberculin skin test. The distribution of affected crew members around the vessel and their minimal or absent contact with the source case implicated the ventilation system in the spread of the disease.

Subsequently the bacteria, *Legionella pneumophila*, has been widely implicated in causing epidemics of both Legionnaires' Disease and Pontiac Fever, the spread of the infection occurring either by infected water droplets in the incoming air or by contamination of internal fittings for example shower heads or jacuzzis.

Legionnaires' Disease is an illness principally characterised by pneumonia. The incubation period between exposure to the organism and the development of symptoms ranges between 2 and 10 days. The onset is abrupt with a high fever, headache, shivering and muscle pains, followed by a nonproductive cough and usually breathlessness. About 30% of patients develop gastrointestinal upset with diarrhoea and vomiting and about a half become confused. The fatality rate is around 10%. Person-to-person spread has not been described.

This building-related infection was first described in 1976 (Fraser et al., 1977), following an epidemic of pneumonia amongst a group of American Legion conventioneers. The pneumonia occurred not only amongst the conventioneers but also in those walking in the street alongside the hotel (Broad Street pneumonia). The source of the Legionella bacteria was not identified at the time but in the light of subsequent knowledge it is likely to have arisen

from the hotel's air-conditioning system. Two years later, in 1978 (Dondero et al., 1980), the airborne spread of this bacterium via a ventilation system was clearly demonstrated in a hospital outbreak in America. A flash flood affecting the basement of the hospital closed down the cooling towers supplying the ventilation system and an auxiliary cooling tower, unserviced for 2 years, automatically came into operation. This cooling tower was downwind and in close proximity to two of the air intakes of the air-conditioning system. The tower was operational for 30 days and the first cases of Legionnaires' Disease presented 3 days after this cooling tower commenced functioning. Patients, visitors and staff occupying rooms supplied by air from these air intakes were more likely to develop the disease than those receiving air from air intakes located elsewhere in the building. The importance of the location of cooling tower in relationship to the air intakes of ventilation systems has been amply demonstrated. Thirteen cases of Legionnaires' Disease occurred in a hotel complex (Band et al., 1981), in this instance infected air from a nearby cooling tower being drawn down a chimney supplying a meeting room in the hotel. A further study described 68 cases amongst hospital patients in a new district hospital (Badenoch, 1986, 1987) where the cooling tower was located above the air intake to the air-conditioning system.

The passive ingress of infected droplets into domestic dwellings may also occur (Report of an ad-hoc committee,1986). An epidemic of 33 cases of Legionnaires' Disease occurring in a city suburb, the patients living around a factory with an infected cooling tower. Cases occurred downwind of this cooling tower up to a distance of 1700 m and they included individuals confined to their homes by cardiac disease who appeared to have been infected through sitting by an open window. A further study in the same city (Bhopal et al., 1991) examined the places of residence of community-acquired, non-travel, non-outbreak cases of Legionnaires' Disease. They demonstrated a non-linear dose–response, with a relative risk of more than 3.0 in those living 0.5–0.75 km from one.

Water storage systems within buildings have also been implicated as sources of this bacterium causing disease, not only in a hospital situation with immunosuppressed patients (Tobin et al., 1980) but also in hotels with apparently healthy people (Tobin et al., 1981).

The pneumonia is commoner in males than females. At risk individuals include smokers, alcoholics and those with cancer, diabetes, renal disease and chronic respiratory disease.

The treatment of Legionnaires' Disease includes antibiotic therapy with erythromycin and/or rifampicin.

The diagnosis of Legionnaires' Disease is made by the measurement of specific antibody in the blood (these may take some weeks to be measurable)

or by culturing the organism from sputum, bronchial washings or lung tissue.

The most dramatic outbreak of disease due to airborne *L. pneumophila* within a building occurred in 1968 (Glick et al., 1978). in the Health Department in Pontiac, USA, when 95% of employees developed a benign, self-limiting, febrile illness subsequently to be named Pontiac Fever. At the time of this investigation an unidentified bacteria was isolated from the air-conditioning system which some years later was identified as *L. pneumophila*. As is seen so frequently in outbreaks related to Legionella-design, structural and maintenance faults were identified in this building.

At least 12 serogroups of *Legionella pneumophila* have been identified. *Legionella pneumophila* serogroup 1 is most commonly associated with Legionnaires' Disease but other serogroups of *L. pneumophila* and other species of *Legionella* have been identified causing disease in man. *L. pneumophila* is frequently present in the water systems of hotels and hospitals. However subtyping the *L. pneumophila* serogroup 1 using a monoclonal antibody system has shown that most of these organisms only rarely cause disease in man (Watkins et al., 1985).

Various environmental conditions favour the growth of this organism including the presence of corrosion, water stagnation, temperatures of 20–40°C and organic contamination.

21.7.3 Fungal infections

Fungi can become an IAQ problem because of their universal presence indoors. The most common indoor moulds are *Aspergillus*, *Penicillium*, *Cladosporium* and *Alternaria*. There is mounting evidence that respiratory symptoms in children may result from mould exposure in the home.

Fungal infections in man related to buildings have in almost all instances involved hospital buildings and usually immunocompromised patients. The fungal species most frequently implicated is *Aspergillus* either contaminating incoming air to the ventilation unit or the ducts of the ventilation unit itself. Over a 6- month period in a North Carolina hospital (Sarrubbi et al., 1982), while construction work was being carried out in the vicinity of the ventilation air intake, there was a 6-fold increase in sputum isolation of *Aspergillus flavus*. Investigation of the ventilation unit revealed *Aspergillus* growing on the external louvres and defective filters within the unit.

The most serious fungal infections are most likely to occur in immunocompromised patients. This has been well described in a renal transplant unit (Kyriakides et al., 1976), where over a 5-month period three renal transplant patients developed infections with *A. fumigatus*. It was found that the external grill of one of three ducts supplying the unit had corroded and fallen off,

allowing contamination of the ducting itself with pigeon droppings from which Aspergillus species were cultured. The fan supplying this duct was malfunctioning leading to reversed airflow down the contaminated duct and the dissemination of Aspergillus into the transplant unit.

Other important fungal infections known to be transmitted by air include histoplasmosis, coccidiomycosis, blastomycosis and cryptococcosis. All cause systemic disease in unselected exposed populations.

Table 21.6 summarises the laboratory tests available for the diagnosis of illnesses caused by biological agents.

TABLE 21.6

Laboratory tests available for illnesses caused by biological agents

Disease	Organisms or agents	Laboratory tests	
		Patient	Environment
Infections	Viruses: Common cold, Measles, Influenza, Psittacosis Bacteria: Tuberculosis, Staphylococcal Fungi: Cryptococcosis	Tests as recommended in infectious disease textbooks; increase in specific antibodies; smears and culture for bacteria from patients	Not indicated
	Legionella sp. (Legionella pneumonia, Pontiac fever)	Cultivation: Respiratory secretions [sputum, trans-tracheal aspiration (TTA), broncho-alveolar lavage (BAL)]. (Transported at 4°C in sterile containers, protected from drying). NB: Saline should not be used in collection fluids. Serology: May be useful to identify an outbreak. (Retro- spective diagnosis in patients: Acute and con-valescent-phase sera) (*L. pneumophila*: 14 serogroups)	Cultivation from suspected sources (cooling tower, shower fittings etc.) indicated if >1 case or if the patient has not been abroad recently. Isolation of *Legionella* sp. is not a proof that the source is identified. Typing of strains isolated from patient and the environment is necessary.
Aspergillus	*Pseudomonas*, etc;	Mainly immunocompromised patients, hospitals. Diagnosis: Blood culture, culture of respiratory secretions, etc.	

(continued)

TABLE 21.6 (*continuation*)

Disease	Organisms or agents	Laboratory tests	
		Patient	Environment
Asthma, rhinitis, etc.	Moulds (pollen), algae animal products mites, insect debris, cockroach, etc.	Cultivation: not indicated. Serology: IgE: Total IgE. Specific IgE (RIST, RAST, Elisa) Skin prick tests	Cultivation: As a rule not indicated. Exception for special situations.
Allergic alveolitis, hyper-sensitivity pneumonitis humidifier fever	Moulds, bacteria Birds droppings Bacteria, algae (a number of different microorganisms implicated. Contaminated humidifiers)	Cultivation: Not indicated. Serology: Tests for IgG antibodies indicated if causative agent is known/suspected. Positive test: Indicates exposure, but is not diagnostic of disease. Negative test: Disease not caused by antigen used, but the patient may have been exposed to other antigens or toxins. Verification: Epidemiologic follow-up	Cultivation: As rule not indicated. Use of humidifier sludge as antigen for testing of precipitating antibodies. Serology: Elevated IgG levels in a group of people may be an indication of abnormal exposure. Preventive measures should be instituted.
"Toxicosis" ("Organic dust toxic syndrome")	Endotoxins, exotoxins (mycotoxins)	Cultivation: Not indicated Serology: No antibody response	Endotoxin: Only for research. Culture: Usually not indicated. Air sampling may be useful to verify exposure
Sick Building Syndrome (SBS)	Not identified	No specific tests available	No single test

21.8 DERMATITIS

The signs of allergic contact dermatitis are eczematoid rash or urticaria; erythema, oedema, scaling, and vesicle formation which may leave post-inflammatory hyperpigmentation. If the exposure is chronic there may be lichenified erythematous scaling eruptions. Allergic contact dermatitic erup-

tions start 1–2 days after exposure, and resolve in 1–2 days.

A number of factors including low humidities which may be associated with high air velocities, high temperatures and airborne fibre may lead to a dermatitis.

21.9 "SICK BUILDING SYNDROME"

Sick Building Syndrome is a worldwide problem. The symptomatology of this syndrome is varied, but there are a small number of characteristic symptoms which may occur singly or in combination (Appendix 4):

- nasal:
  most commonly nasal obstruction (usually described as nasal stuffiness) or nasal irritation with rhinorrhoea;
- ocular:
  dryness or irritation of the eyes;
- oropharyngeal:
  dryness of the throat;
- cutaneous:
  dryness and irritation of the skin, occasionally associated with a rash on exposed skin surfaces;
- general manifestations:
  abnormal fatigue or tiredness, general malaise, headache or feeling heavy-headed.

The symptoms of sick building syndrome all have potential major causes unrelated to the building. The important point to elicit is the timing of the symptoms. Sick building symptoms improve on days away from the problem building and recur on return to the building (Robertson and Burge, 1985). Symptoms may start within 15–30 min of entering a building or may be delayed for several hours. Some symptoms improve within 30 min of leaving the building, some require several hours to improve. The skin changes may take longer to improve than this. The more severe symptoms occur on most work days, however many workers complain of less frequent symptoms.In practice symptoms that are present on most days or in most weeks which improve away from a building, can reasonably be attributed to the sick building syndrome. The diagnosis is established by questionnaire. There is no place for any specific medical examination for symptomatic workers with sick building syndrome. There are no specific appearances to the nasal mucosa in building related rhinitis compared to other forms of rhinitis. Throat examination in symptomatic workers is usually normal. Slit-lamp examination of the eyes of workers complaining of eye irritation has demonstrated an increase in the rate of tear film breakup (Franck, 1986) but this is not a routine test for

the evaluation of this symptom. There is no relevant examination for lethargy, headache or heavy-headedness, except to exclude other causes.

Air conditioning is used in many different situations for the purposes of comfort, safety and even noise abatement, it is used in large blocks of flats or individual dwellings in hot climates (for example detached houses, hospitals, hotels, department stores, city office blocks, museums, libraries containing valuable documents). It is also used in numerous industries where humidification is necessary, such as printing and high-tech industries of electronics, data processing and magnetic tape manufacture. There are, therefore, millions of people living or working in premises where the ventilation is regulated and where use is made of air-conditioning systems. The problem is not limited to air-conditioned buildings, it is also present to a usually lesser extent in naturally ventilated buildings.

There have been relatively few systematic studies using randomly selected buildings and relating multiple office building characteristics to the symptoms of sick building syndrome (Finnegan et al., 1984; Hedge and Wilson, 1987; Burge et al., 1987; Skov et al., 1987). The studies have varied in design making comparison difficult but all have shown an excess of symptoms in sealed, air conditioned buildings when compared to naturally ventilated buildings.

The medical management of sick building syndrome should be directed towards effective primary and secondary prevention of the condition as well as advising about specific therapy. A stepwise approach to the investigation of problem buildings should be adopted. Medical evaluation is an important part of the first and last of these steps. They may include examination of employees with and without symptoms, and examinations are usually carried out by an occupational unit. Additional examination of specific exposures such as microbiological exposures may be needed.

The medical examination can incorporate a detailed questionnaire related to symptoms and should ask questions about the psychosocial conditions at work, the relationships of individuals to their colleagues and superiors and the type of work they are performing, all of which may influence symptoms. This requires cooperation between specialists, but must be evaluated by trained persons such as occupational physicians. Questionnaire examinations are normally not necessary as most of the problems in buildings are solved by the previous steps of the investigations. When questionnaires are used they must be standardised and the original questionnaire should be administered again after remedial measures have been carried out.

The first step of the medical management is the involvement of all those dealing with the health and comfort of the employees including personnel and senior management, services engineer, ergonomist and hygienist, health and safety officer and occupational health practitioner. While the specific causes of

sick building syndrome are obscure the most important initial action is a full evaluation of the functioning and maintenance of the air-conditioning system and the building itself and the correction of abnormalities nearly always identified.

The problem should not be underestimated since it has been calculated that costs of absenteeism due to building problems is about eight times greater than the financial benefit gained through energy savings.

21.10 NONSPECIFIC ENVIRONMENTAL HYPERSUSCEPTIBILITY

It is recognised that there is considerable individual variation in susceptibility to environmental agents. There are, however, small numbers of individuals who claim multiple sensitivities. This may be considered under the category of environmental hypersensitivity or multiple chemical sensitivities.

One study has shown this condition to be commoner amongst women than men, frequently the individual has a university education or postgraduate training (Kempton Hayes et al., 1991). The history arises after exposure or believed exposure to environmental chemicals or toxins. The symptoms are nonspecific and multiple and may include behavioural changes, fatigue, depression, psychiatric disorders, musculoskeletal, respiratory, genito-urinary, GI and mucous membrane irritation complaints. Affected persons have varying degrees of morbidity, from mild discomfort to total disability. There are usually no abnormal findings on physical examination. Although abnormalities of complement and lymphocytes have been recorded, no single laboratory test, including serum IgE, is consistently altered. Improvement is associated with avoidance of suspected agents and symptoms recur with re-exposure (Thomson et al., 1985). The syndrome is probably related to a psychosomatic or post-traumatic stress disorder.

There remains a lack of any fundamental scientific basis to this condition and at the present time it is not recognised as a disease entity by most physicians. Clinicians should consider underlying medical conditions which may be treatable.

21.11 SENSORY EFFECTS AND OTHER EFFECTS ON THE NERVOUS SYSTEM

Sensory effects are defined within the context of this report as the perceptual response to environmental exposures. Sensory perceptions are mediated through the sensory systems for odour, touch, itching, freshness, etc. The

senses responding top environmental exposures include hearing, vision, olfaction, taste, and the common chemical sense in the skin and mucus membranes.

Two main classes of sensory perceptions exist in the indoor environment. These both can be adverse or non-adverse. The first class includes perceptions of the surrounding physical or chemical environment. The second class include perceptions of events inside the body or on the body surface.

Responders are often unable to identify a single sensory system as the primary base for sensory irritation by airborne chemical compounds. The sensation of irritation is influenced by a number of factors such as previous exposures skin and mucosal temperature, competing sensory stimulation, etc. Since interactions and adaptations (or facilitations) are characteristics of the sensory systems, the duration of the exposure influences the perception. Comfort and discomfort by definition are psychological concepts and for this reason the related perceptions and or symptoms, even when severe, cannot be documented without using subjective reports.

Sensory effects reported to be associated with indoor air pollution are in most cases resulting from stimulation of several sensory systems. Perceived air quality is for example mainly related to stimulation of both the olfactory and trigeminal nerves.

It is well known that environmental exposures can affect the nervous system. A wide range of effects may be of importance ranging from those at molecular level to behavioural abnormalities.

Since the nerves of the CNS typically do not regenerate, toxic damage to them is usually irreversible. The nerve cells are highly vulnerable to any depletion in the oxygen supply. Furthermore, the nerve cells will be exposed for a long time to chemicals that are able to enter the CNS. The risk of accumulation of hazardous compounds within the CNS is higher than in most other body tissues since the nerve cells are slow in metabolising intruding chemicals. Many solvents affect the nerve cells or the transmission of nerve signals, e.g. by inducing narcotic effects. Although a number of adverse health disorders of the CNS are suspected of being associated with exposure to hazardous pollutants in the environment, there is at this moment no documentation that would implicate non-industrial exposure in homes or offices to indoor air pollution as being related to these endpoints. However, both Parkinson's disease and Alzheimer's disease involve olfactory dysfunctions already at an early stage of development of the disease.

Methods for assessment of effects

Sensory effects such as odour and mucosal irritation are perceptions, and therefore, by definition subjective in nature. The assessment of such subjective aspects of sensory stimulation must involve humans. The olfactory system adapts during prolonged exposure and olfactory measurement should control

for this adaptation. As a consequence of adaption to any odour in indoor air, two different responses may be identified: that of the visitor and that of the occupant. A WHO expert group has recommended that odours should be measured through the immediate response of the unadapted olfactory system (visitor situations). It should be noted that odour intensity measured by visitors does not necessarily correlate with the perceptions of the occupants.

A number of indicators or substitute measures may be used to estimate or predict the odour or mucosal irritation potency of chemicals. The concentration of volatile organic compounds has been suggested as a possible indicator of the overall perceived indoor air quality. Carbon dioxide emitted by human metabolism is often used as an indicator for perceived indoor air quality as affected by bioeffluents. The science of psychophysics offers a variety of sensory models for studying indoor air quality effects and for indoor air quality characterisation (bioassays). Regulatory agencies now require sensitivity, validity, reliability and biological meaningfulness of sensory methods applied for indoor air quality control. Therefore, quality assurance in sensory measurements is mandatory (Berglund et al., 1990).

Individuals, panels and populations do differ in sensory sensitivity, response behaviour and value judgements. Some of these differences are environmentally induced. It is important to specify the target groups of indoor air quality control based on sensory effects and how they relate to large population groups.

Methods for assessing neurotoxicity in animal and human studies include a number of neurophysiological and behavioural diagnostic techniques designed to study central or peripheral nervous functions. Examples of neurobehavioural tests are reaction time, memory, manual dexterity, etc., and examples of electrophysiological techniques are measurements of visual or auditory evoked potentials, nerve conduction velocity, etc. These tests are increasingly being used in the field because of their noninvasive nature. Neurobehavioural tests can be used in experimental laboratory settings as well as epidemiological studies. However, the attribution of abnormal results to acute reversible impairments or to irreversible brain lesions is difficult.

21.12 CANCER AND EFFECTS ON REPRODUCTION

A few indoor air pollutants notably asbestos, radon, formaldehyde and environmental tobacco smoke (ETS) have been associated with cancer. In this context formaldehyde is probably not a human carcinogen. Very few studies have tried to evaluate whether indoor air pollutants affect human reproduction.

Lung cancer is the major cancer which has been associated with indoor air (radon and ETS). Asbestos exposure has been associated to cancer in workers and also in workers family members. However, there is no studies linking lung cancer to asbestos exposures in homes or public buildings originating from construction materials.

Effects on human reproduction have been associated with chemicals in the environment but it is unclear to what extent (if any) exposure to indoor air pollutants is involved.

Although several substances are considered to be human carcinogens by the International Agency for Research on Cancer, only few of them have been linked to human cancer in epidemiologic studies on specific indoor exposures. The main emphasis in these studies has been on the relationship between exposure to ETS ("passive smoking") and lung cancer, a topic on which a relatively large number of studies have been published.

The carcinogenicity of radon decay products for humans has been firmly established in studies among miners at exposure levels exceeding those commonly found in the air in homes. Recently, some epidemiologic studies have addressed radon in homes more directly and some (but not all) of these suggests that exposure to radon decay products is associated with an increased lung cancer incidence. From occupational studies asbestos fibres are known to be able to cause cancer in humans. Benzene is a known cause of leukaemia in occupational exposed humans and may be present in indoor air at low concentrations.

The mutagenicity of indoor air as measured by in vitro techniques has been studied by several investigators. Mutagenicity refers to the capacity of a substance to cause a permanent change in the amount or structure of the genetic material in the organism, resulting in a change in the phenotype characteristics of the organism. The alteration may involve a single gene, a block of genes, or a whole chromosome. Tobacco smoke is a major source of mutagens in the air in homes. In addition, wood smoke from fireplaces and fumes produced by cooking activities have been shown to increase the mutagenicity of the indoor air.

Indoor Air Quality
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Chapter 22

Medical Management in Cases of Building-Related Illnesses[1]

22.1 INTRODUCTION

The worker suffering from a building-related illness is likely to attend either the occupational health department, the general practitioner or the hospital physician. In order to manage the condition effectively with regard to treatment and long-term prevention, there must be communication between those to whom the worker initially presents and those responsible for the workplace. Occupational health departments within industry have the primary role of advising the company on the effect of work on the health of the employees and the effect of the health of the employees on their work. As such the occupational health department has a central role in the management of building-related illnesses (BRI). To do this satisfactorily there must also be close cooperation with the other physicians involved. Building-related illnesses cover a wide spectrum of different diseases. Some are medically well defined with both short and long-term health effects accepted and with reasonable agreement about their management and treatment. Others are less well defined but these still require effective management.

The medical management of BRI should be directed towards effective primary and secondary prevention of the conditions as well as advising about specific therapy. To achieve this aim the medical practitioner cannot work in isolation restricted to reactively treating the individual worker. To successfully manage BRI the medical practitioner must develop a coordinated, proactive approach to the workforce, the workplace and senior management.

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22.2 MEDICAL MANAGEMENT POLICY

The most effective way to manage BRI must be to develop a concerted action policy. As a minimum the policy should seek to involve all interested parties, agree responsibilities, discuss preventative measures and management options and facilitate communication. The policy should be extensive in remit. The agreement on management strategies between groups, prior to the occurrence of health problems, circumvents the defensive, negative attitude of many based on either "not my fault" or "nothing I can do" and can therefore result in remedial action being taken sooner rather than later. The central role of the medical practitioner in bringing together these groups within a constructive, "neutral" framework is important. Much of the policy will be dependent on the particular needs of the workplace and workforce, however the general outline of a management policy could be as follows.

22.3 POLICY DEVELOPMENT COMMITTEE

The coordination of action to both treat and prevent BRI requires the involvement of a number of key personnel within the workplace. The first step therefore would be to involve all those dealing with the health and comfort of the employees into a policy development committee. This should include employee representatives, personnel and senior management, services engineer, ergonomist and hygienist, health and safety officer and occupational health practitioner.

A joint policy outlining an effective, and agreed, management strategy should then be developed. This should state clear areas of responsibility for each individual group but within an overall integrated plan.

22.4 THE ACTION PLAN

22.4.1 Assessment and treatment of affected workers

Responsibility: Occupational physician, primary care physician, general physician.

It is likely that the affected worker will either first attend the occupational health department, his primary care physician or a general physician. Within the workplace the policy should be that he be encouraged by all parties to attend the occupational health department at the earliest opportunity. The hospital physician and general practitioner frequently omit the issue of occu-

pation in the causation of the disease. Subsequent communication between these physicians and the occupational physician, even if an occupational factor is thought to be causal, is likely to be poor (Howe, 1962). An appreciation of the factors contributing to this poor communication may help improved reporting which is essential if the condition is to be effectively managed.

First, lack of knowledge: Although it would be hoped that physicians would have some knowledge of building-related conditions, this may not be the case. Appropriate training and warning given to the employee is likely to increase awareness and lead to a greater likelihood of a physician diagnosing the condition as work-related. Such training should be recorded and the instructions/warnings be a written, integral part of the policy which each worker should receive. Second, ethical issues: It is clearly unethical to disclose any information, particularly to an employer, on patients without their written informed consent. The employee may be reluctant to give such consent either because he feels it unlikely that his company will wish to do anything about his work-related problem or because the worker may feel that his job or promotion may be significantly affected. The general issue to all employees of the policy should also include an encouragement to report the condition, demonstrating a willingness on the part of management to deal with the problem and to specifically include a statement about job security.

Assessment

There are a number of objectives which should be achieved by the physician dealing with a case of BRI. It is assumed that both the "disease" and its "work-relatedness" have been confirmed as discussed elsewhere. The condition then requires to be recorded and reported into the management policy.

22.4.2 Reduction of exposure

The first priority, as with all occupational diseases, is to prevent continued exposure.The urgency of this task will be dependent upon a number of factors such as: (1) the nature of the disease and the degree of certainty about the diagnosis; (2) the ability to recognise the cause(s); and (3) the relative ease or difficulty in reducing exposure.

1. The nature of the disease

As mentioned previously BRI covers a spectrum of conditions. With hypersensitivity reactions, where the diagnosis can be made with some degree of certainty, such as occupational asthma, extrinsic allergic alveolitis (hypersensitivity pneumonitis) and humidifier fever, urgent action is required to find the cause and also prevent re-exposure. At the other end of the spectrum condi-

tions such as work-related neurotoxic symptoms e.g. lethargy, dizziness etc. are more difficult to define and the prevention of re-exposure more difficult to achieve.

2. Ability to recognise cause

Proving the relationship between suspected agent and ill health effect is often impossible. If the cause is not known or the degree of certainty poor then the prevention of further exposure is obviously complex. One of the main criteria for the audit of any remedial action must be the demonstration of reduction in ill health effect which, without some objective measure, may be unconvincing.

3. Difficulties in reducing exposure

There are considerably more difficulties with this in relation to BRI than with many other forms of occupational exposure to hazardous agents. Firstly, the agents are less clearly defined; secondly, there are few relevant exposure standards; and thirdly, methods of exposure reduction are often less well accepted.

Current knowledge and practice are therefore variable and the policy committee needs to develop its own standards given existing guidelines. The means whereby exposure is reduced is otherwise similar to other unacceptable occupational exposures namely either the removal or replacement of the offending agent or its containment and control. If these measures are not achievable or not effective then, given that personal protection is unlikely to be acceptable, the worker needs to be effectively relocated.

22.4.3 Relocation

Responsibility: Supervisor, personnel, occupational health, worker.

The relocation of affected workers currently tends to be handled badly, such as after prolonged and acrimonious debate. Other possible unsatisfactory outcomes include relocation to a completely different work site, retirement on the grounds of ill health, redundancy, resignation or dismissal.

The relocation policy needs to be clear and accepted by all parties. The physician has a central role in advising worker and management. The physician needs to have assessed not only the factors related to the disease as outlined above but also needs to make an assessment of the future health and comfort requirements of the worker as well as the suitability of the proposed relocation. In order to achieve removal from exposure the physician needs to have previously assessed the proposed relocation areas. He may, for instance, have data showing that some areas of the building are associated with lower

prevalences of disease which may therefore result in an improvement in the condition. Supervision, personnel and senior management will also have operational requirements which all need to be considered when advising on the ideal work position. The physician needs to continuously review the effectiveness of the relocation by whatever measures are available.

22.4.4 Counselling

The counselling of a worker affected by an occupational condition is a major responsibility of the physician or occupational health department. The counselling of the individual worker with building-related illness is often difficult. Questions will be asked for which there may be no answer and therefore the physician may be defensive and unhelpful. Questions to be answered will include: what is the disease, what has caused it, what are the short and long-term effects, what are you and the company going to do about it, what treatment should I take, how secure is my job, should I claim compensation? The confidence in answering such questions will depend upon the nature of the disease and the effectiveness of a building-related illness management policy. Such questions should be addressed by the action plan committee and a policy agreed with senior management. If there is an agreed policy then the occupational physician will be more confident about options and the likely outcome. Consideration of these issues at an early stage makes for a consistent and equitable approach when cases occur.

22.5 SPECIFIC TREATMENT

Specific treatment is not usually the province of the occupational physician but usually that of the primary care physician, general practitioner, or hospital specialist.

As previously mentioned, BRI covers a variety of illnesses and removal from exposure rather than specific treatment is the priority.

In many cases the condition causes little morbidity or mortality, is relieved by removal from the workplace, and treatment is usually symptomatic. There have been few studies of specific treatment for all BRI.

22.5.1 Occupational asthma

The treatment of occupational asthma is the same as all other asthma except that treatment is less likely to be successful if exposure is continued. Allergen avoidance is therefore a primary aim (Chan-Yeung et al., 1987).

Treatment for patients with normal lung function and infrequent symptoms should initially consist of bronchodilators such as β-2 agonists given as required (British Thoracic Society, 1990). If the affected worker needs this treatment most days, or has night-time symptoms, then regular inhaled anti-inflammatory treatment should be given. The options include inhaled sodium cromoglycate, inhaled nedocromil sodium and inhaled corticosteroids. The treatment of choice is inhaled steroids and these should be tailored to achieve maximum control. The dose of inhaled steroids may need to be high. Inadequately controlled nocturnal symptoms may require oral β-2 agonists, long-acting inhaled β-2 agonists and/or long-acting oral xanthines. High dose inhaled bronchodilator therapy should be reserved for those not responding to standard measures. Intensive β-2 agonist inhaler therapy over long periods should be avoided. Inhaled ipratropium bromide is also a treatment option. Short courses of oral steroids may be required to achieve control of exacerbations. Only rarely should they be required to be used on a regular basis.

Monitoring of asthma by means of serial peak flow recordings is recognized as a way to monitor the effectiveness of therapy, as well as being specifically useful for monitoring work-relatedness in occupational asthma acknowledging its effort dependency.

22.5.2 Humidifier fever

Humidifier fever is usually a self-limiting condition. Typified by flu-like symptoms, headache, aches and pains in the muscles and joints and fever it usually occurs some hours after exposure on the first day back to work after a rest day (Pickering et al., 1976). Tolerance develops and the symptoms subside 48–72 h later. Symptomatic treatment is usually all that is required.

22.5.3 Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (hypersensitivity pneumonitis) may be acute and completely resolve after removal from exposure. Symptoms are similar to humidifier fever but may also consist of respiratory symptoms such as cough and breathlessness. Acute symptoms usually rapidly resolve within 24–48 h; however, if symptoms are significant then a short course of oral corticosteroid can be given. Sub-acute or chronic forms of the condition may result in diffuse interstitial fibrosis, abnormal lung function and reduction in gas transfer. If the condition becomes chronic with deteriorating lung function then treatment may be tried with more prolonged courses of steroids.

22.5.4 Infections

It is likely that relatively few infections are found to have a greater incidence in buildings such that the building factors are found to be causal.

22.5.5 Legionella

Infections with Legionella are caused by the inhalation of the bacteria which contaminate water reservoirs and which multiply if the water is warm. Legionella causes a number of conditions including a true infection which may produce either pneumonia or a subclinical condition or secondly a condition known as Pontiac Fever. Treatment of the pneumonia is with erythromycin, rifampicin or ciprofloxacin. Pontiac fever is a self-limiting condition with fever and malaise requiring symptomatic treatment only.

22.5.6 Mucous membrane irritation

Eye symptoms

Workers may complain of dry eyes or itching or watering of their eyes. There have been no specific studies of treatment of these conditions due to indoor air quality problems. Ocular problems should be accurately diagnosed prior to treatment as the inappropriate or long-term use of ophthalmic preparations can frequently themselves result in problems (Buckley, 1990). It may be that artificial tears such as hypromellose may help "dry" eyes. A therapeutic trial of sodium cromoglycate could be tried with itching or watering eyes; however, if this is unsuccessful it should be discontinued. Given the nature of the condition it is probably best that more potent treatments such as steroids be avoided unless properly assessed.

Nasal symptoms

Nasal symptoms are also common. These consist predominantly of stuffiness or sneezing and runny nose. Again, there is little specific information on the treatment of these conditions. Topical nasal preparations of corticosteroid, sodium cromoglycate and ipratropium bromide are all available and used predominantly for rhinitis-type symptoms (Wood, 1990). There is little to recommend one more than another and they remain treatment options; however, given the persistent nature of the condition, whether one should be considering long-term topical treatment is open to debate. Certainly vasoconstrictors should be avoided. Newer generation antihistamines with less sedation such as terfenadine or astemizole are effective against a number of the irritant and allergic symptoms, although again whether they should be used indefinitely is questionable.

The role of immunotherapy in building-related allergic disease remains to be demonstrated. Immunotherapy is generally less popular now because of a lower safety profile. The causal allergens are not as yet identified and immunotherapy is not a realistic option.

Skin symptoms and sensations

Some studies have shown "dryness" of the skin to be a problem in some buildings. General skin degreasing should be avoided and if the condition persists then simple emollients applied. True rashes are less common and again avoidance is more important than treatment. Physical drying may be a factor in the production of the rash and simple emollients should be tried initially. Topical steroids or oral antihistamines should not be viewed as a long-term solution, the condition should be properly assessed and causal factors avoided.

22.5.7 Symptoms from the central nervous system

Headache

There is little that is specific about the headache of workers in buildings. It is no more likely to be migrainous than other headaches and usually resolves on removal from the provoking factors. Simple analgesia is therefore all that is required.

The treatment of other neurotoxic symptoms such as lethargy, dizziness and nausea within the specific context of building-related illnesses is unclear.

As can be seen, there is little that is specific about the treatment of building-related illnesses. Underlying pathology should always be looked for to avoid mistaken attribution to "allergy" or "toxic reaction" and subsequent inappropriate and possibly damaging treatment commenced. Avoidance of precipitating factors is more important than long-term treatment.

22.6 HEALTH SURVEILLANCE

Responsibility: Occupational Health

Primary prevention

As part of the medical management of building-related illnesses there needs to be a primary prevention strategy. This will again require continuous input from all members of the development committee. Part of the medical role could be to initiate a health surveillance program which would have a number of aims.

Aims

The aims would be to:

(1) identify pre-existing disease;

(2) identify susceptible individuals;

(3) identify good and bad areas of a building;

(4) monitor long-term changes in building performance;

(5) objectively monitor remedial action; and

(6) provide a baseline level of knowledge.

Either a total or random sample of the office population could be studied and if possible a comparison group should be included.

The surveillance should be performed at regular intervals such as yearly. On specific individuals it should be carried out pre-employment due to the likelihood of pre-existing problems from other workplaces. It may be carried out at more frequent intervals such as on relocation of the individual or the workforce or on request. The exact methodology of the surveillance will depend on measures currently available. A standard questionnaire would be a good reproducible measure of the overall prevalence of some of the building-related illnesses. With frequent sampling, rates of change could be monitored. Objective measures of health effects are more difficult and are addressed elsewhere. Biological effect monitoring could also be employed as a surveillance method.

The data collected from the health surveillance must be used by the management group. It would be particularly helpful for the hygienist and engineers to be able to identify problem areas and can also be used as a form of audit of the effectiveness of remedial action and indeed the primary prevention policy as a whole.

22.7 WORKPLACE ASSESSMENT

Responsibility: Occupational health/supervisor/ergonomist/hygienist.

The occupational health department does have a role in the initial investigation of the workplace. Major problem areas can be highlighted for subsequent investigation. The detailed investigation of the workplace will not be covered here however the results from the workplace inspections need to be passed on to those advising on relocation, etc.

22.8 INDOOR AIR QUALITY

Responsibility: engineer/hygienist/occupational health.

Indoor air quality must also be continuously monitored rather than monitoring only when problems arise. The information should again be input into the management committee.

22.9 CONCLUSIONS

Only by the use of this type of coordinated prevention policy can the situation of building-related illnesses be addressed. Primary prevention and surveillance must happen before cases occur. There must be agreement by those responsible on an action plan. Workers must be aware and informed and reporting encouraged. Surveillance must happen regularly. Information on health from the occupational health department must be coordinated with results from the building services and on indoor air quality from the hygienist if a total quality programme of prevention is to be maintained. The medical management of building-related illnesses can therefore be seen to cover a wide range of different tasks and requires different areas of expertise. Treatment is important but is only a small part of management.

PART IV — REFERENCES

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PART V
Dynamics of Indoor Air Contaminants

This part is devoted to providing an understanding of pollutant generation, diffusion and decay in the indoor environment. Subjects discussed include indoor air quality modelling, emissions from sources of pollution and decay of indoor pollutants.

This part has been prepared by D. Moschandreas. Tables of sources, emission rates and deposition velocities have been reproduced from US EPA publications.

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Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 23

Indoor Air Quality Modelling

Indoor air quality models describe the transport and dispersion of air contaminants throughout a structure and the variation of indoor air contaminant concentrations as a function of source strengths, air-exchange rates, removal mechanisms, and other parameters.

In a mass or energy balance model, the term compartment or zone refers to a region in which spatial variations in contaminant concentration can be ignored over the time period of interest. A single room, a floor, or a house that is well-mixed is usually treated as a single zone. Houses with central forced-air heating systems can be considered to be well-mixed when the circulation fan is operating. Houses with other types of heating systems can also be considered to be well-mixed providing interior doors remain open and a sufficiently long time period is allowed for mixing. For steady-state conditions (when generation of contaminants equals removal), these models reduce to simple algebraic equations. The basic mass balance equation for a single zone model is presented below.

The accuracy of the single zone model depends on the degree of mixing, the time period of concern, and the extent to which the parameters have been characterized. Model performance can be measured by comparing predicted data with actual measured values. After the model has been calibrated, it should be evaluated against other data. It is important to note that the data set used for calibrating the model should not be used to evaluate the model's general predictive capability.

Single zone models may not be adequate when sources and sinks are not uniformly distributed throughout the area or when stratification of contaminants occurs; in such cases multi-zone models may be needed. Multi-zone models may be appropriate, for example, to describe contaminant concentrations when a kerosene heater is operating in one room of a house (zone 1) that is separated from the remainder of the house (zone 2) by closed doors.

Multi-zone models might also be used when predicting contaminant concentrations in a building with multiple floors. These models include separate mass balance equations for each zone, and they are more complex than single zone models.

In general, indoor air quality models are useful tools that can be used: (1) to understand how the factors that affect indoor air quality relate to each other; (2) to predict concentrations of contaminants for places and conditions that cannot be measured; and (3) to estimate required accuracy and precision for monitoring studies.

23.1 AVAILABLE INDOOR AIR QUALITY MODELS

Numerous models have been developed to estimate concentrations of contaminants in indoor air, and some of these also estimate inhalation exposures. The Air and Energy Engineering Research Laboratory (AEERL) at EPA has developed a multi-zone indoor air quality model (Sparks, 1988) which can be used on an IBM-PC or compatible computer.

The AEERL model treats each room as a well-mixed chamber that can contain both sources and sinks. Source terms which can be included are random on/off sources (for example, cigarettes), sources that are on for specific periods of time (for example, kerosene heaters), steady-state sources (for example, moth crystals), and sources with high initial emission rates followed by a low steady-state rate of emission (for example, floor wax). The model also allows the impact of heating, ventilating, and air-conditioning (HVAC) systems, air cleaners, and inter-room air flows on indoor air quality to be evaluated. The model has been verified *to a limited extent* with experimental data from EPA's indoor air quality test house.

The AEERL model is user-friendly and has a menu-driven user interface. Output from the model can be displayed if a graphics adapter and monitor are available.

Further discussion on single zone, multi-zone, and exposure models can be found in NRC (1981); Wadden and Scheff (1983); Nagda, Rector, and Koontz (1987); McNall et al. (1985); Sexton and Ryan (1988); Sexton and Hayward (1987); Axley (1987); and Repace (1987). For exposure models see also Chapter 14.

23.2 MASS BALANCE EQUATION

The primary source for the following discussion is Nagda et al. (1987); other useful sources include NRC (1981) and Wadden and Scheff (1983).

The generation and removal of contaminants in indoor environments can be described mathematically based on the conservation of mass:

$$\text{Rate of accumulation} = \text{rate of [input} + \text{generation} - \text{output} - \text{sinks]} \tag{23.1}$$

or

$$\frac{V\mathrm{d}C_{\mathrm{in}}}{\mathrm{d}t} = \text{rate of change in mass due to:}$$

[Infiltration of outdoor air] + [Generation indoors] – [Exfiltration of indoor air] – [Indoor removal of contaminants] (23.2)

where V = the indoor volume, and C_{in}= the indoor concentration of any pollutant under investigation.

Parameters in the mass balance equation must be evaluated independently. Some parameters, such as volumes and surface areas, can be measured directly or can be easily obtained from blueprints. Others such as ventilation rates and source emission/removal rates are more difficult to obtain.

23.2.1 Infiltration and exfiltration

The infiltration of contaminants from the outdoors depends on the product of the outdoor contaminant concentrations (C_{o}) and the volume rate of air exchange (vV), where v is the air exchange rate in air changes per hour (ach).

Not all outdoor air contamination that moves into a structure reaches the inside; some fraction, F_{b}, is intercepted by the cracks and crevices in the building envelope which decreases the amount that actually reaches the indoor air. The overall relationship for the change in indoor contaminant concentrations due to infiltration from outdoor air is, therefore, given by:

$$V\,\mathrm{d}C_{\mathrm{i(infil)}} = (1 - F_{\mathrm{b}})\,vV\,C_{\mathrm{o}}\,\mathrm{d}t$$

Removal of contaminants due to the exfiltration of indoor air is the product of the volume rate of air exchange (vV) and the concentration of air leaving the structure, C_{e}. In cases of good mixing, $C_{\mathrm{e}}$is the same as C_{i} and the exfiltration term over a period of time is given by:

$$V\,\mathrm{d}C_{\mathrm{i(exfil)}} = (1 - F_{\mathrm{b}})\,vV\,C_{\mathrm{i}}\,\mathrm{d}t$$

23.2.2 Generation and removal of contaminants

The generation of contaminants indoors (source term) over a period of time can be expressed as $S\,\mathrm{d}t$, where S is the contaminant emission rate,

$$V\,\mathrm{d}C_{\mathrm{i(gen)}} = S\,\mathrm{d}t$$

The removal of indoor contaminants is due to chemical reactions, adsorption of contaminants on indoor surfaces, and removal by mechanical means

through air cleaning devices. The rate of decay due to chemical reactions and/or adsorption over a period of time is given by λdt, where λ is the overall decay rate of the contaminant.

Removal by air cleaning devices depends on the volume flow-rate of air handled by the device, q, and on the efficiency of the device, F (that is, the proportion of contaminants removed). Removal by air cleaning devices can be expressed as $qFC_i dt$.

The expression for total removal over a period of time is then given by:

$$V\, dC_{i(rem)} = \lambda dt - qF\, C_i\, dt$$

23.2.3 Effective volume

The concentration of contaminants indoors also depends on the indoor volume. The actual volume that is available for contaminant dispersal depends on the degree of air circulation, and it is known as the effective volume (kV), where k is a dimensionless factor. The value of k becomes 1 when the entire indoor air volume is available for contaminant dispersal. The value of k is less than 1 when there is no forced mixing and the degree of circulation depends on thermal gradients indoors.

23.2.4 Generalized mass balance equation

Equation 23.2 can be written as a generalized mass balance equation for predicting indoor concentrations. Assuming uniformly mixed conditions and an effective volume, kV, rather than the total indoor volume, V, Eq. 23.2 becomes:

$$kV\, dC_i = (1 - F_b)\, vkVC_o\, dt + Sdt - vkVC_i\, dt - \lambda dt - qFC_i\, dt \tag{23.3}$$

or

$$\frac{dC_i}{dt} = (1 - F_b)\, vC_o + \frac{S}{kV} - vC_i - \frac{\lambda}{kV} - \frac{qFC_i}{kV} \tag{23.4}$$

where C_i = indoor concentration (mass/volume); F_b = fraction of outdoor concentration intercepted by the building envelope (dimensionless fraction); v = air exchange rate (1/time); C_o = outdoor concentration (mass/volume); S = indoor source generation rate (mass/time); kV = effective indoor volume where k is a dimensionless fraction ;λ = decay rate (mass/time); q = flow rate through air cleaning device (volume/time); and F = efficiency of the air cleaning device (dimensionless fraction).

Mixing factor: Because the extent of air mixing in the interior varies a mixing factor, m, may be introduced which modifies the air exchange to yield an effective air exchange rate of exfiltration of a contaminant. The mixing factor is the ratio of the concentration of the exiting air to the concentration of the indoor air. When the two concentrations are the same, the air is said to be completely mixed, and m equals one. When contaminants are exhausted directly from their source, m will be > 1. However, the more usual case is for m to be < 1 because mixing is not complete.

The value of m has been estimated to be in the range of 0.33–1.0 (Wadden and Scheff, 1983). In general, mixing between rooms in residential structures is usually complete in less than 1 h; therefore, the complete mixing assumption is often used to estimate average concentrations over time periods greater than 1 h.

The effective air exfiltration rate for a given contaminant is the product of the mixing factor, m, and the air exchange rate, v, and it is given by mv.

Therefore, Eq. 23.4 becomes:

$$\frac{dC_i}{dt} = (1 - F_b)\, vC_o + \frac{S}{kV} - mv\, C_i - \frac{\lambda}{kV} - \frac{qdC_i}{kV} \tag{23.5}$$

23.3 USE OF THE MASS BALANCE EQUATION

The basic mass balance equation can be used for many applications, and the solutions to the equation will vary depending on initial assumptions. Some examples for applications include using the equation to predict the build-up of contaminants as a result of the infiltration of contaminated outdoor air or from using and indoor source for varying time periods; to evaluate the effectiveness of mechanical ventilation; or to determine the effect of air exchange rates and other variables on indoor contaminant concentrations.

23.3.1 Equilibrium concentrations

One application of the model is to compute the equilibrium (steady-state) concentrations that would be achieved under a variety of conditions. The simplest case assumes a nonreactive contaminant, no indoor sources ($S = 0$), no cleaning devices ($F = 0$), no capture of outdoor contaminants by the indoor air ($F_b = 0$), and uniform mixing ($m = 1$). Under these conditions, Eq. 23.5 reduces to:

$$\frac{dC_i}{dt} = vC_o - vC_i$$

In this simple case, when equilibrium is reached (that is, when the indoor air concentration is constant), $dC_i/dt = 0$, then the indoor concentration equals the outdoor concentration.

Effects of indoor sources

If we now introduce an indoor source of contaminants, Eq. 23.5 becomes:

$$\frac{dC_i}{dt} = v(C_o - C_i) + \frac{S}{kV}$$

and the equilibrium concentration (when $dC_i/dt = 0$) would be given by:

$$C_i = C_o + \frac{S}{kVv} \tag{23.6}$$

Other terms can be introduced in a similar way.

Effect of time

Another application is to determine under a given set of conditions how much time is required to reach equilibrium or what concentration will result after a given period of time. Using the simple case in Eq. 23.6 where we assume an initial indoor concentration, C_i, and a final indoor concentration equal to a constant outdoor concentration, C_o, then the indoor concentration at any time, t, is given by:

$$C_{i,t} = C_o + (C_i - C_o)e^{-v(t-t_0)} \tag{23.7}$$

For example, we can calculate the indoor concentration of carbon monoxide (CO) after 2 h in a 100 m^3 space assuming that the outdoor concentration is 9 ppm; the initial CO indoors is 3 ppm; there are no indoor sources or cleaning devices; the air exchange rate is 0.3 ach; and uniform mixing of the indoor air occurs. The result is:

$$C_{2h} = 9 \text{ ppm} + (3 \text{ ppm} - 9 \text{ ppm})\ (0.55) = 5.7 \text{ ppm}$$

After 2 h the indoor concentration under the stated conditions equals 5.7 ppm or about 60% of outdoor concentration.

23.3.2 Quantifying parameters

Values for the input parameters which are needed either to make simple computations or to use available indoor air quality models are derived by direct measurements or by combining special knowledge of specific conditions with

TABLE 23.1

Major sources of infiltration measured during weatherization studies

Source	Relative amount of leakage	
	Range (%)	Average (%)
Walls	18–50	35
Ceiling details	3–30	18
Heating system	3–28	15
Windows and doors	6–22	15
Fireplaces	0–30	12
Vents in conditioned spaces	2–12	5
Diffusion through walls		<1

Source: ASHRAE (1985).

values derived from the literature or specific research. The following discussion provides some basis for assisting the user in defining values for specific applications.

Infiltration and exfiltration: The infiltration rate of a given building can vary considerably depending on the weather conditions, occupant activities, and the operation of appliances and HVAC equipment.

Weatherization studies on houses in the United States have shown that major sources of air leakage are walls and floors, ceilings, heating systems, windows and doors, and fireplaces. Air leaks through vents and diffusion through walls are minor sources. The importance of these individual sources will vary depending on the region of the country and construction techniques. Table 23.1 identifies specific sources of air leakage and ranges of infiltration that have been measured for each.

Air exchange rates: Air exchange rates for input into models can be obtained in one of three ways.

One approach is to use an average number of air exchanges per hour that have been developed for different types of structures and conditions. This method provides only ballpark estimates, since air exchange rates cannot be reliably estimated from a visual inspection of the building, its age, or construction. Air exchange rates can also be determined using empirical models which are statistical fits to measurements over a long period of time at specific sites. These models incorporate temperature differentials between indoors and outdoors and wind speed with empirically derived regression constants to obtain air exchange rates. The accuracy of empirical models is about 40%, but individual predictions can vary by 100% (ASHRAE, 1989). The best way to

obtain air exchange rates is to measure them directly using tracer gas methods. If direct measurements are not possible, air exchange rates can also be determined indirectly by measuring or calculating air leakage rates and converting these rates to air exchange rates.

A model developed by the Lawrence Berkeley Laboratory (Sherman and Grimsrud, 1980) has been widely used as the basis for indirect calculations and it is summarized below. The accuracy of these calculations will depend on the accuracy of the required inputs. The methods of calculating the air exchange rates from leakage rate data in residences is as follows (ASHRAE, 1989):

(1) Use Table 23.2 to determine the effective air leakage area from each possible source. For example, the leakage area of 100 ft of uncaulked sills is: 100 ft $\times$ 0.19 in^2/ft = 27.0 in^2.

(2) Add the individual leakage areas to obtain a total effective leakage area. The total effective leakage area can also be measured the using fan pressurization.

(3) Using the effective leakage area, calculate the airflow rate due to infiltration:

$$Q = L(At + Bu^2)^{0.5}$$

where Q = airflow rate, cfm; L = effective leakage area, in^2; t = average indoor–outdoor temperature difference for the time interval of calculation, F; A = stack coefficient $cfm^2\ in^{-4}\ °F^{-1}$.

The stack coefficient for residences is a function of the number of stories: A = 0.0156 for one story; A = 0.0313 for two stories; A = 0.0471 for three stories; B = wind coefficient, $(cfm^2)\ (in^{-4})\ (mph^{-2})$.

The wind coefficient depends on the number of stories and the degree of shielding around the house = average wind speed measured for the time interval of interest, mph.

(4) Calculate the infiltration rate by dividing Q by the building volume.

23.3.3 Statistical methods

Another approach for predicting indoor pollutant concentrations is based on statistical methods. Regression analysis, including multi-linear regression techniques, is used in indoor statistical models. A statistical model was developed using linear regression analysis to predict indoor concentrations of nitrogen dioxide based on recent survey data. The empirical model employed multi-linear regression techniques and data from five cities: Boston, California, Portage, Saint Louis, Watertown. The statistical model is the following regression equation (23.8).

TABLE 23.2

Effective leakage area of building components (0.016 in water)

Component	Best est.	Max	Min
Sill foundation–wall			
Caulked, in^2/ft of perimeter	0.04	0.06	0.02
Not caulked, in^2/ft of perimeter	0.19	0.19	0.05
Joints between ceiling and walls			
Joints, in^2/ft of wall (only if not taped or plastered and no vapour barrier)	0.07	0.12	0.02
Windows			
Casement			
Weatherstripped, in^2/ft^2 of window	0.011	0.017	0.006
Not weatherstripped, in^2/ft^2 of window	0.023	0.034	0.011
Awning			
Weatherstripped, in^2/ft^2/of window	0.011	0.017	0.006
Not weatherstripped, in^2/ft^2 of window	0.023	0.034	0.011
Single Hung			
Weatherstripped, in^2/ft^2 of window	0.032	0.042	0.026
Not weatherstripped, in^2/ft^2 of window	0.063	0.083	0.052
Double Hung			
Weatherstripped, in^2/ft^2 of window	0.043	0.063	0.023
Not weatherstripped, in^2/ft^2 of window	0.086	0.126	0.046
Single Slider			
Weatherstripped, in^2/ft^2 of window	0.026	0.039	0.013
Not weatherstripped, in^2/ft^2 of window	0.052	0.077	0.026
Double Slider			
Weatherstripped, in^2/ft^2 of window	0.037	0.054	0.02
Not weatherstripped, in^2/ft^2 of window	0.074	0.11	0.04
Doors			
Single Door			
Weatherstripped, in^2/ft^2 of door	0.114	0.215	0.043
Not weatherstripped, in^2/ft^2 of door	0.157	0.243	0.086
Double Door			
Weatherstripped, in^2/ft^2 of door	0.114	0.215	0.043
Not weatherstripped, in^2/ft^2 of door	0.16	0.32	0.1
Access to Attic or Crawl space			
Weatherstripped, in^2/ft^2 per access	2.8	2.8	1.2

(continued)

TABLE 23.2 (*continuation*)

Component	Best est.	Max	Min
Not weatherstripped, in^2/ft^2 per access	4.6	4.6	1.6
Wall–window frame			
Wood Frame Wall			
Caulked, in^2/ft^2 of window	0.004	0.007	0.004
No caulking, in^2/ft^2 of window	0.024	0.039	0.022
Masonry Wall			
Caulked, in^2/ft^2 of window	0.019	0.03	0.016
No caulking, in^2/ft^2 of window	0.093	0.15	0.082
Wall–door frame			
Wood Frame Wall			
Caulked, in^2/ft^2 of window	0.004	0.004	0.001
No caulking, in^2/ft^2 of window	0.024	0.024	0.009
Masonry Wall			
Caulked, in^2/ft^2 of window	0.0143	0.0143	0.004
No caulking, in^2/ft^2 of window	0.072	0.072	0.024
Domestic hot water systems			
Gas Water Heater (only if in conditioned space), in^2 per unit	3.1	3.9	2.325
Electric outlets and light fixtures			
Electric Outlets and Switches			
Gasketed, in^2 per outlet and switch	0	0	0
No gasketed, in^2 per outlet and switch	0.076	0.16	0
Recessed Light Fixtures, in^2 per fixture	1.6	3.10	1.6
Pipe and duct penetrations through envelope			
Pipes			
Caulked or sealed, in^2 per pipe	0	0	0
Not gasketed, in^2 per outlet and switch	0.076	0.16	0
Recessed Light Fixtures, in^2 per fixture	1.6	3.10	1.6
Pipe and duct penetrations through envelope			
Pipes			
Caulked or sealed, in^2 per pipe	0.155	0.31	0
Not caulked of sealed, in^2 per pipe	9.30	1.55	0.31

(continued)

TABLE 23.2 (*continuation*)

Component	Best est.	Max	Min
Ducts			
Sealed or with continuous vapour barrier, in^2 per duct	0.25	0.25	0
Unsealed and without vapour barrier, in^2 per duct	3.7	3.7	2.2
Fireplace			
Without Insert			
Damper closed, in^2 per unit	10.7	13.0	8.4
Damper open, in^2 per unit	54.0	59.0	50.0
With Insert			
Damper closed, in^2 per unit	5.6	7.1	4.03
Damper open or absent, in^2 per unit	10.0	14.0	6.2
Exhaust fans			
Kitchen Fan			
Damper closed, in^2 per fan	0.775	1.1	0.47
Damper open, in^2 per fan	6.0	6.5	5.6
Bathroom Fan			
Damper closed, in^2 per fan	1.7	1.9	1.6
Damper open, in^2 per fan	3.1	3.4	2.8
Dryer Vent			
Damper closed, in^2 per fan	0.47	0.9	0
Heating ductwork-forced air systems (only in unconditioned space)			
Joints taped or caulked, in^2 per house	11	11	5
Joints not taped/caulked, in^2 per house	22	22	11
Furnace-forced air systems (only if in conditioned space)			
Sealed combustion furnace per unit	0	0	0
Retention head burner furnace, in^2 per unit	5	6.2	3.1
Retention head plus stack damper, in^2 per unit	3.7	4.6	2.8
Furnace with stack damper, in^2 per unit	4.6	6.2	3.1
Air conditioner			
Wall or window unit, in^2 per unit	3.7	5.6	0

Source: Reprinted with permission from the 1985 ASHRAE Handbook-Fundamentals published by the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.

$$C_{ss} = b + m \cdot C_{out} \tag{23.8}$$

$$b = (S/V)/(a + k)$$

$$m = aP/(a + k)$$

where C_{ss} is steady state concentration (μg/h); S is indoor source strength (μg/h); V is volume of room (m^3); a is air exchange rate (1/h); k is reactive removal rate (1/h); p is penetration factor; and C_{out} is outdoor air concentration ($\mu g/m^3$). b can be defined as the pollutants's apparent indoor source strength, and m can be defined as the penetration coefficient of the ambient pollutant concentration.

Source and penetration terms were evaluated in the bedrooms of the population of homes studied using both winter and summer data sets. A seasonal variation was expected in term of window use, appliance use, temperature gradients, and resident activity patterns. The final regression model used is shown in Eq. 23.9.

$$C_{bed} = \beta_0 - \beta_1 (C_{out}) + \beta_2(\text{SEAS}) + \beta_3 (C_{out} \cdot \text{SEAS}) + \beta_4 (\text{PILOTS}) + \beta_5 (\text{SITE}) + \beta_6 (C_{out} \cdot \text{SITE}) + E \tag{23.9}$$

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Chapter 24

Sources and Sinks in the Indoor Environment

24.1 SOURCES OF INDOOR AIR POLLUTION AND THEIR EMISSION RATES

In addition to ventilation rate and outdoor air pollution, the air quality of indoor environments is affected by many factors, among which generation of air pollution indoors (indoor sources) and removal of air pollution indoors (indoor sinks) are critical in determining indoor pollutant concentrations.

Table 24.1 provides a detailed tabulation of sources and associated contaminants. Product categories and examples of products are identified for major source categories including consumer and commercial sources, building sources, personal sources, and outdoor sources. Examples of contaminants which have been identified for the product categories are also given.

Specific compounds are grouped according to general categories of contaminants (for example, vinyl chloride is a VOC which has been measured from building materials). Examples of building materials are included (pressed wood products, construction adhesive, insulating materials, plastic piping, and vinyl or plastic wall coverings). The reader should not attempt to associate specific contaminants with specific products.

There are several methods for determining emission rates. Table 24.2 summarizes the source testing methods.

Dynamic flow-through testing can be used to measure indoor emission rates either in small (< 5 m^3) environmental test chambers or large (12–30 m^3) test chambers. ASTM standard D5116-90 contains the process for using chambers to determine emission rates; additionally the Commission of the European Communities has a guideline for the same purpose (ECA, 1991).

Larger scale studies, test houses and field studies, have also been used to measure or estimate emission rates from indoor pollution sources.

Different types of source combinations can be modelled, but the equations may become complex. Also, a given source can have different emissions at different times depending on the conditions of the measurements. For example, the rate of emissions from a kerosene heater that uses a wick will depend on the burner setting, age of wick, type of fuel used, age of heater, and other

TABLE 24.1

Sources of indoor air contaminants

Sources	Contaminants
Consumer and Commercial Products	
Cleaners and waxes: aerosol bathroom cleaner; unpressurized aerosol window cleaner; liquid all purpose cleaner; powdered abrasive cleaner; dishwashing detergent; concentrated spot remover; liquid floor wax; aerosol furniture wax; aerosol and solid room deodorants; paste furniture wax; oven cleaners	*Particulates*: nonmetals (phosphates, other inert powders). *VOCs*: aromatic hydrocarbons (toluene, *p*-dichlorobenzene); halogenated hydrocarbons (perchloroethylene; methylene chloride; 1,1,1-trichloroethane); alcohols; ketones (acetone, methyl ethyl ketone); aldehydes (formaldehyde); esters (alkyl ethoxylate); ethers.
Paints and associated supplies: paints (oil, urethane, acrylic); varnishes and shellac; wood stains; paint thinners; paint brush cleaners; paint removers	*Particulates*: metals (lead, mercury, chromium). *VOCs*: aromatic hydrocarbons (toluene); aliphatic hydrocarbons (*n*-hexane, heptane); halogenated hydrocarbons (methylene chloride, propylene dichloride); alcohols; ketones (methyl ethyl ketone, methyl isobutyl ketone); esters (ethylacetate) Txi B, Texanal; ethers (methyl ether, ethyl ether, butyl ether).
Pesticides: termite treatment of homes; aerosol all-purpose household pesticides; roach killer (powder, liquid, spray); flea killer (powder, liquid dip, aerosol); mould and mildew inhibitors; house-plant insecticides; moth repellents; rodenticides (rat or mouse killer); fungicides (household disinfectants)	*Particulates*: nonmetals (sulphur, lime). *VOCs*: aliphatic hydrocarbons (ketone); aromatic hydrocarbons (xylene); halogenated hydrocarbons (chlordane, *p*-dichlorobenzene, heptachlor, chloropyrifos, diazinon); ketones (methyl isobutyl ketone); organic sulphur/phosphorus compounds (malathion).
Adhesives: rubber cement; plastic model glue; floor tile adhesive; ceramic adhesive; carpet adhesive; all-purpose adhesives	*VOCs*: aliphatic hydrocarbons (hexane, heptane); aromatic hydrocarbons; halogenated hydrocarbons; alcohols; organic nitrogen compounds (amines); ketones (acetone, methyl ethyl ketone); esters (vinyl acetate); ethers.
Cosmetic / Personal Care Products: perfume; personal deodorants (aerosols, solids); body powder (talc); shampoo and body soaps; rubbing alcohol; hair sprays	*VOCs*: alcohols (propylene glycol, ethyl alcohol isopropyl alcohol); ketones (acetone); aldehydes (formaldehyde, acetaldehyde); esters; ethers (methyl ether, ethyl ether, butylether).

(continued)

TABLE 24.1 (*continuation*)

Sources	Contaminants
Automotive Products: hydraulic fluids; motor oils; gasoline; automotive cleaners; automotive waxes	*VOCs*: aliphatic hydrocarbons (kerosene, mineral spirits); aromatic hydrocarbons (benzene, toluene, xylene); halogenated hydrocarbons (perchloroethylene); alcohols (ethylene glycol, isopropyl alcohol); ketones (methyl ethyl ketone); amines (triethanolamine, isopropanolamine).
Hobby Supplies: photographic chemicals; specialty adhesives; clay dust; wood filters	*Particulates*: nonmetals (fibres, smoke). *VOCs*: aliphatic hydrocarbons (kerosene, hexane, heptane); aromatic hydrocarbons (toluene, xylene, benzene); halogenated hydrocarbons (methylene chloride, ethylene chloride); alcohols (benzyl alcohol, ethanol, methanol, isopropyl alcohol); aldehydes (formaldehyde, acetaldehyde); ketones (methyl isobutyl ketone, acetone); esters [di-(ethylhexyl)phthalate] (DEHP); ethers (ethylene glycol ether); amines (ethylene diamine).
Furnishings and Clothing: carpets; upholstered furniture; plastic furniture; shower curtains; draperies; blankets; mattresses	*VOCs*: aromatic hydrocarbons (styrene, brominated aromatics), 4-PC (4-phenylcyclohexene); halogenated hydrocarbons (vinyl chloride); unsaturated hydrocarbons (4-vinylcyclohexene); aldehydes (formaldehyde); ethers; esters (DEHP).
Building sources	
Building Materials: pressed wood products construction adhesive; insulating materials; plastic piping; vinyl or plastic wall coverings	*Particulates*: fibres (fibreglass, asbestos). *VOCs*: aliphatic hydrocarbons (*n*-decane, *n*-dodecane); aromatic hydrocarbons (toluene, styrene, ethylbenzene); halogenated hydrocarbons (vinyl chloride); aldehydes (formaldehyde); ketones (2-propanone, 2-butanone); ethers; esters (urethane, ethylacetate, DEHP). *Radioactive Contaminants*: radon gas.
Heating, Ventilating and Air-Conditioning Systems: furnaces (carbon-based fuels); air conditioner reservoirs	*Inorganic Gases*: sulphur dioxide, nitrogen oxides, carbon monoxide, carbon dioxide. *Particulates*: nonmetals (fibreglass, moulds, smoke). *VOCs*: aliphatic hydrocarbons (methane). *Polynuclear Aromatic Hydrocarbons*: benzo(a)pyrene

(continued)

TABLE 24.1 (*continuation*)

Sources	Contaminants
Garages: vehicular exhaust; stored chemicals (pesticides, paints, solvents, gasoline)	*Inorganic Gases*: sulphur dioxide, nitrogen oxides, carbon monoxide. *Particulates*: metals (lead, nickel, platinum, palladium). *VOCs*: aromatic hydrocarbons (benzene); chlorinated hydrocarbons, other substituted hydrocarbons. *Polynuclear Aromatic Hydrocarbons*: benzo(a)pyrene
Combustion Appliances: unvented heaters (kerosene, gas); gas cooking stoves; wood-burning stoves and fireplaces	*Inorganic Gases*: sulphur dioxide, nitrogen oxides, carbon monoxide, carbon dioxide. *VOCs*: aliphatic hydrocarbons (propane, butane, isobutane); aldehydes (acetaldehyde, acrolein). *Polynuclear Aromatic Hydrocarbons*: benzo(a)pyrene. *Radioactive Contaminants*: radon.
Personal Sources	
Tobacco Smoke	*Over 3800 Compounds Including*: Inorganic Gases; Metals; Radioactive Contaminants; VOCs; Organic Nitrogen Compounds; Aldehydes; Ketones; Polynuclear Aromatic Hydrocarbons; Particulates.
Human or Biological Origin: animal faeces; pets; indoor plants (spores, pollen); metabolic products; pathogens	*Inorganic Gases*: ammonia. *Particulates*: nonmetals (dander, faeces). *VOCs*: aliphatic hydrocarbons (methane); aromatic hydrocarbons (toluene); aldehydes (acetaldehyde).
Outdoor Sources	
Soils and Rocks	*Radioactive Contaminants*: radon gas
Outdoor Air: industrial emissions; contaminated groundwater; vehicular exhaust; area sources (wastewater treatment plants, landfills)	*Inorganic gases*: carbon monoxide, sulphur dioxide, nitrogen dioxide, ozone. *Particulates*: metals (lead, other metals of respirable size range); nonmetals (fibres). *VOCs*: aliphatic hydrocarbons; aromatic hydrocarbons; halogenated hydrocarbons; aldehydes and ketones; alcohols; esters; ethers; organic nitrogen compounds; organic sulphur/phosphorous compounds. *Polynuclear Aromatic Hydrocarbons*

(continued)

TABLE 24.1 (*continuation*)

Sources	Contaminants
Potable Water volatilization of VOCs during showering, bathing, other uses of potable water	*Radioactive Contaminants*: radon gas. *VOCs*: halogenated hydrocarbons (1,1,1,-trichloroethane, chloroform, trichloroethylene, tetrachloroethylene).
Humidifiers	*Particulates*: aerosolized asbestos and minerals, biological contaminants.
Contaminated Groundwater seepage into basements	*Radioactive Contaminants* *VOCs*

Source: Adapted from U.S. EPA (1987).

factors. In addition, it should be noted that different methodologies for the measurement of the emission rate can yield different results. Table 24.3 gives examples of some emission rates for selected sources and contaminants.

24.2 SINKS AND DECAY RATES

A wide range of sources and source combinations are possible in indoor environments. Some sources emit intermittently in a random fashion (tobacco products), some emit periodically for limited and specified periods of time (gas heaters), some emit at rates that are high initially but reduced over short or long periods of time (waxes), presswood moth balls may emit at a more uniform rate over a period of time.

This section focuses on characterizing sources and sinks of materials found in indoor environments, and is based on an article by B.A. Tichenor (1992).

There are two main categories of indoor materials and furnishings: (1) dry materials, and (2) wet materials.

Dry materials (the majority of indoor construction and furnishings), include wood products, floor and ceiling, coverages, insulation materials, HVAC systems and presswood furnishings. Wet materials include paints, stains, varnishes, adhesives, caulks and sealants. These materials are applied "wet" and the resulting emissions are high and decay in short periods of time.

Materials present indoors in large surfaces may act as sinks, i.e. they adsorb and later re-emit vapour-phase (see Table 24.5 mostly for inorganic pollutants). Indoor sinks include carpets, rugs, walls, ceilings, HVAC systems and other furnishings.

TABLE 24.2

Source testing methods — chemical emission

Laboratory studies

Extraction and Direct Analysis

- Provide information on material composition
- Do not provide emission composition or emission rate data

Static Headspace

- Provide information on emission composition[1]
- Do not provide emission rate data

Dynamic chamber studies

Small Chambers

- Provide emission composition and emission rate data under controlled environmental conditions
- Chamber size may limit use for some material sources (for example, furniture, work stations)

Large Chambers

- Provide emission composition and emission rate data under controlled environmental conditions
- Large chambers may be required for evaluating emission during the application phase of wet materials

Full-scale studies

Test Houses

- Provide emission composition and emission rate data under "semi-controlled" environmental conditions; sink factors must be considered
- Very useful for validating exposed models using material emission rate

Field Studies

- Provide integrated emission profile of all sources and reemitting sinks under uncontrolled conditions
- Emission rate determinations are generally not possible
- Differentiating between source and sink emission is extremely difficult.

Source: P. Tichenor, B.A., private communication (1992).

[1] Attention has to be paid in case of products which are applied as thin films on a support for chamber studies. If headspace is measured for the bulk material considerable differences of the emission composition may result (A. Colombo, M. De Bortoli, H. Knoppel, H. Schauenburg, and H. Vissers, 1991. Small chamber tests and headspace analysis of volatile organic compounds emitted from household products. Indoor Air, 1, pp. 13–21).

TABLE 24.3

Sources emission rates for selected contaminants

Contaminant and material	Source emission rate	Reference
Formaldehyde		
medium density fibreboard[a]	1.5 mg/m^2/h	CPSC (1985)
particle board	0.560 mg/m^2/h initially 0.1–0.2 mg/m^2/h after 6 to 27 months	
textiles, resilient flooring	0.01 mg/m^2/h	Matthews et al. (1984)
carpeting, ceiling tiles, carpet pad	0.01 mg/m^2/h	
fibrous glass insulation, softwood plywood	0.02 mg/m^2/h	
hardwood panelling	0.11–0.28 mg/m^2/h	
particleboard underlay	0.30 mg/m^2/h	
industrial particleboard	0.31 mg/m^2/h	
medium density fibreboard	1.5 mg/m^2/h	
UFFI simulated wall panel	0.23 mg/m^2/h	
gas burner, gas oven	0.67 mg/h	
kerosene heater, convective	0.33 mg/h	
kerosene heater, radiant	1.3 mg/h	
cigarettes	0.5 mg/h	
oven[b]	11.4(9.9–14.2) µg/kal(n=5)	Traynor et al. (1979)
top burner[c]	5.2(2.0–12.0) µg/kal(n=5)	
Perchloroethylene[d]		
50% polyester, 50% rayon	220 µg/m^2/h	Tichenor and Sparks (1988)
rayon	55 µg/m^2/h	
polyester knit	430 µg/m^2/h	
acetate	6700 µg/m^2/h	
acrylic knit	56 µg/m^2/h	
wool blend	990 µg/m^2/h, 1200 µg/m^2/yr	
cotton	440 µg/m^2/h	
linen	570 µg/m^2/h	
65% polyester, 35% cotton	350 µg/m^2/h	
80% rayon, 15% flax	180 µg/m^2/h	
Para-dichlorobenzene	1.44 mg/cm^2/h	Clayton and Stephenson (1988)
Carbon Monoxide		
gas stove[e]	61.7±3.58 µg/kJ(n=192)	Borrazzo et al. (1987)

(continued)

TABLE 24.3 (*continuation*)

Contaminant and material	Source emission rate	Reference
oven	950(650–1600) μg/Kcal (n=6)	Traynor et al. (1979)
stove, top burner	890(720–1090) μg/Kcal(n=4)	
kerosene heater, blue flame	125.9 μg/kJ	Porter (1984)
reduced fuel consumption[f]	1.40 μg/kJ	
kerosene heater, white flame	23.9 μg/kJ	
reduced fuel consumption[f]	3.00 μg/kJ	
kerosene heater, convective	1.26±0.49 10^{-6} ft^3/Btu/h	Ritchie and Arnold (1984)
kerosene heater, radiant	1.16±0.48 10^{-6} ft^3/Btu/h	
wood stove, airtight	10–140 cm^3/h(n=7)	Traynor et al. (1987)
wood stove, not airtight	220–1800 cm^3/h(n=4)	
Carbon Dioxide		
oven	200,000 μg/kcal (195,000–250,000) μg/kcal(n=6)	Traynor et al. (1979)
stove, top burner	205,000 μg/kcal(196,000–217,000) μg/kcal(n=3)	
kerosene heater, convective	1376±200 10^{-6} ft^3/Btu/h	Ritchie and Arnold (1984)
kerosene heater, radiant	1379±240 10^{-6} ft^3/Btu/h	
Nitrogen Dioxide		
gas stoves	10.5±1.43 μg/kJ(n=192)	Borrazzo et al. (1987)
oven	62(44–74) μg/kcal(n=11)	Traynor et al. (1979)
stove, top burner	85(69–100) μg/kcal(n=4)	
kerosene heater, blue flame	4.7 μg/kJ	Porter (1984)
reduced fuel consumption[f]	1.15 μg/kJ	
kerosene heater, white flame	7.2 μg/kJ	
reduced fuel consumption[f]	1.61 μg/kJ	
kerosene heater, convective	0.211±0.042 10^{-6} ft^3/Btu/h	Ritchie and Arnold (1984)
kerosene heater, radiant	0.025±0.008 $10^{-6}$$ft^3$/Btu/h	
Nitric Oxide		
gas stoves	16.8±1.37 μg/kJ(n=192)	Borrazzo et al. (1987)
oven	29(14–50) μg/kcal(n=11)	Traynor et al. (1979)
stove, top burner	31(21–47) μg/kcal(n=4)	

(continued)

TABLE 24.3 (*continuation*)

Contaminant and material	Source emission rate	Reference
kerosene heater, blue flame	0.1 μg/kJ	Porter (1984)
reduced fuel consumption[f]	—	
kerosene heater, white flame	14.4 μg/kJ	
reduced fuel consumption[f]	0.52 μg/kJ	
kerosene heater, convective	$0.411 \pm 0.105\ 10^{-6}$ ft^3/Btu/h	Ritchie and Arnold (1984)
kerosene heater, radiant	$0.014 \pm 0.008\ 10^{-6}$ ft^3/Btu/h	
Sulphur Dioxide		
oven	0.8(0.5–1.0)μg/kcal(n=11)	Traynor et al. (1979)
stove, top burner	0.8(0.6–0.9)μg/kcal(n=4)	
kerosene heater, convective	$0.078 \pm 0.010\ 10^{-6}$ ft^3/Btu/h	Ritchie and Arnold (1984)
kerosene heater, radiant	$0.079 \pm 0.017\ 10^{-6}$ ft^3/Btu/h	
Hydrogen Cyanide		
oven	1.8(1.6–2.3) μg/kcal(n=3)	Traynor et al. (1979)
stove, top burner	0.07 μg/kcal	
Total Suspended Particulates		
wood stove, airtight	2.5–8.7 mg/h(n=7)	Traynor et al. (1985)
wood stove, not airtight	16–230 mg/h(n=4)	
no stove, background	1.1,1.6 mg/h(n=2)	
Benzo(a)pyrene [b]		
wood stove, airtight	0.02–0.76 μg/h(n=7)	Traynor et al. (1985)
wood stove, airtight	2.2–57 μg/h(n=4)	
no stove, background	<0.01μg/h(n=2)	

[a] Based on CPSC analysis of existing data.
[b] Oven operated for 1 h at 180°C (350°F).
[c] Operated with water-filled cooking pots.
[d] Emission from dry cleaned fabrics. The reader is cautioned by Tichenor et al. to use results in a qualitative way, not for emissions calculations.
[e] Emission factors are based on statistical analysis of existing data by various investigators.
[f] 75% of normal fuel consumption.
[g] Outdoor penetration factor of 0.48.
[h] Outdoor penetration factor of 0.30.

TABLE 24.4

Sink testing methods — organic chemicals

Packed columns	Provide data on mass adsorbed but not on adsorption and desorption rates Only applicable to high surface area material (for example, carpet fibres)
Dynamic chamber tests	Provide data on mass adsorbed and rates of adsorption and desorption under controlled environmental conditions
Test house studies	Provide data on rates of adsorption and desorption under "semi-controlled" environmental conditions for multiple sink materials Useful for validating dynamic chamber sink results using IAQ models

Source: Tichenor, B.A., private communication (1992).

The behaviour of indoor sources and sinks is determined by the following agents: (1) mass transfer; (2) environmental variables; and (3) material characteristics/composition. Mass transfer processes include evaporation, adsorption, absorption, diffusion and convection. These processes govern emissions of vapour-phase organics from indoor materials and sink interactions.

The source emission rates and sink behaviour of organics associated with indoor materials depend upon the following environmental parameters: temperature, humidity, air exchange rate, and velocity and turbulence of the boundary layer of air above the emitting surface. Clearly, the characteristics and composition of indoor materials have a major impact on their emission and sink/decay rates. The number and types of chemicals emitted are of obvious importance. Vapour pressure, diffusion coefficients, molecular weight and size affect emission characteristics. For sink materials, the type of material and type of surface (smooth, rough, fleecy) are significant. Additionally, the surface area and masses of material used affect the total mass emitted from sources and the total mass adsorbed on and desorbed from sinks.

Sink evaluation methods parallel the methods used to determine emission rates. Methods used for sink evaluation are summarized in Table 24.4.

IAQ models constitute the research tool that links sources/sinks with physical indoor quantities, occupant activity patterns and HVAC system operation to calculate indoor concentrations.

Good agreement between data and model prediction illustrates that the combination of well designed models and sound input data provides a powerful

prediction tool for IAQ calculations. Moreover, the figure demonstrates that the effect of sinks is to prolong high-level indoor concentrations; this is especially true for wet sources. Clearly, knowledge of source strengths and sink behaviour is necessary as it affects the ability of the indoor air quality expert to predict and eventually control indoor air pollutant concentration.

The removal term, R (sink), is often unknown, and in general R may depend on the physical and chemical characteristics of the interior space. The removal term may be incorporated into the source term to give a net source term or it can be evaluated as a function of the decay rate (λ) or deposition velocity (v_{dep}).

TABLE 24.5

Decay rates for selected contaminants

Contaminant and material	Decay rate	Reference
Nitrogen Dioxide based on steel chamber studies		
highest removal		Billick and Nagda (1987)
unpainted plaster board	>8.4/h	
cement block	8.4/h	
wool carpet	6.0/h	
high removal		
used bricks	4.2/h	
masonite	4.1/h	
polyurethane foam	3.7/h	
moderate removal		
cotton/polyester bedspread	2.7/h	
painted plasterboard	2.6/h	
plywood (1/4 in)	2.6/h	
acrylic fibre or nylon carpet	1.9–2.0/h	
ceiling tile (wood fibre)	1.9/h	
all vinyl wall-covering with proper backing	1.9/h	
low removal		
particleboard	0.7/h	
ceramic tile	0.7/h	
cotton terrycloth	0.3/h	
wool(80%) polyester(20%) fabric	0.3/h	
window glass	<0.1/h	
galvanized metal duct	0	

(continued)

TABLE 24.5 (continuation)

Contaminant and material	Decay rate	Reference
formica countertop	0	
vinyl or asphalt tile	0	
Formaldehyde		
based on concentration measurements	–0.0182 to –0.0231/month (av. = –0.0195)	Versar (1985)
based on "fast" chamber studies		U.S. CPSC (1985)
particleboard underlay	–0.078/mo (av.)	
panelling	–0.093/mo (av.)	
medium density fibreboard	–0.053/mo (av.)	
based on concentration data for Houston	0.0013 to –0.0028/mo (av.)	Stock and Sixto (1985)
Particles <1 μ diameter	0.05/h	Dockery et al. (1980)
Radon	1.258×10^{-4}/min	

Table 24.5 provides examples of decay rates and deposition velocities for selected contaminants. The caveats for the source term also apply to the removal term. Removal expressed as a function of the decay rate, area of the sink term, and the contaminant mass (VC_i) is given by:

$$R = \lambda VC_i$$

Removal expressed as deposition is given by:

$$R = v_{dep} \, a \, C_i$$

The relationship between λ and v_{dep} is then:

$$v_{dep} = \lambda \frac{V}{a}$$

The literature also offers few examples of deposition velocities for a few indoor pollutants on specific surfaces and indoor areas (see Table 24.6).

TABLE 24.6

Deposition velocities for selected contaminants on various surfaces and indoor areas

Contaminant and material	Deposition velocity	Reference
Sulphur Dioxide		
carpet		Walsh et al. (1977)
pink tufted (pH=4.1)		
pile	0.031 cm/sec	
backing	0.006 cm/sec	
orange (pH=3.5)		
pile	0.053 cm/sec	
backing	0.014 cm/sec	
green (pH=6.7)		
pile	0.074 cm/sec	
backing	0.016 cm/sec	
white (pH=9.2)		
pile	0.072 cm/sec	
backing	0.012 cm/sec	
mustard (pH=4.8)		
pile	0.022 cm/sec	
backing	0.017 cm/sec	
dark green (unused) (pH=4.3)		
pile	0.020 cm/sec	
backing	0.011 cm/sec	
dark green (used) (pH=4.5)		
pile	0.029 cm/sec	
backing	0.014 cm/sec	
embossed vinyl	0.096 cm/sec	Walsh et al. (1977)
wallpaper	0.007 cm/sec	
paint		Walsh et al. (1977)
gloss	0.033 cm/sec	
emulsion	0.128 cm/sec	
Nitrogen Dioxide		
flooring materials		
carpet	0.55–3.46 m/h(n=5)	Miyasaki (1984)
tatami facing	0.46 m/h	

(continued)

TABLE 24.6 (*continuation*)

Contaminant and material	Deposition velocity	Reference
floor sheet	0.04 to 0.09 m/h(n=3)	
plastic tile	0.12 m/h	
ceramic tile	0.15 m/h	
bath mat	1.97 m/h	
needle punch	0.47 m/h	
wall materials[a]		
wall paper	0.06, 0.08 m/h(n=2)	
printed plywood	0.05 m/h	
ceiling materials[a]		Miyazaki (1984)
insulation board	4.31 m/h	
painted insulation board	2.13 m/h	
plaster board	0.66 m/h	
wooden cement board	1.17 m/h	
asbestos cement board	1.47 m/h	
Nitric Oxide		
flooring materials[a]		Miyazaki (1984)
carpet	1 0.01 m/h	
tatami facing	0.01 m/h	
floor sheet 1	0.00 m/h	
needle punch	0.03 m/h	
ceiling materials		Miyazaki (1984)
insulation board	0.00 m/h	
painted insulation board	0.04 m/h	
plaster board	0.11 m/h	
wooden cement board	0.12 m/h	
Ozone		
fabrics		Sabersky et al. (1973)
cotton muslin	0.88–6.52 cm/min	
lamb's wool	0.24–6.34 cm/min	
nylon	0.03–1.92 cm/min	
linen	0.33–0.56 cm/min	

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PART VI
Indoor Air Quality Investigations in Buildings

This part discusses the methods available for performing indoor air quality investigations in buildings. One chapter is concerned with the general study approach and the engineering analysis of buildings. Another chapter deals with the strategies for measuring indoor air pollutants and air exchange rate. The last chapter systematically describes the methods for sampling and analysis of indoor air pollutants.

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Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 25

Methodology of Building Investigation I: Data Collection and Analysis

25.1 GENERAL REMARKS

There are different reasons for making an indoor air quality investigation in a given indoor environment, one of the most frequent being to respond to a complaint about bad indoor air quality. Depending on the nature of the complaint, the number of people affected, and the type of room or building involved, various tools can be used to carry out an indoor air quality investigation.

While some indoor air problems may be solved using very simple tools, others may require sophisticated knowledge and instruments. For example, it may be easy to identify the source of cooking odours that are annoying the occupants of a nearby office and solve the problems by installing appropriate exhaust fans. On the other hand, attempts to identify the reasons for the headache reported by the occupants of a room may not lead to conclusive results despite detailed medical examination of the persons and thorough analysis of indoor air for chemical and biological contaminants.

Building investigations may be classified into two major groups:

1. Investigations originating from complaints of building occupants on indoor air quality or the presence within the building of pollution giving rise to health concerns. Buildings presenting these kind of problems are called "problem buildings" and require a "problem building investigation".
2. Investigations aimed at surveying the building's stock or particular features of it or investigations aimed at developing analytical methodologies.

A healthy building may be defined as a building where agents that cause health effects are absent or at low levels, and the great majority of the occupants feel comfortable.

Conversely, the definition of "problem building" includes two conditions: buildings where occupants complain about the air quality or manifest air

quality-related symptoms and buildings where there are no complaints but agents that may cause risk are present (i.e., asbestos, radon, legionella, etc.).

The criterion for the health risk factors is compliance with air quality guideline values, which are not, however, available for every pollutant and even less for mixtures of pollutants.

The criterion for comfort is the judgement expressed by the occupants: in this case it has to be decided at what percentage of satisfied occupants the comfort criterion is considered to be met.

The strategy for investigating indoor air quality in a building strictly depends on the ultimate targets, be they air concentrations of pollutants, ventilation rates to be met, or positive judgements by the occupants to be achieved.

Generally, problem building investigations are performed stepwise. Various strategies have been developed using different step sequences (ECA, 1989a).

Several guidelines and recommendations have been developed for building investigations: the reader may refer to the U.S. EPA Building Air Quality (U.S. EPA, 91a), the U.S. NIOSH Guidance for Indoor Air Quality Investigations (U.S. NIOSH, 1987), the IRSST Strategy for Studying Air Quality in Office Buildings (IRSST, 1989), and the Nordtest Report on Indoor Climate Problems: Investigation and Remedial Measures (Nordtest, 1993).

In the following, we will discuss a four-step procedure of investigation (see Table 25.1) which is generally adaptable to any building. The purpose is to indicate a systematic method of study by which specific problems can be identified and solved. Engineering analysis is a particularly useful tool for identifying deficiencies in the building design, construction and operation.

25.2 COLLECTION OF GENERAL DATA

The first step aims at collecting general data that are indispensable to characterize the building and the site of its location (Table 25.2). It involves discussion with responsible and affected parties. It usually involves phone calls, meetings, and interviews. Usually it will also include a walk-through of the building and inspection of the affected spaces and ventilation system(s) or other equipment. The purpose of this step is to define the scope, goals, and objectives of the investigation and to establish the time frame, nature, and scope of the building problems. Ideally, it includes formulation of hypotheses that can be tested in the following steps.

Based on this step, skilled investigators can often identify building problems or contaminant sources that may be responsible for the air quality

TABLE 25.1

Essential steps in indoor air quality investigation

Step	Tools	Information provided
1. General data collection	Consultation/Meetings/ Initial walk-through/Visual inspection	General information
		Condition of room and equipment
		Condition of heating/cooling and ventilation system
		Number/activities/behaviour of occupants
2. Collection of information from occupants	Questionnaire	Detailed record of occupant complaints
	Diaries	Time and place of occurrence of symptom(s)
		Detailed list of occupant activities
	Interview	Type and severity of symptom(s)
3. Collection of on-site data	Detailed inspection	HVAC condition
	Basic measurements	Temperature, humidity, CO_2
		List of sources
		Potential pollutant(s)
Engineering analysis is an alternative or a complementary tool to phase 2 or 3		
4. Advanced measurements	Instrumentation	Air exchange rate
		Concentration of chemical/biological pollutants in the air
	Medical examination of occupants	Exclusion of other influencing factors
	Determination of clinical parameters	Medical confirmation of symptoms

problems and/or reported occupant health and comfort symptoms. In many cases, addressing these building problems will resolve the occupant health and comfort problems. In some cases, however, the nature of the occupant health problems warrants immediate medical attention. Examples include reports or suspected cases of Legionnaires' disease, Pontiac fever, hypersensitivity pneumonitis, or epidemic infectious disease.

TABLE 25.2
General data collection

Main features of the building	Characteristics of the area and nature of the soil
	Maps and use of the indoor spaces
	Building materials
Activities and occupants	
Inventory of potential sources of indoor pollution	Building materials, carpeting, furniture
	Instruments, equipment, HVAC
	Human activity
	Outdoor environment
Programme of maintenance operations	Cleaning
	Periodic maintenance
	Extraordinary interventions

After the completion of the first phase, some investigators may choose to administer questionnaires, conduct interviews, or use other techniques to develop a more systematic picture of the nature and distribution of health and comfort problems (step 2). Alternatively, an engineering analysis can be conducted before further investigations are planned.

Some investigators will augment their professional team to include experts in microbiology, volatile organic chemicals, ventilation, psychology, or survey research. It is important to note that the nature of the investigation and the problem resolution generally reflects the expertise of the lead investigator. Dr. Lars Mølhave of Aarhus, Denmark, pointed this out at "Indoor Air' 87" in Berlin. Engineers find engineering problems, physicians find medical problems, and psychologists focus on perceptual and attitudinal problems.

If at this point in the investigation it is not clear that certain identified sources or environmental controls should be remedied, then a further phase is planned. While planning this phase, the engineering analysis should be considered. It facilitates a more detailed and systematic understanding of the building and especially the environmental control systems. It provides an opportunity to investigate the building in a non-intrusive manner that is often more productive than measurement or inspection of the occupied spaces. We will return to the engineering analysis in more detail after briefly reviewing the remaining phases of the general investigation sequence.

25.3 COLLECTION OF ON-SITE DATA

The third step is on-site inspection and direct verification of the conditions of the building (see Table 25.3). It is usually referred to as the "qualitative diagnostics" and involves detailed inspection of the facility and may involve measurement of basic environmental parameters. It may also verify the proper functioning of the building's environmental control systems (HVAC, lighting and acoustic). Most investigators will measure temperature, humidity, and, perhaps, CO_2 during this phase. This phase almost always includes very detailed inspection of the HVAC equipment and the occupied spaces. Investigators identify in detail specific characteristics of important contaminant sources and the building-and personnel-based environmental control systems.

TABLE 25.3

On-site data collection

Inspection of the building	HVAC
	Environment
	Equipments
	Furnishings, carpeting, textiles
Detailed examination of the building	

25.4 ENGINEERING ANALYSIS

One of the most important and effective tools for investigating problem buildings is "engineering analysis". Its use often facilitates identification of deficiencies in the building design, construction or operation that contribute to indoor air quality problems. Based on engineering analysis, building operators can often resolve problems more economically and quickly than they can determine the precise causes of occupant complaints by air sampling or occupant surveys. In this section we discuss engineering analysis in the context of the generic environmental problems.

In its most widely advocated form, a building investigation (or building diagnostics) follows several stages or phases. The least expensive and most productive portions of these investigations are usually the initial ones. Thus, environmental problems in buildings that cannot be resolved during the first or second phase are less likely to be resolved by extensive and expensive measurements which are usually part of a more advanced phase.

The engineering analysis is conducted as part of the qualitative diagnostics, as an alternative or complement to phase 2 or 3.

25.4.1 General

The purpose of the engineering analysis in a problem building investigation is to determine the ability of the building or its design to handle the contaminant and thermal loads. Investigators who by-pass this phase of a building investigation often learn later that they could have identified probable causes of occupant symptoms and comfort complaints far earlier and more economically if they had performed a building engineering analysis.

Many indoor environmental problems occur because buildings are not operating according to the design. This can result from poor design, construction or operation, or from an improper use of the building by the occupants. It may result from poor communication of the design intent to the operators, from inadequate training of building operators, from a lack of sufficient operator control over building operations, or from a problem in the building equipment. Whatever its cause, it is extremely common in problem buildings. Engineering analysis can be a cost-effective way of determining the factors that contribute to the air quality (or other environmental) problem and practical solutions to the problems.

Table 25.4 shows some possible causes of building environmental problems that can be identified by engineering analysis. Notice that the problems can begin at any stage of a building's life-cycle from design to use.

In many cases, buildings (as designed) are simply not able to handle the loads. The engineer or designer may not have received sufficient information on the actual loads when designing the building. This is common in tenant-occupied buildings, particularly in speculative office buildings, but it is also common in large public or private institutions. The long lag-time between design and occupancy means that planning during design is often out-dated by organizational changes that occur during the construction period.

If the loads have changed since the building was designed, without sufficient consideration by the facility managers, spaces may be used too intensively or for purposes for which they were not designed. This often happens when there is a change in tenants, when buildings are remodelled, or when building occupants move or increase their density within their spaces. It can result from a change in occupant activities, equipment, or schedules. It can also happen when there is change in procedures, equipment and materials used for housekeeping, pest control, or building equipment maintenance.

The loads of concern relate to air pollutants, thermal conditions, illumination requirements and sources, noise, vibration, among others. A complete

TABLE 25.4

Problems and probable causes identified during engineering analysis

Project phase	Problem type	Possible cause
Design	Insufficient load information	Unknown tenancy or occupant activities
	Insufficient load information	Poor client–designer–engineer communication
	Inappropriate capacity (too large or small)	Improper design
Construction	Installation of improper components	Poor construction activities, unavailable components, insufficient overseeing by designers, construction managers, or inspectors
	Improper installation	Poor workmanship, improper commissioning
Operation	Insufficient pollutant removal capacity	Inadequate design, or system control program
	Inadequate system performance	Insufficient or inappropriate operation/maintenance program, schedule, modes
	Unaddressed load changes	Inadequate evaluation of intended space use changes, changes in density of occupants, or changes in equipment and emissions

engineering analysis must consider air quality, thermal, and other environmental loads just as an adequate building must be capable of handling them all. They can be either assumed or projected loads used for purposes of design, or they can be calculated or measured loads based on analysis or observations of the building.

When investigating possible air quality and climate problems in buildings, it is extremely important to determine the conformance of the actual building to the building design, and the ability of the designed or actual building to handle the actual loads. In fact, a "problem building" by definition, is one that does not adequately handle all of the loads. Engineering analysis is a direct method of determining the way in which the building fails to handle the loads imposed on it.

25.4.2 Steps in an engineering analysis:

Determine scope of analysis; identify building problem areas

- Determine activities, use patterns, densities (current and historical) in the building
- Identify significant modification to building shell, equipment, spatial configuration and allocation
- List possible contaminants or contaminant types based on complaints, occupant activities, other information available
- Identify potential problematic contaminants, that is those that could plausibly result in complaints
- Identify probable contaminant sources, driving forces, and pathways: sources may be local or distributed, and they may be continuous or intermittent; driving forces for air pollutants are pressure differences between spaces; pathways are openings, conduits, shafts, chases, cracks, etc.
- Determine thermal loads due to climate, occupants, equipment and its use patterns
- Determine environmental control system capacities: cooling, heating, ventilating
- Identify system operating schedules, programmed modes, and control scheme
- Evaluate suitability (capacity program) of system for identified loads. This may involve a computer simulation of building operation, simple manual calculations, or a combination of the two.

Information sources for an engineering analysis:

The engineering analysis requires a review of the following (depending on their availability):

- plans (construction and "as-built" or record drawings)
- specifications
- design calculations, program information, activity inventory
- installed equipment
- operation procedures
- building manual
- calculated loads (assumed or measured; past, present or future); included are both thermal and air pollution loads.

Define scope

The first step is to identify the relevant systems and building areas for the analysis. This is done to limit the scope of the analysis to those parts of the building relevant to the hypothesized problem. Hypotheses are formed by discussion with the client, building manager, building engineer, or other parties involved in requesting the investigation. Written and graphic materials are then obtained as available.

Design assumptions

Additional information should be gathered on the assumptions designers made during building design: what were the assumed or projected uses of the building; what were the loads attendant to those uses; what instructions did the designers receive regarding the occupant density, activities, schedules, pollution source-generating activities and equipment, and other "programming" information on which the design was based? Investigators may obtain this type of information from other design documents and available notes from the program for the building design. In many buildings, very little such information is actually communicated to the designer from the owner or other client. In many cases such information is not readily available and empirical determinations must be relied upon to form the basis of the engineering analysis.

Identify modifications

It is important to determine what the designed building was, whether it was actually constructed, and whether it has been modified since. "As-built" or "record" drawings prepared during and after the construction are useful at this stage, but they are not always available. Original design drawings and specifications, equipment data sheets from manufacturers, and other product and equipment submittals will be useful in determining the nature of the system. Interviews with available designers, contractors, building managers and engineering personnel may be helpful in identifying documents and supplementing the information they contain.

Identify potential contaminants

Contaminant and thermal loads should be determined as carefully and completely as possible in order to evaluate the theoretical ability of the building design and construction to handle the loads imposed upon it. On the basis of the walk-through and initial phase consultations, a listing of possible contaminant sources and thermal loads should be available. Additional potentially important contaminants may be identified by occupants or operator reports. Contaminant sources outside the building should not be ignored.

Identify possible problem contaminants

Investigators will rely on their experience and observations to determine which contaminants are potential contributors to poor environmental quality. The sources and pathways of distribution for these contaminants should be identified to focus the analysis on the most plausible causes of the problems. Keep in mind that pressure differences between interior spaces or between the building and the exterior are the driving forces for the movement of contaminants from one location to another.

Determine thermal loads

External and internal sources of heat or cold determine the building's thermal environment. Office equipment and other appliances have become important sources of thermal loads as they have proliferated in modern offices. It will be important to verify system capacity against the actual loads imposed on it.

Determine equipment capacities

During the walk-through and HVAC inspection, equipment sizes and capacities will have been noted. A walk-through inspection with notations of installed equipment labels permits verification of the actual construction. Equipment capacities are often listed on labels attached to the equipment. Information on loads in the spaces of concern should have been obtained during the normal walk-through procedures in the investigation stages previous to the engineering analysis. If this information has not been obtained, has changed, or is otherwise incomplete or unreliable, it should be obtained through systematic observation of the occupied spaces of concern (including equipment and storage rooms).

Identify system operating schedules

Operational schedules and sequences are required to create a complete data file for simulation of the actual conditions and loads. Often building operational hours are simply inadequate or inappropriate for the actual use patterns. While occupant-activity related loads may correspond to occupancy hours, building materials, furnishings and equipment will be emitting chemicals continuously. Systems that operate only during "normal" work-day hours may require extended schedules to adequately address building loads.

The investigators should determine the plan for the actual operation and maintenance of the building and attempt to verify its implementation based on available documentation. Computer-controlled HVAC systems usually produce records, either electronic, hard copy, or both. Maintenance records will reveal the actual intervention carried out.

Verify system performance against criteria

By calculation, sometimes by computer simulation, the building performance can be evaluated against the criteria established for the building. This will allow determination of the adequacy of the system in-place for the loads. The simulation may be a simplified one for only a certain space where occupant health and comfort problems are prevalent, or it may be an entire zone of the building ventilation system. It will provide a reasonable evaluation of the adequacy of the ventilation system and other environmental control systems.

Report

Upon completion of engineering analysis, investigators prepare a report with an executive summary, if warranted. The report should contain a complete listing of all the information obtained as input to the analysis and a detailed description of the simulation process. Where appropriate, it should contain recommendations for remediation of deficiencies.

The documentation of the process becomes a valuable asset for building operators and engineers. It will provide the basis for future analyses that may eliminate or reduce the occurrence of environmental problems, More often than not, the absence of clear, comprehensive information is one of the key factors in the occurrence of building environmental problems.

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Indoor Air Quality
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Chapter 26

Methodology of Building Investigation II: Strategies for Measurement of Indoor Air Pollution and Air Exchange Measurements

26.1 INTRODUCTION

In contrast to the situation encountered in outdoor air analysis where the concentrations of a number of pollutants are monitored continuously, indoor air pollution is not measured on a continuous basis in view of the large number of individual indoor spaces with different sources and pollution patterns. In general, it is also not possible to introduce bulky or noisy analytical equipment into indoor environments. As a first consequence of this situation, analysis of indoor pollutants is usually broken down into a sampling step which is performed on-site with relatively small, silent and inexpensive equipment, and a separation/identification step which is performed in the laboratory using complex instrumentation if necessary. This section refers to the sampling step.

As a further consequence, indoor air analysis often has to solve the difficult task of representatively characterizing the air in enclosed spaces by only a small number of samples or even by just one sample. Thus it is clear that the sampling strategy is of greatest importance in carrying out indoor measurements.

The sampling strategies described in this document apply to the case where a certain knowledge of the potential sources and pollutants is available. For building investigations aimed at identifying causes of complaints, this knowledge has to be obtained at previous steps of the investigation as described in Chapter 25. Developing a sampling strategy means to answer the questions: when, how often, for what period of time, and where samples should be taken. The answer to these questions depends essentially on three parameters:

(a) the dynamic character of indoor air pollution, i.e. its particular features causing a great variability of indoor pollution levels in space and time;
(b) the objective of a measurement;
(c) the pollutant or pollutant class of interest.

Due consideration of the dynamic character of indoor air pollution caused by the variability of sources of pollution, and of ventilation and climatic conditions, has to be given at the origin of any design of a sampling strategy and influences the answers to all the above questions.

It depends on the objective of an investigation which pollutants have to be measured and whether maximum or mean concentrations or the variation of concentration with time have to be determined.

Interest in a given pollutant decides on the sources which have to be considered. Whether these sources emit pollutants in a continuous (e.g., building materials) or discontinuous way (e.g., human activities) will influence the decision on the time, duration and frequency of sampling.

Because of the strong impact of the sampling strategy on the result of any indoor measurement and the lack of any general agreement — be it on the national or the international level — on what sampling strategy should be used under what conditions, an expert group was established in 1988 by the European Concerted Action "Indoor Air Quality and Its Impact on Man" to address this question. Chapter 26 and 27 are mostly based on the report of this expert group (ECA, 1989b).

There is a large variety of situations and pollutants in the different types of indoor spaces, which would call for quite a large set of sampling procedures to be established. However, there are a number of general considerations that apply to most circumstances. These general considerations cover the dynamics of indoor air pollution and the questions of why, when, how long, how often and where sampling has to be carried out. Chapter 26 deals with these topics and also includes a brief introduction to air exchange rate measurements and to basic requirements for quality assurance.

In principle, the information given below has been prepared for an assessment of chemical substances. However, much of the content of this chapter may also apply to microbiological indoor pollutants, although in the case of these agents, temperature and relative humidity will probably have a much more pronounced influence on the pollutant level than in the case of chemical substances. The specific problems concerning the sampling and detection of microorganisms are discussed in aSection 27.5.

26.2 THE DYNAMICS OF INDOOR AIR POLLUTION

Indoor air pollution in non-industrial buildings is a dynamic phenomenon rather than a static one. The dynamics are characterized by the variability of source emissions, types of different indoor spaces, and different ventilation and climatic conditions. The situation is further complicated by the various types of pollutants. These are the following:

- gases and vapours (inorganic and organic)
- particulate matter
- radioactive particles/gases (radon and its daughters)
- biological agents

The sources which contribute to indoor air pollution are summarized in Table 26.1. They can be divided into those with continuous emissions (long-term emission, constant source strength) and discontinuous emissions (short-term emission, variable source strength). These two groups may further be subdivided into those emitting regularly (R, constant time pattern) and those emitting irregularly (I, variable time pattern) (Seifert and Ullrich, 1987). While the magnitude of emissions from continuous sources often depends on temperature, relative humidity, and sometimes air velocity, and varies within a time-scale of months, discontinuous emissions are much more time-dependent and may change within hours or minutes.

An understanding of the dynamic character of indoor air pollution is important in order to design a proper sampling strategy as well as to evaluate the results correctly. In selecting the parameters of the sampling strategy one should also take into account that parameters not related to the measurement itself (e.g., building and occupant-related variables, such as outdoor sources, variability in source strength, ventilation rate, etc.) have an impact on the measured concentration. Table 26.2 illustrates how all these parameters are interrelated.

26.3 SAMPLING OBJECTIVES

As the sampling objective determines the procedure to be followed during sampling, it is of utmost importance to define it clearly before starting sampling. Among the possible objectives, the following play the most important role: determination of population exposure, reaction to complaints (including the identification of emission sources), control of the success of mitigation measures, and check of compliance with reference or guide values.

To determine the exposure of the population, either average or maximum exposure levels may be needed. If a sufficiently large number of cases chosen at random is investigated, a frequency distribution of the concentrations can be established, from which both the "normal" and the extreme concentrations can be derived. A compilation of indoor VOC data (WHO, 1989) shows that population exposure to VOC is comparable in different countries.

In many instances, the air inside an enclosed space is analysed following a request from complaining occupants. In most of these, acute effects, such as eye irritation, irritation of mucous membranes or bad odour perception, play

TABLE 26.1

Sources of indoor air pollutants and their tentative assignment to emission types (ECA, 1989b)

Source	Type of emission		Pollutant			
	Continuous	Intermittent	VIC	VOC	PM	RA
Building related:						
Building materials	R		+	+	+	(+)
Renovation of building	R,I			+	+	
Furnishings	R			+	+	
Ventilation system	R,I	R,I		+	+	
Polluted soil/ground	R,I			+		+
Fungi, mould, mites	R			+	+	
Product-related:						
Household and consumer products		R,I		+		
Cleaning procedures		I	(+)	+		
Hobby works		I	+			
Various activities (cooking, smoking, etc.)		I	+	+	+	
Others:						
Bioeffluents		I		+		
Outdoor air, traffic		I	+	+		
Industries in the same building		R,I		+		
Occupational exposure (with subsequent exhalation and desorption from clothes and body)	R,I		(+)	+		
Car-related activities		I		+		
Combustion, heating		R,I	+	+	+	
"Negative" sources:						
Ventilation	R,I	R,I	+	+	+	+
Sinks (decay, fleecy surfaces)	R,I		+	+		
Deposition	R	I			+	(+)

R = Regular; I = Irregular; VIC = Volatile inorganic compounds; VOC = Volatile organic compounds; PM = Particulate matter; RA = Radon and daughters.

TABLE 26.2

Interrelationship between different indoor parameters illustrating the dynamics of the indoor environment (ECA, 1989b)

Parameter	Outdoor air	Ventilation rate	Ventilation efficiency	Source strength	Sinks
Sampling-related					
time of sampling	+			+	
duration of sampling	+	+		+	
frequency of sampling	+			+	
sampling location	+		+	+	+
Building-related					
age of building		+		+	+
source strength		+	+	+	
temperature				+	
rel. humidity	+			+	
Occupant-related					
activity pattern		+		+	
number of persons		+	+	+	+
Other					
season	+	+			

a dominant role. To check if such complaint may be caused by a pollutant, it is often most useful to get information on the maximum concentration likely to occur in a room, i.e. the worst-case situation. Such worst-case considerations are especially important if groups at risk are to be looked at. They can be simulated by reducing the ventilation rate or by changing other indoor climate parameters, provided these parameters increase the emission rate. As an example, the increase of formaldehyde concentrations at higher temperature or relative humidity may be mentioned. However, care has to be taken not to choose conditions never encountered in practice.

Because of the dynamic character of indoor air pollution, correlations between pollutant concentrations and complaints of building occupants may be obscured if the measurements of both, i.e. the questionnaire inquiry of symptoms and the sampling of pollutants, are not performed simultaneously (Hodgson et al., 1991).

If significant levels of pollution are observed in a room, it is generally desirable to know the sources in view of mitigation measures. As more than one single building material, piece of equipment, etc. may be responsible for the emissions, the sampling strategy would have to be adjusted properly (see Chapter 26.6).

Controlling the success of mitigation measures does not call for special conditions provided the two measurements (one before and the other after the mitigation measures have been taken) are carried out under comparable conditions.

Table 26.3 presents a matrix which relates the sampling objectives and different parameters to be considered when developing an appropriate sampling strategy.

Because in many cases the ultimate goal of indoor air measurements is an evaluation of potential negative health impacts, the sampling procedure may have to be adjusted to these needs. Table 26.4 gives a summary of the interdependencies between the potential adverse health effects, the information one would like to have available on the exposure situation, and the sampling procedures to be applied.

As concerns the checking of compliance with a given guide value, the boundary conditions defined together with this value have to be respected. If such boundary conditions have not been defined, the sampling strategy should be adjusted according to the rules given in the following sections.

TABLE 26.3

Matrix relating sampling objectives and different parameters to develop sampling strategy (ECA, 1989b)

Sampling objective	Sampling condition	Type of sampling	Duration of sampling
Population exposure			
average concentration	normal	two seasons	long-term
maximum concentration	worst case	any time	short-term
Complaints			
chronic effects	worst case	any time[1]	long-term
acute effects	worst case	any time[1]	short-term
Effects of remedial actions	normal	before and after	short/long term
Test for compliance	normal or worst case	any time	period of reference value

[1] According to activities, etc.

TABLE 26.4

Matrix relating health effect, exposure situation and sampling procedure to be applied (ECA, 1989b)

Potential health effect	Desired information on exposure	Type of sampling	Condition of sampling	Examples
Irritation	average exposure	long-term	real-life conditions	passive sampling: formaldehyde; nitrogen dioxide
	peak exposure	short-term	worst case	active sampling (continuous monitoring): formaldehyde; nitrogen dioxide
	exposure above fixed level	repeated short-term	real-life conditions	continuous monitoring: nitrogen dioxide
Toxic effect	exposure above fixed level	repeated short-term	real-life conditions	continuous (personal) monitoring: carbon monoxide
	average exposure	long-term	real-life conditions	active or passive sampling: pesticides
Carcinogenic effect	average exposure	long-term	real-life conditions	active or passive sampling: radon; benzene

26.4 TIME OF SAMPLING

The variation with time of the concentration level of a pollutant in an indoor environment is a well-known phenomenon. Parameters like the age of the building, the season, the time of the day, etc. (see Table 26.2) all influence the result of an indoor air measurement. Therefore, it has to be considered carefully when such measurements are carried out.

It is clear that different results will be obtained under otherwise identical conditions if one sample is taken in a room with doors and windows closed and the other following an extensive ventilation of the room. Furthermore, the occupants may contribute to the level of pollutants in one way or the other, e.g. through their various activities (an example is the increase in suspended

TABLE 26.5

Formaldehyde concentrations at different times after aeration (Deimel, 1978)

Room	Formaldehyde concentrations		
	Immediately after aeration	45 min after aeration	16–20 h after aeration
1	0.23	0.32	0.44
2	0.14	0.24	0.38
3	0.06	0.18	0.32
4	0.03	0.17	0.30

particulate matter concentrations in an occupied room). Over-night measurements in empty rooms may then be meaningless. Thus, the history of a room prior to and during sampling is of utmost importance and must be documented.

It is difficult to give a definite, but generally valid, recommendation for the time of sampling which could apply under all circumstances. However, the way of proceeding described in the following example is likely to be applicable also in a number of other cases.

In many cases, the question has to be answered whether a measurement has to address the worst-case situation (highest pollutant concentration or occupant exposure), or rather the normal or overall situation. Depending on the objective, the ventilation preceding the sampling, plays a more or less important role. After intensive ventilation, many substances emitted by flat surface materials require several hours to re-establish the steady state concentration at the normal ventilation rate. An example of this effect is the behaviour of formaldehyde levels (Deimel, 1978). Table 26.5 gives the formaldehyde concentrations in four classrooms at distinct intervals after ventilation. It should be noticed that such high concentrations which repeatedly pass over the guideline of 0.1 ppm, occur extremely rarely today.

The season of the year may have a strong influence on average concentration levels. The example in Figure 26.1 shows the development of VOC concentrations in the living rooms of 488 German homes during one year, as determined by Seifert et al. (1989). The concentrations were measured by passive sampling over two-week periods under normal living and ventilation conditions. The intensified ventilation occurring in summer months leads to reduced concentration levels, compared with winter months.

It should be emphasized that indoor pollutant concentrations and their variation in time may also be influenced by the level of outdoor air pollution.

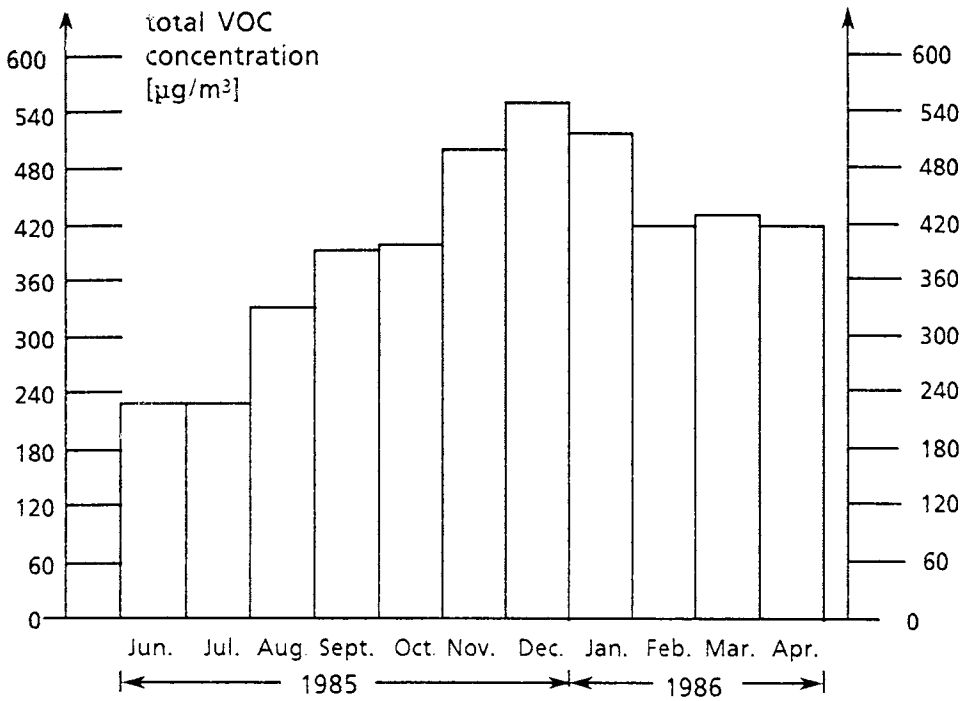

Fig. 26.1: Monthly mean values of the total VOC concentrations measured in 488 German homes. Reprinted from Seifert et al. (1989) with kind permission of Elsevier Science Ltd., Kidlington, Oxford, UK.

Hence, it is necessary to have information about the quality of outdoor air if it is likely that it contains significant amounts of the pollutants studied (e.g., NO_2 or hydrocarbons emitted from vehicles under perhaps unfavourable meteorological conditions). The polluted outdoor air reaches the indoor air with a certain time lag depending on the air exchange rate. Thus, care must be taken, if short-term indoor measurements are carried out in the course of an episode of elevated pollution levels in outdoor air, not to assign elevated indoor concentrations erroneously to indoor sources when infiltration processes are the main cause.

26.5 DURATION AND FREQUENCY OF SAMPLING

The term "duration and frequency of sampling" addresses the questions of how long and how often samples are to be taken. Duration of sampling is the time period over which the sampler collects a sample. The sampling frequency is defined as the number of samples taken over a given time interval (e.g., one year).

TABLE 26.6

Concentration intervals of different time averaged maximum NO_2 concentrations of 12 Dutch households (Lebret et al. 1987)

Place of sampling	Maximum concentrations ($\mu g/m^3$) over		
	1 min	1 h	24 h
Kitchen	400–3800	230–2050	53–480
Living room	195–1000	100–880	49–260
Bedroom	57–800	48–720	22–100

Although it is beyond the scope of this chapter to go into details of sampling techniques, it should be mentioned that short-term measurements are mostly carried out through active sampling, e.g. by drawing air through a collecting medium, while long-term averages (one day or more) are generally obtained through passive sampling, which does not require a pump. It should be noted however, that active sampling can also be used if long-term averages are required, as is the case for sampling suspended particulate matter.

As the analytical result gives the average concentration of the pollutant over the sampling period (time-weighted average concentration), extending the duration of sampling will result in an increasing levelling out of peak concentration and lead to a loss of information on fluctuations of pollutant levels.

As an example, Table 26.6 gives quantitative information about the way in which increasing sampling durations lead to an evenness in values detected. The table assembles data from a Dutch inquiry on nitrogen dioxide levels resulting from gas flame exhalations, in which the same real-time values are evaluated considering different sampling periods (Lebret et al., 1987).

The time required to take an air sample depends on:

- the lower detection limit of the analytical method;
- the potential health effect of the pollutant(s) in question (acute or chronic);
- the emission characteristics of the source(s) and other factors influencing the concentration levels;
- any specific objective of the measurement.

In practice, the lower bound of the duration of sampling is solely determined by the minimum mass of pollutant in a sample required to exceed the detection limit of the analytical method. The analytical laboratory should be consulted about the sampling requirements given the available analytical method.

Small, silent personal pumps or passive sampling devices are preferred for carrying out measurements in occupied spaces in order to minimize the annoyance to the occupants. Because of the low sample flow rates of these devices, long durations of sampling are often required to collect a sufficiently large amount of air. Particularly for diffusive samplers, durations of sampling of up to several days may be required.

The upper bound of the duration of sampling is highly dependent on the objective in question. If sampling objectives are the assessment of health risks, the sampling duration should preferably not exceed the averaging time of guideline values (see WHO, 1987). In general, for pollutants with chronic effects the average exposure is of interest and relatively long sampling durations (>24 h) are preferable. However, where acute effects (e.g. irritation) are concerned, peak exposures are important and sampling duration should be as short as feasible (<1 h); occasionally one would even prefer a direct-reading instrument, if available. For worst-case situations (maximum concentration likely to occur) the duration of sampling should be less or equal to the period during which a source is active and/or a factor influencing source emissions (e.g. temperature or humidity) or air concentration (e.g., reduced ventilation rate) is present. When compliance with a reference value or action level is to be checked, one should always sample over the full period for which the value is defined.

The frequency of sampling or the number of samples taken at different times necessary to characterize the air in a given space depends on the individual case. As occupant activities and ventilation characteristics may vary from day to day or display a seasonal pattern, no single sample taken at one particular time can give a reliable indication of the overall distribution of exposures. Only detailed observation and knowledge of the factors influencing the concentration level can give a clue to whether a single sample represents the high, medium or low side of the exposure distribution. However, the benefits gained from taking a larger number of samples over a certain period must be looked at in the light of the increasing costs of sampling and analysis involved. A single sample or two consecutive samples on one day may suffice if the factors influencing concentration levels are controlled or simulated to suit the purpose of the measurement. For instance, information on the maximum concentration likely to occur can be obtained by simulating a worst-case situation.

However, there are other sampling objectives that require more samples to be taken. One of these is the assessment of average exposures or exposure distributions. Since, generally, seasonal patterns are likely to exist, at least two sampling periods should be selected, one in summer and one in winter. In each period several independent samples may be needed. The total number of

samples required depends on the desired confidence limit of the results. Another example relates to measurements carried out to demonstrate non-compliance with a reference value or action level in order to justify remedial actions. In view of the sometimes high costs of such actions, the result of a single measurement may easily be challenged as not being representative. In this case, the taking of more samples covering different (simulated) conditions is recommended. In practice, three independent measurements will suffice to prove the probability of non-compliance.

26.6 SAMPLING LOCATION

As in most instances the ultimate goal of indoor measurements is an exposure assessment, the best location of a sampler is the person whose exposure has to be determined. However, this strategy can only be applied in a limited number of cases.

If measurements are carried out at fixed indoor locations, practical constraints generally limit the number of air analyses. Consequently, not every room in a flat or building can be controlled. The decision about which room(s) should be given priority is easy if the source — the emissions of which are to be measured — is present in only one room. The situation is more difficult if more than one room is concerned. In such cases, the decision where to sample has to be taken in the light of the sampling objective. As an example, one could mention the determination of nitrogen dioxide emitted from a gas stove. If the maximum concentration is required, sampling should be carried out in the kitchen. If one has to assess the general exposure of a young child or a sick person, the bedroom will probably be more appropriate.

Besides varying from one room to the next, concentrations of indoor pollutants may also vary within one and the same room, e.g. due to a poor ventilation efficiency or the presence of a strong source, e.g., a gas appliance. This leads to the question as to where in a room the appropriate sampling location is. Again, the answer to this question depends on the objective of the measurement. If no special objective is foreseen, as a general rule, the sampler or the probe should be located in the centre of the room at breathing height (e.g., 1–1.5 m above the floor). However, especially in the case of long-term measurements, a somewhat higher location (1.5–2 m) may be appropriate to avoid interference by or annoyance to the occupants. It would only be necessary to deviate from this rule if one has to assume a pronounced concentration gradient due to a strong source, Again, emissions from gas stoves may serve to illustrate this situation. It has been shown that nitrogen dioxide concentrations resulting from gas stove emissions are significantly higher above than below the stove (Seifert, 1984; Goldstein et al., 1988).

If a location other than the centre of the room is chosen for special reasons, possible differences in ventilation efficiencies should also be considered carefully. Furthermore, in the case of some passive samplers (those with large collection surfaces), accurate results can only be expected if the air surrounding the passive sampler exhibits a minimum of turbulence. Hence, locating these passive samplers in a corner or close to pieces of furniture should be avoided unless there are special reasons to do so, as described in the next paragraph.

The higher pollutant concentrations that are likely to occur close to an emission source can also be used to locate such a source. An appropriate procedure would be to divide the room into sub-spaces, each containing only one source. If measurements take place in steady-state conditions, there will be a concentration gradient between sub-spaces with and without the source. Using this procedure in connection with passive samplers, a shelf could be identified as the major source of toluene in a room (Abraham et al., 1981).

To be able to evaluate the measured concentration level of indoor pollutants, it may be useful to foresee an additional measurement in the outdoor air close to the building. The respective sampler should be located at a sheltered place at reasonable distance from the outer walls of the building or — in the case of an air-conditioned building — from the air intake (minimum: 2 m; maximum 5 m).

26.7 ACTIVE AND PASSIVE SAMPLING

During active sampling a definite air volume is pulled through filters or sorbent cartridges using sampling pumps. The air volume to be sampled depends on the sampler characteristics and on the analytical tool specifically adopted, since it has a direct influence on the Method Detection Limit (MDL). The duration of sampling is consequentially a function of the sampling flow rate. This latter can range from a maximum being low enough to prevent perturbing the room air concentrations and a minimum determined by the capacity of pumps to generate and maintain constant and measurable flow rates.

Active sampling is suitable for both short-time and, following appropriate reduction of the sampled air flow, for long-time measuring intervals.

In the case of active personal exposure monitors (PEM), the pump, the flow controller, and the battery pack must be placed together in a soft case worn on a belt or shoulder strap.

In passive sampling the air sampling rate is governed by the diffusion of air within the diffusion channel of the sampler, and by the air velocity at the

entrance area of the sampler, and is considerably smaller than sampling rates generated by pumps. Sampling rates of specific compounds are a function of their diffusion coefficients. Sampling times of one to several days are generally required to ensure that a sufficient amount of pollutant is available for analysis. Passive sampling devices are considerably smaller, less costly and involve no annoyance to room users.

A more exhaustive description of sampling techniques is given in the next chapter.

26.8 AIR EXCHANGE RATE MEASUREMENT

Air moves in and out of buildings at varying rates depending on a number of factors relating to the structural properties of the building and the local meteorological conditions. Two terms are used to describe how air enters a building: infiltration and ventilation. Both are measured as air exchange rate, or air changes per hour (ACH).

Air exchange between outdoors and indoors, in whatever form, dilutes contaminants generated indoors with outdoor air. However, when contaminant levels are higher outdoors than indoors, contaminant migration into the building will occur.

Tracer gas decay is a direct measurement of air exchange rate. An inert gas which is easily detected at very low concentrations is released and uniformly mixed within the building. Assuming the gas does not react chemically or physically with the surrounding materials, the gas concentration will decrease as air is exchanged between indoors and outdoors. The rate of decrease is proportional to the infiltration rate. Ideally, successive measurements over extended periods of time are necessary to determine the relationship between the rate of air infiltration and meteorological conditions for a specific building.

There are three key assumptions of the tracer gas technique: the tracer gas mixes perfectly and instantaneously; the effective volume of the enclosure is known; and the factors that influence air infiltration remain unchanged throughout the measurement period (Liddament and Thompson, 1983). Imperfect mixing occurs when air movement is impeded by flow resistances or when air is trapped by the effects of stratification. This causes a spatial variation in the concentration of the tracer gas within the structure. Sampling locations may be biased by this effect. Fans are often used to mix the tracer gas with the building air. Effective volume is assumed to be the physical volume of the occupied space. Areas which contain dead spaces that do not communicate with the rest of the living space will reduce the effective volume. Variation in conditions during the measurement period, such as door openings

or meteorological changes, will cause a departure from the exponential decay curve and the equation on which infiltration is calculated will no longer hold. Due to the influence of weather conditions it is preferable to have on site measurements of wind speed and temperature.

There are several different types of gases used as tracers: helium, nitrous oxide, carbon dioxide, carbon monoxide, sulphur hexafluoride (SF_6), and perfluorocarbons (PF). These tracer gases share certain desirable characteristics; they are non-toxic at concentrations normally used in such studies, non-allergenic, inert, and can be detected easily and at low concentrations. The gases which are used most frequently are SF_6 and perfluorocarbons. Carbon dioxide or carbon monoxide can be used if initial concentrations are substantially above background but well below concentrations of health concern.

While it is true that each of the tracers mentioned above are extremely unreactive, it is not certain that they do not adsorb on or desorb from indoor sources. The U.S. EPA is concluding chamber studies on SF_6 to determine if such effects occur and whether any such effects may tend to influence air exchange measurements using this tracer (Tucker, 1990). More research is clearly needed in this area.

Within the category of tracer gas methods for determining air exchange rates two methods can be distinguished. The first method, pulse release, involves the release of a known amount of gas, mixing it thoroughly within the space, and taking grab air samples at specified intervals to determine the decrease in concentration with time. Another approach is the constant release rate technique: the tracer gas is released at a constant known rate, usually via a permeation tube and a steady state concentration is established. Integrated samples are collected with absorption tubes, usually over one to several days. The air exchange rate is proportional to the amount of tracer gas which is collected, given the volume of the space and the release rate.

The grab sampling approach and the integrated sampling approach find the widest application in this field and are summarized below. As the grab sampling method most frequently uses SF_6 as a tracer and the integrated sampling method uses perfluorocarbon as a tracer, they are presented according to tracer gas. Other tracers could be used with either method; for example, CO_2 can be used as a tracer and a continuous CO_2 monitor used to measure decay. However, the basic principles of determining the air exchange rate are the same (Yocom and McCarthy, 1991).

Tracer gas diluition: sulphur hexafluoride (SF_6)

Specific instructions for this method can be found in ASTM (1983) — Standard Method for Determining Air Leakage Rate by Tracer Dilution. The basic apparatus includes: tracer gas, sample collection containers and pump,

syringes, circulating fans, and a stop-watch. Examples of sampling containers are Tedlar™ bags or gas-tight syringes. Meteorological parameters that are recorded include: wind speed and direction, temperature (indoor and outdoor), relative humidity and barometric pressure.

Two types of tracer gas monitors can be used with this technique, depending on the expected concentration of SF_6. Both instruments should be equipped with chart recorders. For SF_6 concentrations in the range 1–500 ppm, a portable infrared gas analyzer is used. For SF_6 concentrations in the ppb range, a gas chromatograph (GC) with an electron capture detector is used. A field GC is preferable so that the concentration of SF_6 can be immediately verified and optimum sample integrity maintained. However, if the laboratory is in reasonable proximity to the site, samples can be transported for analysis. Suitable quality control procedures should be implemented to verify sample integrity in such cases (Yocom and McCarthy, 1991).

Tracer gas dilution: perfluorocarbon (PF)

The perfluorocarbon (PF) technique, developed by Diez and Coté (1982), uses a permeation tube to release this tracer at a known rate for an extended period of time. The method is further described in Dietz et al. (1985). The sampling devices which are passive diffusion tubes collect integrated samples over several days. Tubes are analyzed at a laboratory by a gas chromatograph with an electron capture detector. There are two advantages to this technique: it is simple to deploy and it provides a measure of the average infiltration rate over an extended time period. The major disadvantages are that the analytical portion of the method is complex and sophisticated, the cost of PF is high, and few laboratories are equipped to handle these samples on a routine basis.

The required field equipment for this method is the permeation tube and the collection tubes. Three different perfluorocarbons have been used: perfluorodimethylcyclohexane, perfluoromethylcyclohexane, and perfluorodimethyl-cyclobutane. The source is a fluoroelastomer plug impregnated with a known mass of one of these compounds and sealed. The PF diffuses from the plug end at a constant rate and at a concentration which is determined analytically. Dietz and Coté (1982) report emission rates in the range of 3–20 nl/min. The number of sources deployed in a particular building depends on the number of main rooms and an estimation of the probable effective mixing.

Passive samplers are small tubes approximately 3 inches long and 0.25 inch in diameter. The adsorbent material, 50 mg of Ambersorb™, is held in place with glass wool or a stainless steel screen. The number of passive sampler deployed depends on the size and number of rooms in the building. The duration of sampling depends on several factors including: detection limit of PF on the collection tubes, source emission rate, and field program considera-

tions. Sampling times of four to seven days and up to several weeks have been used successfully with one collection tube. Longer integration times are possible by using sequential collection devices (Yocom and McCarthy, 1991).

26.9 QUALITY ASSURANCE

Due to financial and other constraints, there is a tendency to evaluate the quality of the air in a room based on a minimum of analytical results. This tendency can be understood if one considers that often parameters not linked to the sampling and analytical procedure, e.g. the ventilation rate, will have a greater impact on the result than analytical parameters. Therefore, analytical quality assurance often plays a minor role in practical indoor air analysis. However, depending on available funds, it is strongly recommended that full quality assurance be guaranteed. The parameters that should be included in a quality assurance programme of indoor air sampling are summarized in Table 26.7. Since they are comparable to those used in the quality assurance programmes for outdoor and workplace air investigations, reference is made to the practice established in these fields. (IUPAC, 1982).

One of the parameters in Table 26.7 deserves a special mention since it is related exclusively to the indoor environment. The air velocity observed in some rooms may be so small that a depletion of monitored vapours may result in the close vicinity of a passive sampler. Such depletion will result in an

TABLE 26.7

Parameters to be considered in the quality assurance of indoor air sampling using various sampling procedures (ECA, 1989b)

Parameter	Sampling procedure applied		
	Grab sampling	Active sampling (pump)	Passive sampling (diffusion)
Blank value of sampling equipment	x	x	x
Break through volume*		x	
Uptake rate			x
Desorption efficiency	x	x	x
Storage stability	x	x	x
Sample flow/volume	x	x	
Air velocity near sampler			x

* Of particular importance for high volatile compounds.

(apparent) increase in the diffusion path length of the sampler, thus reducing the diffusion rate.

The required minimum air velocity depends to a large extent on the diameter of the sampler's opening. For a passive sampler with a small opening of only 7 mm^2 (diameter: 3 mm) and diffusion rates between 4 and 8 cm^3/h, quantitative sampling was achieved down to the lowest measurable air velocity of <0.1 cm/sec (De Bortoli et al., 1989). With commercially available samplers minimum air velocities required for quantitative sampling vary between 0.7 and 7 cm/sec (Matthews et al., 1989).

Indoor Air Quality
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Chapter 27

Methodology of Building Investigation III: Sampling and Analysis of Indoor Air

27.1 INORGANIC POLLUTANTS

27.1.1 Carbon dioxide

Carbon dioxide concentrations, usually in the range of percent, can be detected with the methods used by industrial hygienists. Air is pumped in a canister, subsequently analyzed by gas chromatography (GC) and determined with a thermal conductivity detector or other techniques. Colorimetric direct reading detector tubes, non-dispersive infrared spectrophotometry (NDIR) and photoacoustic-IR detectors are excellent methods for its accurate determination; these instruments are useful for continuous monitoring and recording of patterns of CO_2 in indoor air (Yocom and McCarthy, 1991).

27.1.2 Carbon monoxide

Usually the CO detection underlies the employment of a non-dispersive infrared analyzer (NDIR). The infrared radiation passes through parallel optical cells, one with air sample, the second with CO-free air as reference: the difference in absorbance relates to CO concentration. The measured absorption is limited to one or more of the characteristic wavelengths of the infrared spectrum of CO.

Other instruments named gas filter correlation (GFC) techniques, make use of optical filters to limit the photometers' sensitivity to the IR band of interest. In GFC instruments IR radiation passes through a spinning filter wheel containing a sealed CO reference cell and a nitrogen reference cell. The IR beam then passes through a chamber containing the air sample and CO is detected.

Recently a more specific and sensitive detector in the IR field has been developed: the photoacoustic-IR detector. It is based on the fact that if a gas is irradiated with light of a wavelength coinciding with an absorption maxima of

the gas, the temperature of the gas increases due to absorption. If the gas is in a chamber of constant volume the pressure will increase; if the light is modulated by a chopper there will be a pressure variation, and the sound wave can be detected by a condenser microphone. By using light of a selected wavelength, a given gas can be selectively detected. The light source normally is in the IR band with an optical filter to restrict the wavelength. The response time is in the range of 30–100 sec for one to five gases and the technique presents good specificity and sensitivity for a wide number of substances.

Another CO detector is based on electrochemical oxidation: sample air passes through an electrochemical cell where the oxidation of CO to CO_2 produces a signal proportional to CO concentration.

There are many instruments commercially available, based on the reported principles: they are reliable and sensitive, can provide a continuous record of the CO concentrations, and many of them could be hand-held for personal monitoring.

The miniaturized CO monitoring devices usually operate on the principle of the electrochemical or catalytic oxidation of CO (Yocom and McCarthy, 1991).

27.1.3 Nitrogen dioxide

Nitrogen dioxide determinations have been carried out with the aid of passive samplers, as reported by Palmes at al. (1976). The sampler consists of a tube of 7 cm length and 10 mm internal diameter, containing on its bottom a wire-gauze impregnated with triethanolamine, acting as adsorbent. After exposure, the nitrite formed is eluted with water and determined photometrically, e.g., following the Saltzman procedure.

Sampling periods of one or even two weeks have been reported by many authors making use of the Palmes sampler, whereas others, more interested in better time resolution, sample for only 48 h (Nagda et al., 1987).

In newly available passive samplers made by filters treated with triethanolamine or liquid solution bubblers, the wind effects are suppressed by hydrophobic fibre filters.

Commercially available instruments for direct reading determination are based on: wet chemistry absorption and colorimetric analysis; chemiluminescent reaction with O_3; electrochemical sensor; diffusion and electrochemistry (NO_2 diffuses into an electrochemical cell) (Yocom and McCarthy, 1991).

27.1.4 Sulphur dioxide

The U.S. EPA reference method for SO_2 measurement in outdoor ambient air is the pararosaniline method: the air sample passes through a bubbler to

form a stable complex which is colorimetrically detected after reaction with pararosaniline.

Several direct-reading instruments for SO_2 monitoring are available, using pararosaniline- and electrochemical reaction. Flame photometric detection (FPD) is based on sulphur specific emissions (this is not specific for SO_2, all sulphur containing compounds interfere); using the electrochemical method, SO_2 is adsorbed onto an electrocatalytic electrode where an electric current proportional to the SO_2 concentration is generated (Yocom and McCarthy, 1991).

27.1.5 Ozone

The U.S. EPA reference method for O_3 is based on the gas-phase chemiluminescence deriving from the reaction between ethylene and O_3. There are two other methods: one is based on the UV absorption of O_3 (compared with a reference) and the other on the photometric detection of the chemiluminescence resulting from the reaction between O_3 and rhodamine-B.

A direct reading portable photometric detector is available, the signal deriving from the chemiluminescence of the reaction between O_3 and ethylene gas (Yocom and McCarthy, 1991).

27.2 ORGANIC POLLUTANTS

27.2.1 Introduction

Organic indoor pollution has several peculiarities if compared with pollution involving other classes of chemical or physical agents. Some typical characteristics of organic indoor pollution are :

- presence of complex mixtures of compounds;
- large variety and variability of sources; and
- large variety of chemical species.

Due to the widespread physico-chemical properties of organic indoor pollutants, a WHO Working Group has categorized the entire range of compounds into four groups according to their boiling points and to the different methods used to collect them from air, as indicated in Table 27.1. Further significant distinctions within each group exist according to the polarity, the hydrophilic or hydrophobic (respectively lipophilic) character, the reactivity, and the resistance to thermal degradation. A variety of sampling and analytical methods have therefore to be available for covering the whole spectrum of organic compounds. Furthermore, the choice of the detection and in particular of the

TABLE 27.1

Classification of indoor organic pollutants (WHO, 1989)

Category	Description	Abbreviation	Boiling-point range (°C)[a]	Sampling methods typically used in field studies
1	Very volatile organic compounds (incl. gases)	VVOC	<0 to 50–100	Batch sampling adsorption on charcoal
2	Volatile organic compounds	VOC	50–100 to 240–260	Adsorption on Tenax, carbon molecular black or charcoal
3	Semivolatile organic compounds	SVOC	240–260 to 380–400	Adsorption on polyurethane foam or XAD-2[b]
4	Organic compounds associated with particulate matter or particulate organic matter	POM	>380	Collection on filters

[a] Polar compounds appear at the higher end of the range.
[b] Styrene–divinylbenzene co-polymer

sampling method is influenced by the objective of pollution measurements because it determines whether:

- the presence and concentration of pre-selected "target" compounds has to be determined or a survey is requested of which pollutants are present at which concentrations,
- short-term peak values or long-term averages of the pollutant concentrations have to be detected or
- a local pollutant concentration (or an environmental sample), the concentration in the breathing zone of a person (or a personal sample) or the emissions from an indoor source have to be measured.

Most of the analytical methods currently available have been developed for analysing outdoor or workplace air pollutants and, where necessary, have been adapted for the measurement of indoor pollutant concentrations or exposures. They are usually designed to cope with the following conditions:

- Typical indoor (and outdoor) concentrations of most air pollutants (organic compounds in particular) are too low for direct analysis with most of the detection methods currently available. Therefore, during the sampling procedure the pollutants are usually preconcentrated. Preconcentration is particularly important for identifying unknown compounds, i.e. for survey analysis.
- The indoor environment consists of a large number of separate spaces

with different pollution patterns and levels. Therefore, many measurements need to be taken for a representative characterization of indoor pollution. The necessity of taking many measurements calls for simple, lightweight and inexpensive equipment.

- Indoor spaces are usually occupied by people. If the occupants of these spaces are to be disturbed as little as possible, the analytical instrumentation should be small and silent.

The answer to these characteristics of indoor environments are analytical methods which are composed of two steps:

(1) Sampling. This step is performed by lightweight, small, silent, and inexpensive equipment. During sampling the pollutants are usually separated from the bulk of the air thereby achieving a preconcentration.

(2) Separation and identification. This step is typically performed in the laboratory and therefore also complex and expensive instrumentation may be used. It includes separating the often complex mixtures of indoor pollutants by high resolution gas chromatography (HRGC) or high performance liquid chromatography (HPLC), and their identification by chromatographic retention times, by mass spectrometry (MS) or by a combination of both methods.

Whereas the sampling procedures have to be adapted to the specific requirements of the indoor environment, the methods for separating and identifying organic indoor air pollutants are also used in other fields of organic environmental analysis such as outdoor air and water analysis. Therefore, a brief introduction on sampling methods will precede the more detailed description concerning the determination of the most important pollutant classes.

An important part of the analysis of organic indoor pollutants is the procedure for transferring the pollutants from the sampling equipment to the separation and identification instruments. This procedure has a strong influence on the volume of air which has to be sampled and on the overall analytical method as will be discussed below (Knöppel, 1992).

27.2.2 Sampling methods

Three different principles have been applied to the sampling of volatile organic pollutants from ambient or indoor air (see Figure 27.1).

(1) The Grab method takes a whole-air sample by either opening the valve of an evacuated cylinder or pumping air into it (using, e.g., a membrane pump).

(2) During active sampling an appropriate amount of air is drawn by a pump through a solid or liquid sorbent where the pollutants are trapped. For indoor air sampling usually small and silent so-called personal pumps are used. There are two critical parameters for active sampling: the breakthrough volume and the sample flow rate. The breakthrough volume is defined as the

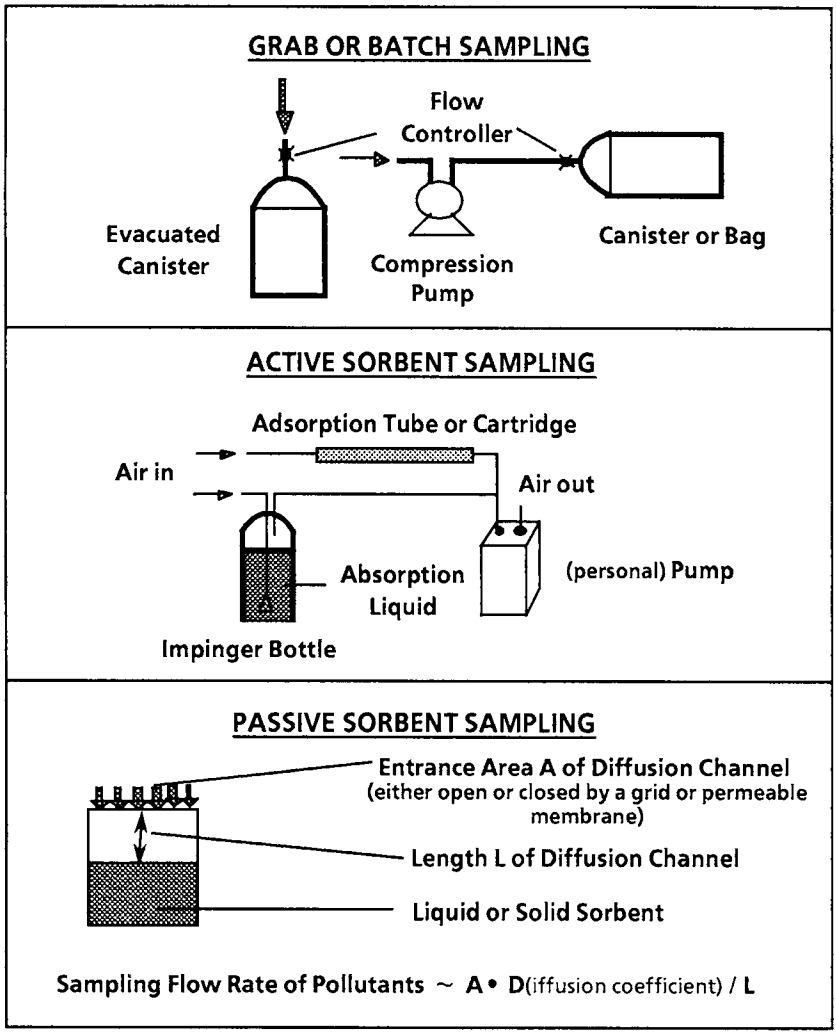

Fig. 27.1: Illustration of sampling principles for organic indoor pollutants (Knöppel, 1992).

volume of air which can be passed through a trapping medium (can be a liquid or a solid) before an analyte of interest is detected in a back-up sampler. For solid sorbents the breakthrough volume depends on the amount of sorbent in a sampling tube, on the concentration of pollutants in the air sample, and on the physico-chemical characteristics of the sorbent (in particular its specific surface area; see Table 27.4) and of the pollutants (in particular on their volatility and hydro- or lipophilicity). For unpolar sorbents, which are used most, the more volatile and hydrophilic a pollutant is, the smaller the break-

through volume.

The sample flow rate must be low enough to provide sufficient contact time between the air and the sorbent for diffusion of the pollutants to the sorbent surface. At sample flow rates which are too high the breakthrough volume is reduced.

(3) Passive sampling is based on the diffusion of pollutants from the entrance opening (surface area A) of a sampling cylinder (where the pollutant concentration is assumed to be that of the surrounding or bulk air) to the surface of a sorbent (usually solid, but also liquid) which has a well defined distance from the entrance and where the pollutant concentration is assumed to be negligible compared to the bulk air concentration. Ideally the sampling flow rate follows Fick's first law of diffusion and is determined by the geometry of the sampler and the diffusion coefficient of the pollutants according to the equation:

$$F = 3600 \cdot A \cdot D/L$$

with F [cm^3/h] = sampling flow rate of a pollutant; A [cm^2] = entrance area of diffusion channel (either open or closed by a grid or permeable membrane); D [cm^2/s] = diffusion coefficient of the pollutant (see Table 27.2); L [cm] = length of diffusion channel.

It is important that no air turbulence disturbs the diffusion process in the space between the sampler opening and the sorbent surface since this is a prerequisite for the determination of bulk air concentrations. Therefore either the diameter of the entrance area A must be small in comparison to the diffusion length L or the entrance opening must be closed by a permeable diaphragm.

Passive sampling devices do not require a pump and flow regulation system and are therefore considerably smaller, less costly and less obtrusive than active sampling devices. They are therefore ideally suited for personal sampling, but are also useful for environmental sampling.

Table 27.3 summarizes some basic features of the three sampling principles.

Grab sampling allows high sampling velocities and short sampling times. It is therefore particularly suitable for the determination of short term peak values of concentrations but can also be adapted to the measurement of longer term average concentrations.

For *active sampling* the sample flow rate is limited since the organic molecules must be in contact with the sorbing medium sufficiently long to allow for diffusion from the air to the sorbent. Moreover the sorbent bed acts as a flow resistance.

TABLE 27.2

Observed diffusion coefficients of some organic indoor pollutants (25°C and 760 mmHg) (values taken from Lugg, 1968)

Pollutant	10^2·Diffusion coefficient (cm^2/sec)	Pollutant	10^2·Diffusion coefficient (cm^2/sec)
Aliphatics		**Ketones**	
Pentane	8.42	Acetone	10.49
Hexane	7.32	Methylethylketone	9.03
Octane	6.16		
		Chlorinated hydrocarbons	
Aromatics		Dichloromethane	10.37
Benzene	9.32	Chloroform	8.88
Toluene	8.49	Trichloroethene	8.75
Ethylbenzene	7.55	Tetrachloroethene	7.97
o-Xylene	7.27	1,1,1-trichloroethane	7.94
m-Xylene	6.88	Chlorobenzene	7.47
p-Xylene	6.70		
1,3,5-trimethylbenzene	6.63		
n-Propylbenzene	6.69		
Styrene	7.01		

The useful sampling rate of *passive samplers* is limited by the air velocity at the entrance area of the sampler. At low air velocities the pollutant concentration in the vicinity of the entrance of the diffusion channel decreases compared with the bulk air concentration since there is no complete replacement of the molecules diffused into the sampler. This leads to an apparent increase of the diffusion channel length and to a reduction of the sampling rate compared to the theoretically expected one. The effect of the air velocity on the sampling rate can be reduced using samplers with small entrance areas (and, hence, low sampling rates). Such samplers are most useful for the determination of longer term (1–2 weeks) average concentrations (see Section 27.2.4).

Grab or whole-air sampling can be used for the determination of environmental concentrations, whereas active sampling with lightweight silent personal pumps and, even more easily, passive sampling can also be used for personal monitoring (Knöppel, 1992).

TABLE 27.3

Characteristics of different sampling approaches for organic indoor air pollutants (Knöppel, 1992)

Type	Sampling principle	Typical sample volume [l]	Typical sampling velocity
Grab, batch or whole sampling	vacuum	1–6	≤30 l/min
	compression pump	1–20	(smaller velocities with flow restrictor)
	cryo-condensation	1–50	
Active sampling (solid or liquid sorbent)	(personal) aspiration pump	0.5–2 (thermal elution)	4 ml/min – 4 l/min (smaller velocities with intermittent sampling)
		50–600 (solvent elution)	
Passive sampling (solid or liquid sorbent)	molecular diffusion	0.5–2 (thermal elution)	0.5 ml/min – 100 ml/min* (depending on geometry)
		50–600 (solvent elution)	

* The higher the sampling velocity the stronger its dependence on air movement.

27.2.3 Sample transfer to the analytical equipment

There are essentially four ways used to transfer samples to the separation and identification equipment:

1. Direct injection of part of a grab sample or an aliquot of an absorption liquid into a HRGC or HPLC system;
2. Solvent extraction of the trapped pollutants from an absorption liquid or a solid sorbent and injection of an aliquot of the extract into the HRGC or HPLC systems;
3. Thermal elution of adsorbed compounds from a solid sorbent by means of a pure carrier gas, usually helium; in this case the desorbed compounds are cryogenically reconcentrated in a capillary which is directly connected with the analytical HRGC column. Cryo-concentration may also be used to introduce larger amounts of a grab sample into the HRGC column. The cryotrap is flash heated in order to guarantee a rapid injection of the compounds into the gas chromatograph.
4. For SVOC and POM the use of Supercritical Fluid Extraction (SFE) increasingly substitutes the conventional extraction techniques. SFE allows

one to highly concentrate extracted compounds since the removal of the extraction solvent (usually CO_2) proceeds spontaneously when taking the system back to normal pressure and temperature. The sample transfer is realized by re-solubilizing the extracted compounds in a small amount of conventional solvent and injecting an aliquot of the resulting solution into a HRGC or HPLC system (off-line transfer), or even by cryo-focusing them within a capillary and injecting (flash-heating) them directly into a HRGC system (on-line transfer). The use of SFE for the analysis of semivolatile and particle bound organic indoor pollutants is still under investigation.

The sample transfer method has an important influence on the overall sensitivity of the analytical method. Sorption on a solid sorbent and thermal elution is the most sensitive method. Using this method all compounds collected from an air sample are available for one analysis, the analysis of the sample being, however, unrepeatable. In principle the same is true if a whole air sample is cryo-concentrated prior to HRGC separation. However, the water contained in the air sample may block the cryotrap.

Solvent extraction leads to a dilution of the sampled compounds in the solvent, of which usually only a small fraction (typically 0.1–1%) can be injected in the HRGC or HPLC system. For VOC, evaporation of the solvent would lead to losses of the sampled compounds.

A particular case results, if an adsorption liquid contains a reagent which reacts specifically with a single compound or compound class. In indoor air analysis this technique is mostly applied to the detection of carbonylic compounds (aldehydes and ketones) using dinitrophenylhydrazine (DNPH) as a reactant. In this case non volatile reaction products (dinitrophenylhydrazones) are formed and the absorption liquid (e.g. acetonitrile) or the extraction solvent may be vaporized without sample loss leading to a considerable gain in detection sensitivity (Knöppel, 1992).

27.2.4 Volatile organic compounds

Active sorbent sampling

The most widely used procedure for sampling volatile organic compounds from ambient or indoor air is to pass a suitably large air volume through a solid sorbent material that retains the compounds of interest (Namiesnik, 1988). Silent, relatively small and light weight battery driven pumps are available which maintain an adjustable constant sample flow. The sample flow has to be calibrated.

There are many types of solid sorbents, most of which fall into one of three categories: organic polymer resins (e.g. Tenax TA, XAD), carbon based sorbents (e.g. Carbotrap, Carboxen, Carbosieve, activated charcoal) and inorganic

sorbents (e.g. silica gel, alumina, Florisil, molecular sieves). Of these, inorganic sorbents are normally not used for VOC sampling because they are thermally not very stable, are hydrophilic and have therefore a high affinity for water which makes thermal elution impossible. Carbon based sorbents are hydrophobic and have a high thermal stability. However, only graphitized carbon black (like Carbotrap) has a sufficiently low sorbent–air distribution coefficient (or adsorption strength) to allow thermal elution of the full range of VOC at temperatures which do not induce thermal degradation of some compounds. For VVOC Carboxen and Carbosieve can also be used. Tenax TA has a good thermal stability, a low affinity for water and allows thermal elution of a wide range of VOC (i.e., from C_6- up to C_{15}- alkanes) at a temperature of 250°C. Sorption on Tenax TA has therefore been used most widely for sampling of VOC.

Table 27.4 summarizes the physical characteristics of a few of the most commonly used sorbents for active sampling of VOC from indoor air. For equal weights of sorbent (typically 0.1 to 1 g), the smaller the specific surface area of the sorbent, the smaller the breakthrough volume (see Section 27.2.2) of a volatile organic compound. Similarly, the higher the specific surface area of a sorbent is, the more volatile compounds are retained on the cartridge at a given air sample volume. On the other hand, the higher the specific surface area, the higher the required elution temperature. Due to these limitations none of the sorbents allows to trap and thermally elute the whole range of VOC and VVOC

TABLE 27.4

Characteristics of solid sorbents more frequently used for VOC sampling (Knöppel, 1992).

Sorbent	Sorbent type	Approximate specific surface area (m^2/g)	Maximum elution temp. (°C)	Useful range for *n*-alkanes
Carbotrap C	graphitized carbon black	12	400	C_7–>C_{15}
Tenax TA	organic porous polymer resins (poly-*m*-terphenyl ether)	35	250–300	C_6–>C_{15}
Carbotrap™	graphitized carbon black	100	400	C_5–>C_{15}
Carbosieve™ S-III	spherical carbon molecular sieve	550	>400	≥C_2 (mostly VVOC)
Charcoal (mostly for passive samplers)	charcoal	>1000	–	(≥C_2)

as indicated in the table. From this point of view a combination of sorbents gives the best promise for efficient sampling and thermal desorption of a wide range of volatile organic compounds provided the sample air passes through sorbents with increasing specific surface area. It should be noted, however, that extending the volatility range of a sample to highly volatile compounds is only useful if the HRGC separation is appropriate, i.e. if the temperature program can start at sufficiently low (sub-ambient) temperatures.

Passive sorbent sampling

Commercial passive sorbent samplers for volatile organic compounds use active charcoal as sorbent. Due to the strong sorption capacity of charcoal the gas phase concentration C_s of sorbed compounds at the surface of the sorbent is negligible, even for most VVOC. However, charcoal requires solvent elution of the sampled compounds with its inherent lower sensitivity compared to thermal elution. An interlaboratory comparison test of a commercially available sampler (De Bortoli et al., 1986) has shown that about 500 l of air had to be sampled in order to detect VOC concentrations at the 1–2 $\mu g/m^3$ level, a value that corresponds to the 50 percentiles of the concentrations of many indoor pollutants (WHO, 1989). At a sample flow of 25 ml/min (typical for this sampler) a sampling time of about 14 days is required.

In order to increase the sensitivity of passive samplers, Lewis et al. (1985) have developed a passive sampling device which uses Tenax GC as sorbent and can be thermally eluted. In addition the length of the diffusion channel was reduced yielding sample flows up to 80 ml/min (chloroform), thereby increasing the analytical sensitivity 400–500 fold compared to the above-mentioned charcoal sampler. Using this device, 1 ng of an indoor pollutant at an air concentration of 1 ng/l can be made available for analysis in about a quarter of an hour compared to 14 days using the charcoal based device. A specially designed oven has to be used for thermal desorption of the Tenax passive sampler.

For longer term sampling and more volatile compounds, however, the vapour phase concentration C_s at the Tenax surface is no longer negligible and the sample flow decreases with time.

Apart from this drawback, both described designs need fairly high surface air velocities in order to reach their theoretical (maximum) sample flows. For the sampler of Lewis et al. (1985) sampling rates between 67 and 80 ml/min were reduced to 64% of these values when the air velocities at the entrance area of the sampler was reduced from 50 cm/sec to 5 cm/sec and at lower surface air velocities a further steep decrease of the sample flow has been predicted based on theoretical considerations. On the other hand, the overall frequency distribution of air velocities measured in six occupied houses maximized at 0–2.5 cm/sec (Matthews et al., 1989).

In order to avoid the dependence of the sampling rate from the surface air velocity, De Bortoli et al. (1989) used a glass tube with an internal diameter of 3 mm and a diffusion channel length of 25 mm, filled with a 2.5-cm plug of Tenax TA or Carbotrap as passive sampling device for thermal elution. In view of the geometry of the sampler only small sampling rates are achieved in the order of 0.6 ml/min or 1 l/week so that in one week 1 ng of a compound is sampled if its air concentration is 1 ng/l. This is about twice the sensitivity which can be achieved with the above mentioned commercial charcoal sampler. As a consequence of the non negligible C_s values, however, this sampler has a lower than theoretical sampling rate for more volatile VOC (about 50% for *n*-hexane, 100% for xylene) and may lose these compounds upon exposure to zero air (40% loss of *n*-hexane, no loss of xylene after one week).

Sampling of polar compounds

Tenax and Carbotrap are usually assumed to perform well for sampling unpolar compounds but to be less or not appropriate for sampling polar compounds. However, very little work has been reported on the sampling of polar compounds with Tenax and Carbotrap. From the work reported by Rothweiler et al. (1991) it appears that at comparable boiling points, polar hydrophilic compounds such as acetonitrile, diethylamine, acetic acid, tetrahydrofuran, 1,1-ethanediol, and mercaptoethanol have considerably smaller retention volumes than unpolar compounds. To a smaller degree such an effect can also be observed for polar lipophilic compounds such as chlorobenzene (Health and Safety Executive, 1991).

Irregular responses of polar hydrophilic compounds such as methoxy- and ethoxyethanol observed in the laboratory when analyzing Tenax tubes could be attributed to the interference of water with the HRGC separation on an apolar column (OV-1). Perfect results were obtained when a Carbowax column was used instead.

The limited available experience shows that more work is needed in order to evaluate the usefulness of sorbent sampling and thermal elution for the analysis of polar and in particular of hydrophilic compounds (Knöppel, 1992).

27.2.5 Formaldehyde

There are many different methods available for measuring formaldehyde (HCHO) concentrations including personal and stationary methods and active and passive methods. Evaluations of some of these methods are given in Wallace and Ott (1982), Kennedy et al. (1985a,b), Godish (1985) and Sexton et al. (1986).

Regardless of which method is used, it is important to point out some

general characteristics of HCHO emission. The most important sources of HCHO in the indoor environment are products which contain urea-formaldehyde resins. These resins are mainly used in wood products (particle-board and plywood), urea-formaldehyde foam insulation (UFFI) and in urea-formaldehyde lacquers. Indoor concentrations of HCHO deriving from these products depend on changes in ventilation rate, the operation of air cleaners, indoor and outdoor temperatures, and humidity. The rate of release of HCHO from UFFI and other urea-HCHO products increases with the temperature, wood moisture content, humidity of the surrounding air, and with decreased HCHO concentrations in the surrounding air (Fisk et al., 1987). Therefore HCHO also demonstrates diurnal and seasonal variations. HCHO concentrations may vary by as much as 50% throughout the day and are higher during the summer than during the winter (Gammage and Gupta, 1989).

DNPH HPLC method

This method is most often used in indoor investigations because of its sensitivity, its specificity and the possibility to analyse simultaneously other aldehydes and ketones. The method utilizes solid adsorbent sampling followed by HPLC analysis (U.S. EPA, 1989b). HCHO gas (and other carbonyl compounds contained in the gas-phase) is adsorbed onto acidified 2,4-dinitrophenylhydrazine (DNPH)-coated silica gel cartridges and derivatized to their stable hydrazones. The hydrazones are washed off with acetonitrile, separated by HPLC and detected with a UV detector operating at 365 nm.

Blank values are normally lower than 50 ng/sampling tube. The sampling speed of 0.5–1.5 l/min and sampling periods of up to 24 h allow to determine concentrations from 0.1 ppb to several ppm. When two sampling tubes are used in series, 95±3% (n = 50) of the formed hydrazones are found on the first cartridge.

Samples should be refrigerated after collection, and they can be stored for not more than 90 days. The precision of field replicates should be ±20% or better, and replicate HPLC injections should have a precision of ±10% or better.

The sampling procedure requires a moderate degree of skill and training; the analysis requires a highly skilled person who is proficient in HPLC techniques.

For the passive DNPH HPLC Method, the sampler consists of DNPH-impregnated glass fibre filters which are placed behind diffusion screens and sandwiched between two protective caps (U.S. EPA, 1989b). The size of the completed sampler is 3.8 cm in diameter and 1.3 cm in depth. HCHO and other aldehydes diffuse to the sampler to form the stable DNPH derivative. Precision of this method is comparable to that of active sampling. Advantages of this

method include the smaller size of the sampler, no noise, low unit cost, and the ability of unskilled personnel to place and retrieve the samplers. (U.S. EPA, 1991b).

MBTH bubbler method

The MBTH passive bubbler method has become a standard test method for HCHO by the American Society of Testing and Materials (ASTM, 1990). This method utilizes the Passive Bubbler™ which consists of a glass vial with a septum cap that retains a Knudsen disk. The vial, which contains an aqueous solution of 3-methyl-2-benzothiazolinone hydrazone hydrochloride (MBTH), is inverted for sampling. HCHO diffuses through the Knudsen disk at a constant rate. After collection, a solution of ferric chloride-sulfamic acid is added to form a derivative which is measured in a spectrophotometer at 628 nm. This method allows HCHO to be measured in the range of 25 ppb to 14 ppm for sampling times between 15 min and 8 h. A 4-h sampling time is recommended to measure HCHO concentrations in the range of 50 ppb to 1 ppm.

A major advantage of this method is that laboratory support is not needed. Kits can be purchased that contain a mini-spectrophotometer and all needed reagents (U.S. EPA, 1991b).

Automated colorimetric method

This method uses a commercially available continuous colorimetric gas analyzer and is based on the HCHO selective pararosaniline method in which HCHO is absorbed into sodium tetrachloromercurate (II) solution containing a fixed amount of sulphur dioxide. Acid bleached pararosaniline is added to form a purple dye. The concentration of HCHO is proportional to the intensity of the dye.

The analyzer is portable, provides real time measurements, and has a practical detection limit of about 20 ppb. Disadvantages include cost, and the need for personnel with the expertise to operate the instrument and conduct the required calibrations.

A modified method (Lawrence Berkeley Laboratory pararosaniline method; Miksch et al., 1981) utilizes midget impingers for sample collection and spectrophotometry for analysis. HCHO reacts with pararosaniline in the presence of sodium sulphite to produce a coloured product which is read at 570 nm. The method has a sensitivity of 10 ppb and is not subject to interference from nitrate, nitrite, phenol, ethanol, or higher molecular weight alcohols. Disadvantages are that refrigeration is required during collection and storage (U.S. EPA, 1991b).

Chromotropic acid method

The chromotropic acid procedure (NIOSH, 1977a) utilizes midget impingers to collect HCHO into an absorbing solution. A modified version increases the collection efficiency to 98% eliminating the need for a second impinger for sample collection (Meadows and Rusch, 1983).

HCHO reacts with chromotropic acid–sulphuric acid solution to form a purple-coloured complex, the concentration of which is determined by a spectrophotometer at 580 nm. Samples are typically collected at a flow rate of 1 lpm for 0.5–1 h. The method has a sensitivity of 160 ppb for 15-min samples and 40 ppb for 1-h or longer samples.

This method has been widely used in both residential and non-residential sampling. Advantages are that it is a relatively simple technique which utilizes equipment that is readily available. A moderate degree of training is needed for the sampling and analysis. Disadvantages are that confidence decreases significantly below 0.10 ppm and that concentrated sulphuric acid is used in the analysis. It should not be used in situations where phenol may be present. (U.S. EPA, 1991b).

27.2.6 Pesticides and polychlorinated biphenyls

Pesticide residues in the indoor environment are found as vapours in the air, they may be adsorbed or deposited on indoor surfaces and dust, be contained in treated indoor materials such as wood (including furniture) or leather, and they may accumulate in textiles. Exposure to pesticides may therefore occur through inhalation, skin absorption after contact with contaminated surfaces or house dust and be ingested as a consequence of hand/mouth activities.

There is clear evidence that dust is a particularly useful indicator for the presence of pesticides in indoor environments. Already one of the earliest publications on indoor measurements of pesticides concludes that house dust is a major reservoir for pesticides (Starr et al., 1974). In a study of 217 households in Germany it has been shown that the pentachlorophenol content of house dust correlated well with human exposure as measured by the urine content and in a study in Florida, 13 pesticides were detected in house dust that were not detected in the air.

As a consequence, the identification of potential hazards due to pesticide exposure in the indoor environment or the evaluation of mitigation measures require both, analysis of pesticides in the air and in house dust and surface samples. In addition analysis of water, wood, furniture, leather and textile samples may be required, if sources of pesticides have to be identified. In the

following, sampling and analytical procedures for pesticides in indoor air and in dust are briefly described.

Pesticides in indoor air are collected by using a personal sampling pump for drawing air through a polyurethane foam (PUF) cylinder that is slightly compressed into a borosilicate glass tube.

Commercially available or user made sampling tubes can be used. Air flow rates in the range 1–5 lpm and sampling periods of 4–24 h are typically used. PUF is a preferred sorbent because of its low flow resistance but other sorbents are also used. The sampling pumps have to be calibrated in the laboratory before and after sampling.

Environmental or personal air samples can be collected. For the collection of environmental samples the samplers should be placed at breathing height of the occupants (about 0.9–1.8 m above the floor). Higher concentrations may, however, be found near to the floor where small children may be exposed. Samplers should have a distance of at least 30 cm from any obstacle to insure adequate air flow. The collected samples should be refrigerated until analysis.

After sample collection the PUF plugs are extracted, the extract is concentrated and analyzed using gas chromatography with electron capture detection (GC/ECD). Gas chromatography can also be combined with mass spectrometry/multiple ion detection (GC/MS/MID) for confirmation of the identity of pesticides and to quantify non-chlorinated compounds. Table 27.5 reports pesticides which have been detected in indoor air by the above described methods.

Sampling and analysis methods for floor and surface dust have been reviewed by Roberts et al. (1992). A variety of techniques have been used for estimating exposure to pesticides in dust including: (1) household vacuums, (2) hand-held vacuums, (3) wipe samples, (4) hand rinsing, (5) small cassette samplers, and (6) specially manufactured samplers. With the exception of studies conducted by Que-Hee (1985) and Roberts (1989, 1991), there has been very little effort made to validate the collection techniques used.

Residues that can be dislodged from carpet and floor surface can be sampled to estimate transfer to the skin. Such residues include dust, materials on the dust, and droplets of pesticides that may not be attached to dust. Wipe sampling, which is frequently used, is inherently limited by variability in the performance of the person doing the sampling. The polyurethane foam (PUF) roller sampler has been suggested as an alternative dust pick-up device that can be reproducible. It is a weighted rolling device designed to simulate the transfer of dislodgeable residues and dust to a child's skin. The sampler uses a stainless steel roller assembly on which a PUF cylinder is mounted. The pressure applied by the assembly through the cylinder to the sampled surface (7000 pascals) is similar to the pressure exerted by a crawling, 9-kg child. The PUF cylinder may be used either moistened or dry to evaluate transfer to wet

TABLE 27.5

Pesticides that can be measured using low volume PUF sampling with GC/ECD (U.S. EPA, 1989e)

Chlorinated hydrocarbons		
Aldrin		Methoxychlor
p,p,-DDT		Maxacarbate
p,p,-DDE		Mirex
Dieldrin		*trans*-Nonachlor
Dicofol		Oxychlordane
2,4,5-Trichlorophenol		Pentachlorobenzene
Pentachlorophenol		Folpet
BHC (and-hexachlorocyclohexanes)		Heptachlor
Captan		Heptachlor epoxide
Chlordane, technical		Heptachlorobenzene
Chlorothalonil		Lindane (-BHC)
2,4,-D esters		
Organophosphates	Ureas	Carbamates
Chlorpyrifos	Chlortoluron	Bendicarb
Diazinon	Diuron	Carbaryl
Dichlorvos (DDVP)	Fluormeturon	Carbofuran
Ethylparathion	Linuron	Mexacarbate
Malathion	Tebuthiuron	Propoxur
Methyl parathion		
Ronnel		
Pyrethrin		Triazine
Allethrin	Pyrethrin I	Atrazine
d-*trans*-Allethrin	Pyrethrin II	Propazine
Dicrotophos	Resmethrin	Simazine
Fenvalerate		

or dry skin. An initial laboratory study found no significant differences in transfer rates for 13 pesticide residues on aluminium foil using the dry PUF roller compared to various hand press motions by two subjects.

The Sirchee–Spittler hand-held vacuum is a modification of the Sirchee Evidence Vacuum with a stainless steel (325–500 mesh) screen used in place of a coarse cloth filter. The screen is glued to the bottom of a plastic cup and traps about 95% of house dust. The sampling cup can be readily emptied into a sample envelope by simply tapping it sharply on a hard surface. The screen can be cleaned with compressed air. This technique was used in the Boston Lead Demonstration Project to measure the Pb concentration (ppm) and loading ($\mu g/m^2$) on rugs and bare surfaces to evaluate control measures and estimate exposure. Samples sizes ranging from 25 to 200 mg can be collected in 5 min.

A small high volume surface sampler (HVS3) has been developed for the EPA to collect surface dust samples with repeatable sampling performance. It provides for monitoring and control of air flow and pressure drop across the nozzle. Laboratory tests showed a 5% variation in collection efficiency from sample to sample. The HVS3 showed a collection efficiency of 67–69% fine dust embedded in plush and level loop carpets. The cyclone of the HVS3 retained about 99% of the collected dust, more than 97% of the aldrin, chlordane, chlorpyrifos, dieldrin, and heptachlor on a doped sample of house dust, and over 99.8% of the Pb in natural house dust in laboratory tests. The HVS3 can collect a large, representative sample of up to 200 g in 15 min that can be used for multiple analyses and bioassays. It has been field tested in two pesticide studies and one Pb study on house dust exposures in homes.

To provide adequate identification of the many organics that can be adsorbed onto floor dust, semivolatile organic pesticides and polycyclic aromatic hydrocarbons (PAHs) can be analyzed by a selective method, such as gas chromatography/mass spectrometry (GC/MS). With selected ion monitoring, GC/MS can simultaneously determine 30 target analytes at 0.1–1 ppm levels. Dual-column GC with electron capture detection provides additional sensitivity (0.05 ppm) for quantitation of chlorinated pesticides.

Sample contamination is a major problem in pesticide analysis. Therefore quality control procedures are very important. For each investigation field, analysis and solvent blanks should be included in the measurement program at a level of 5% with a minimum of one of each blanks. Blank levels should not exceed 10 ng/sample for single components and 100 ng/sample for mixtures of several pesticides. Replicate determinations of collection efficiency should be made using spiked samples. Relative standard deviations for the replicate determinations of 15% or less and recoveries of 75% or more are acceptable (U.S. EPA, 1989a,b, 1991b).

Polychlorinated biphenyls (PCBs)

PCBs are complex mixtures of individual congeners. A total of 209 congeners exist ranging from mono- to perchlorinated species. Although sampling and analytical methods for PCBs are similar to those for chlorinated pesticides, the complexity of their mixtures and the growing need to measure the concentrations of a variety of individual PCB congeners, especially those with a coplanar configuration, have led to some particular analytical features. No standarized sampling and analytical method and measurement strategy has yet been established, although work is under progress to develop guidelines for PCB determination in indoor air.

Besides polyurethane foam (PUF), Florisil and XAD2 (styrene–divinylbenzene resin) columns are used for air sampling. Unpolar solvents such as *n*-hexane or toluene are used for extraction. Before GC/ECD or GC/MS analysis, the extracts are usually passed through a clean-up column. SiO_2 columns coated with sulphuric or benzosulphonic acid or Al_2O_3 columns are mostly used for clean-up.

Careful selection of GC columns is important if individual congeners have to be measured. Possible interferences have been discussed in detail by Larsen et al. (1992).

27.2.7 Polynuclear aromatic hydrocarbons

Concentrations of polynuclear aromatic hydrocarbons (PAHs) and related semivolatile organic compounds (SVOCs), have often been found to be higher indoors than outdoors. Depending upon their equilibrium vapor pressures and the temperature of the environment, PAHs are found in the gas phase, distributed between gas and particle phases, or entirely in the particle phase (Cautreels and Van Cauwenberghe, 1978; Ligocki and Pankow, 1989). Because PAHs are generally produced by combustion, they are found in the respirable particle (D_{50} = 3.5 μm) fraction (Miguel and Friedlander, 1978; Van Vaeck and Van Cauwenberghe, 1985; Offermann et al., 1991).

Sampling systems for PAHs must therefore collect both particles and vapour. This is achieved by combining a filter for the collection of particles with — in series — a XAD2 sorbent trap to collect that portion of PAHs which is in the vapour phase (Chuang et al., 1987a; Wilson et al., 1985).

Polyurethane foam, which has low resistance to airflow and has been validated for the collection of pesticides and PCBs (Lewis et al., 1988), does not collect or retain well PAHs with fewer than four rings, such as anthracene or phenanthrene. Additionally, the PUF sample extracts give widely varying results in microbioassays compared to extracts from other sorbents. Non-polar two-ring and larger aromatic compounds and most of the more polar aromatic derivatives are efficiently collected and retained on XAD-2 (Chuang et al.,

1987b). To collect simultaneously both PAHs and nicotine (used as marker for environmental tobacco smoke) or other small SVOCs, XAD-4 resin, which is chemically similar to XAD-2, but with smaller pores and greater surface area, proved to be superior (Chuang et al., 1990).

During sampling with the filter-sorbent combination, part of the PAHs are desorbed from the particles collected on the filter (or revolitilized) and trapped on the sorbent together with the vapour phase PAHs. Revolitilization may contribute a large amount to the PAHs collected on the sorbent which in some cases is of the same order of magnitude as the amount originally in the gas phase (Coutant et al., 1988). Since it is usually not possible to distinguish between vapour phase and revolitilized PAHs, it is also not possible to determine separately the amounts of PAHs originally in the vapour phase and adsorbed on suspended particles. Therefore, except in studies specially designed to measure phase distributions, the filter sorbent pairs are extracted together and the extracted amounts reported as total concentrations.

For recovery of PAHs from filters and sorbents, Soxhlet extraction with ether/hexane for PUF and with dichloromethane for XAD-2 or XAD-4 is mostly used, although the choice of the extraction solvent depends on the polarity of the analytes. Supercritical fluid extraction (SFE) with CO_2 is effective in some cases for non-polar PAH (Hawthorne and Miller, 1986; Wright et al., 1987), although modifiers may be necessary to remove polar materials. The use of SFE for air sample analysis is still under investigation. Analytical procedures usually involve reverse-phase HPLC coupled with fluorescence detection or gas chromatography coupled with mass spectrometric detection (GC/MS). Because GC/MS requires little clean-up it represents the more cost-effective method (Chuang et al.,1987a). To achieve the highest sensitivity, positive chemical ionization (PCI) GC/MS gives good results for PAHs while negative chemical ionization is more appropriate for nitro-PAHs and other electrophilic compounds (Wilson et al., 1991).

27.3 PHYSICAL POLLUTANTS

27.3.1 Particulate matter

Several investigations of the methodology and measurements of personal exposure to respirable particles have been conducted over the last 15 years (Turner et al., 1979; Dockery and Spengler, 1981; Sexton et al., 1984, 1988; Lioy et al., 1990; Buckley et al., 1991). Improvements in methodology have been recommended (Spengler and Soczek, 1984) in order to better understand the relationship between exposures from indoor and outdoor sources and the effects of particle control technologies over time.

When measuring particulate matter (PM) the following steps should be distinguished. Sample collection and sample analysis: during the sampling step the choice of the monitoring device used depends on what particle size is investigated, and on whether personal exposure or micro-environmental conditions have to be estimated or the collection of time integrated samples or continuous monitoring are of interest. Supposing no direct-reading monitoring is used, the analytical step always coincides with the gravimetric analysis of the sample (filter weighing), whereby rigorous atmospheric conditions must be respected in order to attain homogeneous conditioning of the samples. Optional steps are represented by the characterization of the morphology with optical methods (e.g. scanning electron microscopy), and the chemical and physical (e.g. PAHs, radon decay products), or elemental composition with energy-dispersive X-ray fluorescence (XRF).

Concerning the sampling step a more detailed distinction must be made as follows:

- measurement of total suspended particulate matter;
- collection of settled particulate matter;
- measurement of personal exposure to the respirable (PM_{10}) or the inhalable ($PM_{2.5}$) fraction of PM, identified as those portions of aerosols that will respectively deposit in the nasopharyngeal region (aerodynamic diameter $D_{ae} \leq 10$ μm) and the tracheobronchial and alveolar regions of the lung ($D_{ae} \leq 2.5$ μm) (Phalen et al., 1986);
- methodologies aimed at supplying frequency distributions for both the fine (<2.5 μm) and coarse (2.5–10 μm) fractions of PM_{10}.

A recent program named Particle Total Exposure Assessment Methodology (PTEAM) assessed the frequency distribution of PM_{10} total exposure in an urban population (Thomas et al. 1993; Clayton et al., 1993; Mamane et al., 1993). The program included the testing and development of methodologies necessary for measuring personal exposure (Wiener, 1988; Spengler et al., 1989; Anderson et al., 1989) and indoor and outdoor particle PM_{10} and $PM_{2.5}$ concentrations. The 1990 PTEAM Study provided a strong test of the methodology, with on-site filter weighing and collection of approximately 2900 personal, indoor, and outdoor particle samples over a 48-day time period.

Sample collection

For sample collection, impactors and filters are used in combination with pumps. Impactors are instruments consisting of a housing which constrains the air flow past a sensitized sampling plate. Small impactors are available for measurement of personal PM_{10} and $PM_{2.5}$ exposures (MSP, Inc.) and have been used in a series of studies (Kamens et al., 1991; Clayton et al., 1991; Thomas et al. 1993). Aerosols enter the impactor through nozzles in the cover, with

varying diameters depending on the PM cutpoints (PM_{10} or $PM_{2.5}$) of interest. The annular impactor plate is placed directly underneath the nozzles to capture particles with aerodynamic diameters above the cut-point size. Air is sampled at 4 l/min using a flow-controlled battery operated pump and particles smaller than the cut-point are pulled through the centre hole in the impaction plate and onto a 2-μm pore teflon filter and a glass-fibre backing filter. When using personal exposure monitors (PEM), the pump, the flow controller, and the battery pack have to be placed together in a soft case worn on a belt or shoulder strap to minimize inconvenience to surveyed persons. Impactors are placed just below the collar bone using shields to keep clothing and hair away from impactor's entrance. For fixed-site monitoring, similar devices are used as stationary indoor monitors (SIMs) and outdoor monitors (SAMs) with AC power supply, although sampling with dichotomous samplers (e.g. Sierra 244E, Andersen Samplers, Inc.) and high-volume (e.g. PM_{10} Critical Flow Hi-Volume Sampler, Wedding and Associates, Inc.) is usually performed according to reference methods (U.S. EPA, 1990). Dichotomous samples are collected on Teflon filters identical to those used for collecting PEM, SIM and SAM samples while for high-volume samplers large surface glass fibre filters (e.g. Whatman EPM 2000) are used.

Background contamination of the filters is an important issue. In the PTEAM study (Thomas et al., 1993), short sampling times (12 h) and low flow rates (4 l/min) combined to produce very small air sample volumes (approximately 3 m^3). Median mass increase on 51 PEM, SIM, and SAM field blanks was 9.0 μg. The calculated Method Detection Limits (MDL) for PEM, SIM, and SAM samples was equal to 8.3 $\mu g/m^3$ (2.88 m^3). Calculated MDL were 1.0 $\mu g/m^3$ for dichotomous samplers (11 m^3), and 1.5 $\mu g/m^3$ for hi-vol samples (815 m^3) (Thomas et al., 1993).

Nongravimetric samplers, which use techniques such as light scattering and piezoelectric crystal frequency shift, are also available commercially. These samplers have proven useful in screening studies, but all have significant technical limitations, such as poor sensitivities or interference problems. Their utility as exposure monitors, either as PEMs or microenvironment samplers, has not been adequately demonstrated.

Sample analysis

Gravimetric analysis: The amount of particle mass collected is always determined by gravimetric analysis. Filter conditioning and weighing operations are conducted in particular weigh laboratories where atmospheric conditions are held as constant as possible (±3°C, 50±2% relative humidity). Filters are conditioned in this atmosphere for a minimum of 24 h before weighing. Accuracy tolerance criteria require that balances for PEM, SIM, SAM and

dichotomous samples be within ±0.05 mg and balances for high-volume samples be within ±0.005 g of NIST traceable weights (Thomas et al., 1993).

Elemental analysis: After collecting the exposed filters and obtaining the gravimetric results, the filters may be analyzed by energy-dispersive X-ray fluorescence (XRF), whereby the following elements are deemed to be of primary interest: Si, Al, Ca, Fe, Mn, Ni, Pb, As, K, Se, V, Br, and Cd. Scanning electron microscopy (SEM) equipped with an energy dispersive X-ray analyzer (SEM-EDX) are nowadays available for the contemporary determination of size distribution, morphology, and elemental analysis.

Chemical and physical analysis: For the determination of e.g. polynuclear aromatic hydrocarbons, pesticides or radon decay products in suspended or settled PM the reader is referred to specific sections of this book. Techniques are available for the retrieval of condensed organic compounds from dust particles by thermal desorption (Wolkoff and Wilkins, 1993) or SFE (Schlitt et al., 1993).

27.3.2 Asbestos

The analysis of ambient air samples for asbestos has utilized techniques different from those used in occupational settings because typical urban air may contain up to 100 $\mu g/m^3$ of particulate matter in which the researcher is attempting to quantify asbestos concentrations from about 0.1 to perhaps 1000 ng/m^3. Thus, asbestos may constitute only 0.0001 to 1% of the particulate matter in a given air sample. Asbestos found in ambient air has a size distribution such that the vast majority of fibres are too short or too thin to be seen with an optical microscope. In many cases, these fibres and fibrils will be agglomerated with a variety of other materials present in the air samples.

The only effective method of analysis uses electron microscopy to enumerate and size all asbestos fibres (Nicholson and Pundsack, 1973; Samudra et al., 1978). Samples for such analysis are usually collected either on a Nuclepore® (polycarbonate filter with a pore size of 0.4 μm or on a Millipore® (cellulose ester) filter with a pore size of 0.8 μm. In some cases the Millipore is backed by nylon mesh. Samples collected on Nuclepore filters are prepared for direct analysis by carbon coating of the filter to entrap the collected particles. A segment of the coated filter is then mounted on an electron microscope grid, which is placed on a filter paper saturated with chloroform so that the chloroform vapours dissolve the filter material. (Earlier electron microscopic analysis utilized a rub-out technique in which the ash residue was dispersed in a nitrocellulose film on a microscope slide and a portion of the film was then mounted on an electron microscope grid for scanning.)

Samples collected on Millipore filters are prepared for indirect analysis by ashing a portion of the filter in a low-temperature oxygen furnace. This removes the membrane filter material and all organic material collected in the sample. The residue is recovered in a liquid phase, dispersed by ultrasonification, and filtered on a Nuclepore filter. The refiltered material is coated with carbon and mounted on a grid as described above. The samples are then subjected to analysis. Chrysotile asbestos is identified on the basis of its morphology as seen with the electron microscope. The identification of amphiboles is in addition performed by selected area electron diffraction patterns, supplemented by energy-dispersive X-ray analysis. Fibre concentrations in fibres per unit of volume (such as fibres/cm^3, fibres/m^3, etc.) are calculated based on sample volume and filter area counted. In some cases, mass concentrations are reported using fibre volume and density relationships. However, mass concentrations may not be reliable if the sample contains fibrous forms, such as clusters, bundles, and matrices, in which fibre volume is difficult to determine. These materials may constitute most of the asbestos mass in some samples, particularly those reflecting emission sources. Current fibre counting methods do not include those clumps. However, many of them are respirable and to the extent that they are broken apart in the lungs into individual fibres, they may add to the carcinogenic risk. On the other hand, methods that break up fibres generally disperse the clumps as well. In such analyses, the clumps would contribute to the mass.

In much of the earlier analyses of chrysotile concentrations in the United States, the ashed material was either physically dispersed or disrupted by ultrasonification. Thus, no information was obtained on the size distribution of the fibres in the original aerosol. Air concentrations were given only in terms of total mass of asbestos present in a given air volume, usually nanograms per cubic meter (ng/m^3). With the use of Nuclepore filters and appropriate care in the collection of samples and their processing, information on the fibre size distribution can be obtained, and concentrations of fibres of selected dimensions can be calculated.

Current measurements of low-level contamination with asbestos use electron microscope techniques, which determine the total mass of asbestos present in a given volume of air. Previous measurements of concentrations of fibres longer than 5 μm were made using optical microscopy, or from optical microscopy of total particulate samples. Occupational studies used the latter techniques. If information regarding health effects from these studies is to be extrapolated to measurements made in nonindustrial indoor spaces, a relationship between optical fibre counts and mass of asbestos determined by electron microscopy must be established. Crude estimates of a conversion factor relating fibre concentration in fibres per millilitre (f/ml) to airborne

asbestos in micrograms per cubic metre ($\mu g/m^3$) is derived from several studies, and is detailed in the *Airborne Asbestos Health Effects Update* (Nicholson, 1986). These crude conversion factors relating mass concentrations to optical fibre concentrations range from 5 to 150, and by necessity introduce large uncertainties. For conversion of low-mass concentrations to fibre count the geometric mean of the above range of conversion factors, which is 30 $\mu g/m^3/f/ml$ is used in the EPA asbestos health assessment. The geometric standard deviation of this value is 4, and this uncertainty severely limits any extrapolation in which it is used. In the case of amosite, the data of Davis et al. (1978) suggest that a conversion factor of 18 is appropriate. However these data yield lower chrysotile values than all other chrysotile estimates; therefore, they may also be low for amosite (Nicholson, 1986; U.S. EPA, 1987c).

27.3.3 Man-made mineral fibres

For man-made mineral fibres (MMMF), analyses have been restricted largely to the measurement of total airborne mass concentration or, more recently, to the determination of airborne fibre number concentrations by phase contrast optical microscopy (PCOM). The method for sampling personal exposure levels involves drawing a measured volume of air through a filter mounted in a holder that is located in the breathing zone of the subject. Static sampling methods are not recommended for measuring personal exposure. When measuring mass concentrations of MMMF, either cellulose ester membrane or glass fibre filters can be used. The filters are stabilized in air and weighed against control filters, both before and after sampling, to permit correction of weight changes caused by varying humidity. Only cellulose ester filters are used for assessing fibre number concentrations. In this case, the filter is made optically transparent with one of several clearing agents (e.g. triacetin, acetone, or ethylene glycol monomethyl ether) and the fibres present within random areas are counted and classified using PCOM. For this purpose, a fibre is defined as a particle having a length to diameter ratio (aspect ratio) ≥ 3 and a length ≤ 5 μm. Respirable fibres are those with diameters ≤ 3 μm. Fibres with diameters exceeding 3 μm are termed non-respirable fibres.

Sampling strategies should be well-designed, based on careful consideration of "how", "where", and "for how long" to sample, as well as "how many samples" to collect to ensure that the results are comparable. Sampling strategies will vary depending on the reason for sampling, e.g., epidemiology, dust control, etc. Sampling strategy has been discussed in the literature (NIOSH, 1977b; Valic, 1983; WHO, 1984).

Although the basic methods for the determination of total airborne mass and fibre number concentrations in most countries are similar, differences in

the sampling procedure, the filter size and type, the clearing agent, and the microscope used, and, particularly, statistical and subjective errors in counting, contribute to variations in results. In order that results from different countries should be more comparable, a reference method for monitoring MMMF at the work-place based on PCOM was proposed by WHO in 1981 and has been widely used in a slightly modified form (WHO, 1985) since that time. The results of recent slide exchanges among participating laboratories have shown a maximum systematic counting difference of about 1.8 times for this method (Crawford et al., in press).

The contrast of the phase image for the counting of glass fibres was improved by the low-temperature ashing of the membrane filter attached to glass slides by acetone vapours followed by microscopic examination using simple Köhler illumination. Automatic counting methods are currently being developed and may in future provide greater consistency in results.

The improved resolution of electron microscopy and the identification capacity, particularly of the analytical transmission electron microscope (TEM) with selected area electron diffraction (SAED) and energy dispersive X-ray analysis (EDXA), make these methods more suitable for analysis of fibres in the general environment, where MMMF may constitute only a small fraction of the airborne fibrous material. However, to date, use of these methods for the determination of MMMF has been restricted largely to the characterization of airborne fibre sizes in the occupational environment.

In analysis by SEM, the fibres collected on polycarbonate filters can be directly examined. This avoids the need to use transfer techniques that may affect the fibre size distribution. WHO has developed a reference method for SEM, principally to characterize fibre size (WHO, 1985). Using the same sampling method as for the PCOM method, samples are collected on a polycarbonate (Nuclepore) or PVC-copolymer membrane filter (Gelman DM 800) and observed at a magnification of 5000 times. Fibre lengths and diameters are measured from optically enlarged images of photomicrographs.

In the past, methods of sample preparation for TEM have varied considerably, making comparison of values obtained by different investigators difficult. At present, direct transfer preparation techniques involving carbon coating of particles on the surface of a polycarbonate or membrane filter and indirect sample preparation methods, in which attempts have been made to retain the fibre size distribution, are the most widely accepted for analysis of fibres in air and water.

The size distribution of "superfine" MMMF has been successfully measured by a direct transfer sampling method on a membrane filter, subsequent analysis by analytical TEM, and fibre measurement by an image analysis system. On the basis of their results, the authors concluded that a substantial

proportion of the fibres would not have been detected by PCOM or SEM, even fibres longer than 5 μm.

27.3.4 Radon

27.3.4.1 Indoor radon measuring methodology

Introduction

A correct methodology is of great importance in measuring radon concentration in indoor air. Radon gas is usually present in indoor air, coming mainly from soil through small cracks in building foundations and driven by pressure differences, or from building materials, or from domestic use of deep well water through its emanation, or from outdoor air (see also Section 3.4).

Radon atoms decay through a chain, the first four short-lived elements of which are referred to in various ways: radon daughters, radon progeny, radon decay products. These elements, unlike radon, are not inert gases and shortly after their formation attach themselves to aerosol particles; only a small fraction of them remain in unattached form, depending on aerosol size and concentration and on ventilation (Nazaroff and Nero, 1988). These daughters, when inhaled together with air, decay in the lung emitting radiations, in particular alpha particles, and cause, especially those attached to small size aerosol or in unattached form, damage to sensitive lung cells, thereby increasing the probability of cancer developing. However although the dose is mostly due to inhaled radon progeny (and its characteristics) more than to radon, some studies (James, 1987) show that, in domestic ambient air, changes in ventilation rate produce opposite variations in the equilibrium factor F (i.e. the ratio between radon progeny and radon gas concentration) and in the unattached fraction, so that the dose to the lung remains relatively constant at a given radon concentration.

In IAQ investigations, radon and/or radon daughter concentrations in indoor air are usually measured[1], depending on the aim and the available equipment. In any case the radon concentration is related to those of the radon progeny through the equilibrium factor F, whose values are usually in the range 0.2–0.7 for domestic air. Usually 0.5 is assumed, in which case the radon concentration is the double of the radon progeny concentrations (see also Section 3.4.1.1). More recently F = 0.4 is preferably assumed (UNSCEAR, 1988; ICRP 1993).

In this section the general methodology of radon (and radon daughter)

1 In some cases, in order to better identify radon sources and entry points, other quantities can be measured, such as radon or radium concentration in water and radon flux from walls and floors.

concentration measurements for a single dwelling or building is outlined, while in the next sections some main measurement methods, instruments and detectors will be illustrated. For a more detailed analysis, including some measuring protocols, specialised publications should be consulted (e.g. NEA/OECD, 1985; NCRP, 1988; U.S. EPA, 1987a, 1989c; DOE 1990).

Radon concentration temporal variations and measurement duration

For a correct approach to IAQ investigation, concentration variations of radon and its progeny must be taken into account. These variations occur on many time scales, from hourly to annually, and depend on seasonal factors and weather conditions, building characteristics, operation of dwelling heating and refrigerating systems, living habits, source relative strength, etc. Usually concentrations are higher in the evening and night than in the late morning and early afternoon; higher in the winter than in the summer; however it is almost impossible to predict these variations with enough accuracy.

The longer the duration of a measurement the lower in general is its variability: individual very short-time measurements (ca. minutes or a few hours) can show variations higher than a factor of 10. Measurements lasting a few days generally show variations less than a factor of 10 (e.g. Sextro, 1990), while the seasonal variations found in measurements of six-months duration are usually well within a factor of 5 (e.g. Hess et al., 1985; Bochicchio et al., 1992), while annual variations are well within a factor of 2, e.g. measurements carried out over a 5-year period in 40 residences in Grand Junction (Colorado, USA) show a mean coefficient of variation of approximately 22% (Martz et al., 1991).

Because of the magnitude of these variations, 1-year measurements are considered the best compromise to estimate the "average" value.

Length of sampling time

Radon and/or its progeny concentration measurements of four types of duration can be used, depending on the purpose: long-term, short-term, very short-term and continuous monitoring.

(a) *Long-term* (typically one year or some months): these measurements give the best estimates of the average value. In particular 1-year measurements are the most appropriate, except for cases where the dwelling is not lived in for a long period of the year. If for some purposes a quicker estimation is needed, e.g. for screening purposes, a measurement period shorter than one year could be used. This requires, however, a careful seasonal correction factor in order to estimate the average value, thus introducing a potentially high uncertainty that depends on the way the correction factor is obtained. This procedure can be considered reliable only in cases where many previous

determinations of seasonal variations in the same area and in the same type of dwellings were made with results in a narrow range. Long-term measurement instruments are usually available only for radon concentration, because the long-term integrated measurement of radon progeny is very difficult due to technical and scientific problems.

(b) *Short-term* (typically 1–10 days): these measurements can be used for screening purposes, i.e. to quickly discriminate between very low, medium and very high concentrations. Another use of screening measurements is to quickly select in a group of dwellings or rooms those with higher radon concentrations. To minimise underestimations, protocols usually suggest measuring conditions tending to maximise radon concentration (U.S. EPA, 1987a). The results of short-term measurements cannot be used to accurately estimate the long-term average value, and a follow-up long-term measurement is needed (U.S. EPA 1987b, 1992; White et al., 1994).

(c) *Very short-term or grab-sampling* (typically some minutes or tens of minutes): these are generally used only for detailed building diagnostic and/or research purposes.

(d) *Continuous monitoring:* the main purpose of this type of measurement is to control particular occupational environments, e.g. mines, and/or research activity. It could also be used for some of the purposes of short-term and grab-sampling measurements.

Number and location of measuring points

The choice of the type of rooms and points where measurements have to be performed is related to the objective of the investigation.

For *human exposure assessment*, only rooms where people live should be selected, in particular bedrooms, where most time indoors is usually spent. Kitchens and bathrooms should not be selected, also because the particular air conditions present in these rooms can affect the results of some detectors. The measuring points should also be representative of the air inhaled by the people. Moreover the detector location should not be disturbed and far enough from heat and draught sources.

For *building/dwelling investigations*, non-living rooms and other closed spaces, such as basements and cellars, can also be selected for measuring radon concentration. Moreover, the measuring points can be selected in order to discover radon inlet routes.

The number of rooms and measuring points depends on the type and size of the building/dwelling.

For *residential buildings*, one or more rooms (e.g. bedrooms) are needed in the case of single-storey dwellings, where the radon concentration is usually homogeneous, while two or more rooms (e.g. bedroom, living room) are sug-

gested in case of two-storey detached dwellings where the radon concentration is usually higher on the ground floor (where the living room is) than on the first floor (where the bedrooms are). Similar considerations are valid for small offices too.

For *non-residential large buildings*, such as schools, large offices, large stores, hotels, etc., more points and rooms should be selected, depending on the size of the buildings and rooms, the type of building construction, the type of ventilation/heating system and so on. For example the U.S. EPA recommend for schools that all rooms on or below ground level have to be measured (U.S. EPA, 1989d). For other large buildings some preliminary protocol has recently been proposed (Wilson et al., 1993).

Usually one detector per point is used, but the use of two (or eventually more) detectors produce a more reliable measurement (see also below), and could be suggested depending on the objective, the resources and the type of detector.

Measurement quality assurance

A quality assurance program is needed to assess and guarantee the measurement validity, in particular its precision and accuracy (e.g. NCRP, 1988; U.S. EPA, 1989c).

As regards measurement *accuracy*, detectors/instruments have to be first of all calibrated, then checked frequently. Periodic participation in measurement intercomparisons is also to be recommended. Calibration measurements are usually performed in a "radon chamber", in which a known quantity of radon is introduced. In general the calibration of radon daughter detectors is more difficult than the calibration of those for radon, due to many reasons, one of which is that there is no primary standard for radon daughters (NCRP, 1988; Nazaroff, 1988).

As regards measurement *precision*, it is assessed by replicate measurements, i.e. exposing many detectors contemporaneously to the same concentration. This allows an estimate to be made of the coefficient of variation of the detectors at that value of concentration (Goldin, 1984). A periodical check of the precision should also be done.

27.3.4.2 Categories of measurement

Most methods of measuring radon (Rn) and its decay products (RnD) are based on the detection of the alpha particles emitted by these radionuclides during their radioactive decay. A small number of methods are based on the detection of emitted gamma rays and some techniques exist which detect beta ray decays. Useful reviews of these techniques are those of NCRP (1988) and Nazaroff (1988).

It is important to distinguish between methods which measure the concentration of the gas radon and those which measure the concentrations or other characteristics of airborne radon decay products. In Rn and RnD measurement the techniques may be classified as being active or passive. Active techniques are those which require electric power and/or the use of air pumps to collect activity from the air. Passive techniques are those where the detector, while installed at the sampling location, does not require electric power. The air containing Rn and RnD usually enters such passive detectors by free diffusion and the radiation detecting medium itself does not require any power supply (i.e., alpha track plastics or activated charcoal). Passive techniques are usually simple, cost effective and easy to use. They are admirably suited for survey work and for long-term measurements. Some instruments are available where the detector requires a low level of power supplied by a battery but no pump is used. These fall into a class of detection technique intermediate between passive and active.

It is also important to distinguish between the different sampling techniques in terms of their temporal characteristics. These may be divided into three principal types:

(a) Grab sampling:

Here the activity of Rn or RnD in a discrete sample of air taken at a single location in a short period of time (from about 1 sec to about 20 min) is measured. This approach is at best useful for initial screening purposes or for spot-checking of the efficacy of remedial actions. It is of very limited use for determinations of average indoor air radon concentrations which are more appropriately determined on the basis of long-term measurements.

(b) Continuous sampling:

Here air is drawn either continuously (or semi-continuously) for long periods of time through a Rn or RnD detecting instrument. This type of approach gives information on the time dependence of the airborne activities in a building. Information which may be obtained using continuous sampling include the ratio between day-time and night-time concentrations in a building, diurnal variations etc. Such information is quite useful in deciding on strategies used to reduce occupational exposures where occupancy factors are much smaller than in dwellings.

(c) Time integrating sampling:

Techniques using time integration consist of using a device which will yield a single determination of airborne activity averaged over some chosen period from a few days to a year or longer. As time integrating sampling is usually

but not exclusively carried out with inexpensive passive detectors it is the preferred approach in survey work. In reaching a decision on the necessity of remedial action it is generally considered in European Community countries that integrating measurements of minimum duration 3 months should be made. Grab sampling or short-term integrating sampling of a few days are considered inadequate for making accurate estimates of long term exposure of occupants of a building to radon. The recommendations of the Commission of the European Communities (CEC) of February 1990 stress the need and desirability of making long-term (generally one year) integrating measurements in order to determine if indoor air is above or below the appropriate radon reference or actions levels (CEC, 1990). In countries such as the U.S. where real-estate transactions may require urgent evidence of the indoor radon concentration in a building, short-term integrating techniques of a few days duration may be recommended providing they are carried out according to a recognised measurement protocol (White et al., 1990).

27.3.4.3 Instrumentation

The following are brief descriptions of the principal characteristics of the instruments or devices most commonly used to measure concentrations of indoor Rn and RnD.

(a) Scintillation cells

This is one of the oldest and most reliable type of device for measuring the concentration of radon gas. It exists in a number of forms and can be used for grab-sampling or for continuous long term measurements. A radon scintillation cell typically is a small metal cylinder equipped with one or two vacuum-tight inlet/outlet valves mounted on one end. The opposing end of the cylinder is a clear glass or plastic window. The internal volume of such a cell is typically about 100 cm^3. The inner surfaces of the cylinder, excepting the window, is uniformly coated with ZnS(Ag) powder which is a very efficient scintillator for alpha particles. The most common form of such a scintillation cell is called a Lucas cell (Lucas, 1957). The scintillation cell filled with an air sample is placed in optical contact with a photomultiplier tube (PMT). The scintillations or flashes of light caused by the alpha particles from radon, Po-218 and Po-214 which strike the ZnS(Ag) are recorded by the PMT and its associated electronics. Using appropriate calibration and decay scheme factors the radon gas concentration may be determined from the rate at which the pulses are recorded.

In grab sampling mode an air sample is taken into the cell which is then sealed and after a delay of 3 h, to allow for approximate radioactive equilibrium between radon and its short-lived decay products to be reached, a count rate is taken.

Active versions using flow through scintillation cells are commercially available and can be used for continuous monitoring of radon concentrations in a building. They are usually equipped with data storage and printout facilities and a range of cycle time.

(b) Alpha track detectors

A number of plastic or polymeric materials are available which have the property that the primary damage caused in them by the passage of alpha particles remains fixed in them and may be made visible as tracks by means of a suitable etching procedure (Cartwright et al., 1978). These materials are often called Solid State Nuclear Track Detectors (SSNTDs). The commonest of those in use for radon detection are the cellulose nitrate film (LR-115), the thermoset polymer plastic (CR-39) and the polycarbonate plastic (Makrofol). The passage of an alpha particle through a SSNTD produces a narrow primary damage trail or latent track along the length of its path in the material (typically 20–70 μm). The production of a visible track, by chemical or by electrochemical etching, is made possible because the damaged material etches faster than the undamaged or bulk material. The tracks are very characteristic and for chemical etching of CR-39 the tracks appear under transmission optical microscopy as dark three dimensional conically shaped features. These may be identified and counted by a human microscopist or by means of an automatic computerised image analysis system. For LR-115 detectors, etched tracks can be rapidly counted by a "spark-counter" (Cross and Tommasino, 1970).

The use of such alpha track detectors for passive long term integrating measurement of indoor radon is very popular both for large scale surveys and for radon measurements in single buildings (Alter and Oswald, 1983). Most radon alpha track detectors consist of a few cm^2 of the SSNTD material mounted inside a small, almost airtight, closed container. The air containing radon enters the inner volume of the container by diffusion. Alpha particles from radon and its in-grown decay products which strike the SSNTD produce the alpha tracks from which, by means of calibration factors, the mean radon concentration may be determined. These closed radon alpha track detectors typically have dimensions giving them a volume range of about 20–75 cm^3. In some versions of this detector type the SSNTD material is mounted open-faced or bare on a wall in a building to record directly alpha particles from the air. For a variety of operational and other reasons there is a preference for the closed type alpha track detector although open faced types of excellent characteristics using LR-115 are also used extensively (Rannou et al., 1986).

Alpha track detectors are inexpensive, reliable and easy to use. They compare very well in sensitivity and accuracy with other types of radon detectors. A minor disadvantage, however, is that they do not give a measure-

ment in the field and must be sent to a processing laboratory if the user is not equipped to process them.

(c) Charcoal detectors

The adsorption of radon by activated charcoal has been used for many years as a detection method (Cohen and Nason, 1986). The method is very simple. The gamma radiation emitted by radon and its ingrown decay products in the charcoal is measured by means of a gamma ray detector such as Sodium Iodide (NaI(Tl)). A charcoal detector suitable for indoor radon measurements usually consists of a pocket sized flat metal cylinder containing the charcoal. The best versions of these include a diffusion barrier between the charcoal and the air. In this way the rate of radon adsorption is proportional to its concentration in the air. The charcoal method is a short-term integrating method. It is passive, inexpensive and of sufficient sensitivity that the radon concentration at typical indoor levels can be measured using an integrating time of a few days. It has been used extensively for large scale radon surveys, principally in the U.S., and is also very popular for making determinations of indoor radon concentrations in the space of a few days as may be required for real estate transaction purposes. The principal disadvantage of the charcoal detector method is that its useful integrating time is limited to a little over one week. This arises because radon has a radioactive half-life of 3.8 days. Consequently the gamma activity from radon adsorbed at a time in the past more than about two half-lives previously will be decayed to a small fraction of that from the most recently adsorbed radon. Another similar disadvantage, arising again from the half-life of radon, is that following the completion of its exposure in a building the charcoal detector containing the adsorbed radon must be sent quite promptly to the measuring laboratory for analysis before the gamma activity has decayed to an insignificant level. Moreover, the accuracy of the method is sensible to significant variations of radon concentration during exposure period. Some modified versions of charcoal detectors have been developed to overcome these disadvantages. In one of them the continual gamma emission in the charcoal is recorded by a TLD (Thermoluminescent Detector) chip inserted into the charcoal (Stranden et al., 1983). The mean radon concentration can be determined subsequently using a TLD reader. This approach is not in general use.

Unlike alpha track detectors, charcoal detectors are not actually true detectors of radon or its decay products. They are essentially absorbers which collect radon for subsequent gamma counting. The alpha track detectors on the other hand record continually and permanently store the damage caused by alpha particles during the full exposure period. This damage is subsequently revealed by processing as permanent tracks.

(d) Electret detectors

An electret is a material such as some types of aluminized Teflon which when charged will generally retain the charge and associated electric potential for a period of a year or longer. Such charged electrets have been used as electrostatic collectors of charged radon decay products from the air which can then be measured by a scintillation detector, surface barrier detector or other alpha particle detector (Chittaporn et al., 1981). This approach has not been used extensively.

A very reliable application of electrets as radon detectors is, however, commercially available (Kotrappa, 1988). Essentially it consists of a small hollow plastic container inside of which is mounted a charged electret. When air containing radon is admitted to the inner volume of the container the ionization produced, mainly by alpha particles from Radon, Po-218 and Po-214, will cause the electret to be discharged at a rate proportional to the radon concentration. From measurements of the potential of the electret before and after exposure to the air containing radon the mean radon concentration may be determined by means of calibration information for the system. This type of electret-based radon detector has the advantage of being relatively inexpensive, is rechargeable and may be used a number of times between recharges. It also has the advantage that the user of the system can make the radon determination thus obviating the need to send the electret to a processing laboratory. Some disadvantages do, however, exist associated with the presence of humidity and of ionising sources other than radon. These factors may influence the performance of electret-based detectors.

(e) Electronic monitors

A range of electronic detectors are available in which the common feature is the detection of alpha particles from radon and its decay products by surface barrier or similar solid state detectors and associated electronics (Simon and Schell, 1990). These devices are either mains and/or battery operated. In their simplest form radon gas enters a small detecting volume within the instrument by free diffusion through a porous barrier and the mean radon concentration during a measuring period of some hours may be read directly by the user from a panel display. Variable measuring cycle periods can be set for such devices and usually range from 4 to 24 h with the facility to store the data for up to 100 such intervals. Thus many devices of this type can be used as continuous monitors for periods as long as three months when set on a 24-hour cycle and can give more detailed information on the radon temporal variation in a building if set on shorter cycles. In some versions sensitivity is increased by the use of electrostatic deposition of charged radon decay products either directly onto the detector or onto adjacent collecting surfaces. More sophisti-

TABLE 27.6

Most common modes of operation of radon detectors

Detector	Grab	Integrating	Continuous	Active	Passive
Scintillation cell	Yes	–	Yes	Yes	Yes
Alpha track	–	Yes	–	–	Yes
Charcoal detectors	–	Yes	–	–	Yes
Electret	–	Yes	–	–	Yes
Electronic	Yes	–	Yes	Yes	–

cated versions of electronic detectors are available in which air is drawn by a pump through a filter mounted in front of the detector. The concentrations of the individual radon decay products are determined by gross alpha counting procedures or even by alpha spectroscopy. Direct printout of data and the facility to transfer data to a computer are also available. These devices are expensive and therefore more useful for detailed investigations in a small number of buildings rather than for use in large surveys. Simpler hand held electronic monitors of radon may be obtained for use in radon diagnostic studies of a building before and after remediation. They are usually equipped with a small battery operated pump and can be used to identify the routes of radon entry into a building.

Concluding comments

There is a wide and somewhat confusing array of radon detectors which are commercially available. Each has its advantages and disadvantages in terms of technical performance, price, etc. Some of these points have been indicated above. For indoor air quality investigations involving radon surveys or assessment of long-term average radon levels in a building passive detectors such as alpha track or electret versions are best suited. Where detailed indoor radon diagnostics of a building are required for pre- or post-remedial work passive detection methods should be complimented by continuous type devices which ideally should have readout facilities. Table 27.6 summarises the most common modes of operation of the various types of radon detectors described above.

In terms of cost effectiveness of the various techniques presented here it is not possible to make realistic comparisons as each application will be unique. It should be noted that an individual measurement of radon using a commercial passive detector, such as alpha track or charcoal types may be purchased at a cost of between US$ 10 and US$ 20. On the other hand, a state-of-the-art

electronic radon detector complete with readout facilities, memory and the ability to make alpha spectroscopic measurements of radon decay products may cost US$ 6000. This high capital cost may in some applications, such as remediation work, be acceptable. In large-scale survey work the use of passive detectors will usually be more cost effective.

It cannot be over-emphasised that accurate calibration of radon detectors is essential for good quality investigations. Experience, unfortunately, has shown that radon sensitivities quoted by manufacturers for some instruments are not always applicable under field conditions. Both in the European Communities (European Community) and in North America there are facilities at some national laboratories where independent calibration of instruments may take place. Standard radium sources for producing precise amounts of radon are also available and recently in the U.K. standard amounts of radon gas in glass vials have become available (NPL, 1992). With a half-life of 3.8 days these recently available radon standards can only be used for a short period of time. It is worth noting that the Commission of the European Communities (CEC) has for, a number of years, at regular intervals organised intercalibrations of radon detectors as part of its ongoing Radiation Protection Research Programme (Miles et al., 1984; Miles and Olast, 1990).

27.4 ENVIRONMENTAL TOBACCO SMOKE

Exposure to environmental tobacco smoke (ETS) has been measured by various tracers: acrolein, aromatic hydrocarbons, CO, nicotine, oxides of nitrogen, nitrosamines, and inhalable particles. Researchers usually monitor one compound in ETS because it is neither practical nor possible to monitor the full range of compounds. The National Academy of Sciences notes that optimum tracers for ETS should have the following characteristics:

- Unique (or nearly so) to ETS, so that there is minimal contribution from other sources.
- Detectable at low concentrations.
- Similar emission rates among various tobacco products, and
- Consistent ratio between the individual contaminant of interests and the composite pollutant, ETS, under a range of environmental conditions.

No single measure of ETS meets all these criteria, nor does the scientific community accept one measure as representing ETS exposure. The scientific community also does not agree on the specific components of ETS which may be responsible for the adverse health effects attributed to ETS.

However, nicotine is unique to ETS, and its measurement technique has been recently improved. Even under conditions of low smoking rates, easily

measurable increases in respirable particulate matter (RPM) have been recorded above background levels (Repace and Lowery, 1980).

There are several well-known problems with using nicotine: (1) it is highly reactive, making both sampling and analysis difficult; (2) nicotine can be present in both the particulate and the vapour phases; and (3) particulate or vapour phase nicotine can be re-emitted from surfaces on which it has deposited. Although nicotine is a marker for ETS exposure, the relationship between exposure to nicotine and to other components of ETS which may be of health consequence has not been established. This latter problem affects the estimation of health effects of ETS based on nicotine exposure.

Two approaches are described below for the determination of nicotine in indoor air, one for vapour phase and one for total nicotine. The methods which measure only vapour phase nicotine use a solid sorbent trap. Total nicotine is measured using a treated filter media.

Two different solid sorbents have been used to collect vapour phase nicotine Tenax GC™ and XAD-2. The Tenax GC™ method (Thompson et al., 1989) uses a packed tube of 200 mg of Tenax GC™ and personal sampling pump capable of drawing 1.7 l/min.

Thompson et al. (1989) report breakthrough volumes of 20–45 l at concentrations of 70–250 $\mu g/m^3$. Breakthrough volumes (see also Section 27.2.2) should be determined at the concentrations which are anticipated in the particular study. Determining breakthrough is done most easily by using a backup section of Tenax GC™ which can be analyzed separately and varying the sampling time. Determination of breakthrough volumes will define appropriate sampling times.

Both the sampling tube and the Tenax GC™ must be carefully cleaned and conditioned prior to use. Nicotine is thermally desorbed from the Tenax GC™ and analyzed by GC with a nitrogen phosphorus detector (NPD). The limit of detection is 0.07 $\mu g/m^3$ and limit of quantification is 0.17 $\mu g/m^3$ for this method.

A second type of trapping media used to collect nicotine vapour is XAD-2. The NIOSH method for nicotine uses this sorbent, but the sampling and analytical procedures have a limit of detection of 300 $\mu g/m^3$. This is unacceptably high for non-occupational environments. ASTM Committee D22.05 is developing a method which uses XAD-2, and is able to achieve a much lower limit of detection.

The proposed ASTM method uses a 7 cm glass tube containing two sections of XAD-2 separated by a glass wool plug. The front section contains 80 mg and the backup section contains 40 mg of XAD-2. The recommended sample flow rate is 1 l/min. Nicotine is desorbed from the XAD-2 using a solvent of ethyl acetate and triethylamine. Analysis is by GC/NPD. The limits of detection and quantification are 0.17 $\mu g/m^3$ and 1.7 $\mu g/m^3$, respectively, for a 1-h sample. An

8-h sample can reduce these values by an order of magnitude.

The capacity of the XAD-2 tube is approximately 300 μg of nicotine. Assuming a 1 l/min sampling rate and an 8-h duration, the capacity of the tube would be exceeded at concentrations of approximately 625 $\mu g/m^3$ (for an 8-h period).

Total nicotine (vapour and particulate phases) can be collected using a treated filter method as reported by Hammond et al. (1987). Two filters, 37 mm Teflon-coated glass fibre, are assembled in series in a personal sampling cassette. The first collects total or size fractionated particles. The second filter is pretreated with sodium bisulfate and collects vapour phase nicotine. This configuration collects nicotine which is present in the vapour form in the air and nicotine which can volatilize from particles collected on the front filter. Samples are collected using a personal sampling pump at a flow rate of 1.7 l/min. An advantage of this configuration is that respirable particle and nicotine concentrations are measured simultaneously with the same sampling apparatus.

A solvent extraction process is used to extract the nicotine from the filters and analysis is performed by GC/NPD. Two different extraction procedures are used for the front and back filter. The front filter which contains the particulate matter is ultrasonically extracted with dichloromethane. As this solvent cannot be used with an NPD, a second extraction with heptane is done. Nicotine on the bisulfate treated filter is extracted with a series of solvents: ethanol, sodium hydroxide, and heptane. The treated filter collects nicotine efficiently: a breakthrough analysis in a chamber study indicated less than 1% of the nicotine on the first treated filter was present on the backup filter. Hammond et al. (1987) report limits of detection for a sampling flow rate of 1.7 l/min of 0.2 $\mu g/m^3$ for an 8-h sample and 2 $\mu g/m^3$ for a 1-h sample.

Hammond and Leaderer (1987) have modified the sodium bisulfate treated filter method described above from an active sampling technique to a passive one. The method is for vapour phase nicotine and is based on the diffusion of nicotine to a filter treated with sodium bisulfate. Approximately 90% of nicotine in aged ETS is in the vapour phase. The sampling filter cassette consists of a Nucleopore windscreen, sodium bisulfate treated filter and support pad. The filter media is a 37 mm Teflon-coated glass fibre. In a test chamber study, the passive and active sampling methods were compared. The lowest concentration measured accurately was 16 $\mu g/m^3$ over a 5-h period. Samples were also collected over a one-week period and under variable concentrations. There appeared to be a good correlation between the active and passive techniques which indicated that the nicotine was not off-gassing from the treated filter. The analytical technique for this method is the same as that described for the active method.

An analytical issue common to all of the above methods is that nicotine will

adsorb to the glassware used in the collection and extraction procedures. Treating the glassware and the extraction solutions with ammonia suppresses the adsorption of nicotine to glass walls.

Unfortunately, there appears to be little information available on the intercomparison of the precision and accuracy among the various methods.

Biomarkers of ETS exposure

Biomarkers of exposure are actually a measure of dose or uptake and hence an indicator that an exposure has taken place. Biomarkers, within the context of assessing exposure to air contaminants, refer to cellular, biochemical, or molecular measures that are obtained from biological media such as human tissues, cells or fluids and are indicative of human exposure to air contaminants (Collier et al., 1990; Goldstein et al., 1987). The relation between the biomarker and exposure, however, is complex and varies as a function of several factors, including environmental factors and the uptake, distribution, metabolism, and site and mode of action of the compound or compounds of interest.

Ideally, a biomarker of exposure for a specific air contaminant should be chemically specific, have a long half-life in the body, be detectable in trace quantities with high precision, be measurable in samples easily collected by non invasive techniques, be inexpensive to assay, be either the agent that is associated with the effects or strongly associated with the agent of interest, and be quantitatively relatable to a previous exposure regimen. Ideal biomarkers for air contaminants, such as markers for complex mixtures, do not exist.

Numerous biomarkers have been proposed as indicators for ETS (e.g., thiocyanate, carboxyhaemoglobin, nicotine and cotinine, *N*-nitrosoproline, aromatic amines, protein or DNA adducts) (NRC, 1986; U.S. DHHS, 1986). Although these biomarkers demonstrate that an exposure has taken place, they may not be directly related to potential for development of the adverse effect under study (not the contaminant directly implicated in the effect of interest), they can show considerable variability from individual to individual, and they represent only fairly recent exposure (potentially inadequate for chronic outcomes). Furthermore, some of these markers may not be specific to ETS exposure (e.g., carboxyhaemoglobin), while others (e.g. thiocyanate) may not be sensitive enough for ETS exposures.

Nicotine and its metabolite, cotinine, in the saliva, blood, and urine are widely used as biomarkers of active smoking and exposure to ETS and are valuable in determining total or integrated short-term dose to ETS across all environments (NCR, 1986; U.S. DHHS, 1986). Nicotine and cotinine are specific to tobacco and are accurately measured by gas chromatography, radio immunoassay, or high-pressure liquid chromatography in concentrations

down to 1 ng/ml. Nicotine has a half-life typically of about 2 h in the blood and is metabolized to cotinine and excreted in the urine. The short half-life of nicotine makes it a better indicator of very recent exposures rather than a measure of integrated exposure.

Cotinine values in smokers and non-smokers measured in either the laboratory or field setting show considerable variability attributable to individual differences in the uptake distribution, metabolism, and elimination of nicotine. An additional issue that has to be considered in interpreting the field data is that exposure status is determined by respondent self-reporting. This can lead to a misclassification error, which tends to reduce the differences in cotinine levels measured in the ETS-exposed versus non-ETS-exposed groups and to increase the variability in the levels within any exposure category. Within the exposed group, this misclassification error could either increase or decrease the average cotinine levels measured.

It is important to recognize that nicotine and cotinine are actually proxy biomarkers. They may not be the active agents in eliciting the adverse effect under study but merely indicative of the level of passive smoke exposure. Using these measures to estimate cigarette equivalents or to determine equivalent active smoking exposure could result in over- or underestimating exposure to individual or classes of compounds that may be more directly related to the health or nuisance effect of concern. The use of different biomarker proxies (e.g. protein adducts) could result in estimates of much larger cigarette equivalent doses.

Nicotine and cotinine levels in ETS-exposed nonsmokers measured in laboratory and field studies have been used to estimate cigarette equivalent exposure and to equate ETS exposures with active smoker exposures (NCR, 1986; U.S. DHHS, 1986; Jarvis, 1989). On an equivalent cigarette basis, an upper-bound estimate of nicotine dose of 2.5 mg per day for passive smoke exposure has been proposed (Jarvis, 1989). This would translate into the equivalent of about one-fifth of a cigarette per day, or about 0.7% of the average smoker's dose of nicotine (cigarette equivalent dose of other toxins or carcinogens would be different, as described above).

Comparisons of cotinine values in ETS-exposed non-smokers with those measured in smokers ranged from 0.1% to 2%. One analysis proposed that, on average, non-smokers' cotinine levels are 0.5% to 0.7% of those found in cigarette smokers (Jarvis, 1989). It should be noted that these estimations are based on a number of assumptions that may not hold (e.g., the half-life of nicotine and cotinine in smokers and non-smokers is the same).

One of the protein adducts that has been used as a biomarker of active and passive smoking is the 4-aminobiphenyl adduct of haemoglobin (4-ABP-Hb). One advantage of haemoglobin adducts is that their half-life is quite long and

they will persist through the life of a red blood cell which is approximately 120 days. Therefore, levels of 4-ABP-Hb reflect exposures over the past several weeks, rather than the day or two of exposure-integration reflected by cotinine measurements.

Tobacco smoke is the primary environmental source of 4 aminobiphenyl (its use in the dye industry was discontinued decades ago), and smokers have between five and eight times as much 4-ABP-Hb adducts as non-smokers (Hammond et al., 1990; Perera et al., 1987; Maclure et al., 1989). That non-smokers appear to have approximately 10–20% the adduct level as smokers may at first appear to be contradictory to the urinary cotinine ratios of about 1%, but in fact both results are quite consistent with our knowledge of the emissions of various contaminants in MS and SS. Approximately twice as much nicotine is emitted in SS as in MS, but about 31 times as much 4-ABP is emitted in SS as in MS. Thus, compared to MS, SS is 15 times more enriched in 4-ABP than in nicotine. The ratio of biomarkers in those exposed to ETS compared to smokers is 15 times greater for the biomarker 4-ABP-Hb than for the biomarker cotinine, a metabolite of nicotine.

The above discussion indicates that the "cigarette equivalent" dose of those exposed to ETS varies with the compound, so that a passive smoker may receive 1% as much nicotine as an active smoker but 15% as much 4-ABP. These commentaries on the data are preliminary and warrant further investigation, but they do suggest the importance of careful interpretation of biomarkers in estimating dose.

27.5 BIOLOGICAL AGENTS

27.5.1 House dust mites

Although an International Workshop (Platts-Mills and de Weck, 1988) could not recommend airborne sampling as being better than dust sampling for primary measurement of mite infestation, asthma is triggered by inhaled antigen and therefore measurement of airborne levels is likely to be relevant in assessing exposure. There is no standard method for air-sampling for mite allergens, but cascade impactors or high-volume samplers have been used to collect airborne mite allergens on membrane filters (Price et al., 1990). However, sampling periods of 2–24 h are required, whereas exposure to short periods of high mite allergen concentrations is clinically (more) important (Platts-Mills and de Weck, 1988). The reasons for the long sampling times are that mites themselves are not seen in air samples; most allergen is bound to their faecal pellets (fragments of which only become airborne as a result of

disturbance) and consequently very little is associated with particles that are airborne for more than a few minutes. Therefore, it is recommended that dust samples should also be taken (ECA, 1993).

The procedures recommended by a 1987 International Workshop (Platts-Mills and de Weck, 1988) for collection of dust samples have been endorsed by a working group of the EC Collaborative Action "IAQ and Its Impact on Man" (ECA, 1993). Vacuum cleaners can be used to collect settled dust if equipped with a disposable paper collection bag or a special attachment to collect dust on a paper filter. The preferred sites for sampling are:

(a) the entire upper surface of the mattress, which should be sampled for 2 min, and other dust collecting locations in the bed;

(b) an area of 1–3 m^2 of the floor in the living-room and the bedroom (immediately underneath and beside the bed).

Samples can also be taken from upholstered furniture. Less effective and non-standard techniques include shaking blankets in a plastic bag and scraping flat surfaces above floor level with a piece of firm card.

After separation from dust samples by flotation or suspension, numbers and species of mites, and whether they are alive, dead, larval or adult, can be determined under a microscope. However, this is time-consuming and demands considerable training and skill. It has the further drawback of not reflecting mite allergen levels, since faecal pellets, which are the major source of allergens, and disintegrated mite bodies are not quantified (Platts-Mills and de Weck, 1988).

Several immunochemical techniques (Kemeny, 1987) can be used for analysis of allergens in buffered saline or 50% glycerin extracts of 100 mg dust samples (Platts-Mills and de Weck, 1988). Inhibition radioallergosorbent testing (RAST inhibition) measures "relevant" antigenic determinations that have elicited a response in allergic subjects and provides a good estimate of the relative potency of different allergen extracts, but it cannot be used for absolute quantitation of allergen levels. Because human IgE is used, results are difficult to reproduce over an extended period of time and the sensitivity is low, the method is not recommended for routine purposes (Platts-Mills and Chapman, 1987). Other assays, including radial immunodiffusion, rocket immunoelectrophoresis (RIE), enzyme-linked immunosorbent assay (ELISA) and inhibition radioimmunoassay (RIA) measure individual mite allergens. Sandwich radio- or enzyme immunoassays employ either rabbit polyclonal or mouse monoclonal antibody for capture, and affinity-purified antibody or a second monoclonal antibody for detection. These highly specific assays are more sensitive than RAST inhibition, and those using monoclonal antibodies in particular have the great advantage of long-term reproducibility. The detection limits of these methods are approximately 10–20 ng allergen/g dust,

depending on the allergens. As they can be automated, they are suitable for large-scale surveys, but require trained laboratory technicians and sophisticated equipment (Platts-Mills and Chapman, 1987).

An indirect assessment of faecal pellets can be obtained by measuring guanine, a nitrogenous excretory product in the faeces of mites and other arachnids. The intensity of its colour reaction with an azo-compound can be measured spectrophotometrically, or in a semi-quantitative manner using a commercial test-kit. Although they are simple and economical, these methods do not identify the mite species present, and the test-kit appears to be less sensitive and also less reliable than ELISA (ECA, 1993).

Recommended approach to investigations

For case studies in homes, it is recommended (ECA, 1993) that dust samples be collected from the upper mattress surface and the bedroom and living-room floors. If information on the prevalence of different mite species is required, mite counts must be made, but if information about the levels of mite allergen(s) is required an immunochemical assay, preferably ELISA for house dust mite allergen Der p 1, should be employed. For epidemiological investigations it is recommended that dust samples are collected as for case studies and analyzed by an immunochemical assay (preferably ELISA) for at least Der p 1.

Information on the characteristics of the building and the behaviour of the occupants, as far as they might influence the concentration of mite allergens, should be gathered. The EC Working Group has compiled a checklist of points applicable not only in investigations of mites, but also of dander, fungi and bacteria (ECA, 1993). As house dust mites seem rarely to be found in non-industrial workplaces, measurement of house dust mite allergens in these environments is not considered to be necessary. Low levels of house dust mite allergens can however be found in dust deposits in schools, day-care centres and hotels, so that dust sampling and determination of concentrations of allergens by the methods noted above might be relevant in these environments.

27.5.2 Dander from furred animals

The methods for sampling air- and dust-borne mites and/or their allergens can also be used for sampling pet-derived allergens.

Immunochemical assays are used for the detection of the allergens (Lowenstein et al., 1986). A variety of methods for extraction of dust have been described, but no comparative studies have been published and no recommendation can be made, although extraction in ammonium bicarbonate buffer is convenient. The assay techniques include counter-current immunoelectropho-

resis (CCIE), RIE and ELISA, all employing species-specific rabbit antibodies raised against the allergens. RAST-inhibition is also used, but for the same reasons as mentioned in connection with mite allergens is not recommended. Although further standardisation of the relevant allergens is still needed, a standardised ELISA technique for the cat allergen Fel d 1 is currently available, and in the near future a standardised ELISA technique, more sensitive and specific than the CCIE currently used, will also become available for the dog allergen Can f 1 (ECA, 1993).

Recommended approach to investigations

For both case and epidemiological studies in homes, dust samples should be collected from the living room and bedroom floors in the same way as for mites. Because the allergens can also be found in mattress dust, sampling of the mattress surface should be carried out in epidemiological studies and might also be relevant in case studies. It is recommended (ECA, 1993) that the samples should be analysed for Fel d 1 and Can f 1 by ELISA. Allergens from furred animals are rarely detected in office buildings, schools and day-care centres, since these animals are not regularly kept in these environments. However, if pets are present, e.g. in school classrooms, measurement of pet derived allergens might be relevant. It should also be remembered that allergens (mainly cat, dog and horse) deposited on clothing, can be brought into such buildings from the home. For investigations of these buildings, the same sampling and analytical methods should be adopted as for private homes.

27.5.3 Fungi

As far as health is concerned, both viable and non-viable fungal particles are important and therefore both should be sampled. However, many airborne spores will not grow in culture and are not microscopically distinctive, so that they cannot be identified using currently available methods. In sampling for viable particles, reproducibility is low and variability is high (Verhoeff et al., 1990), human activity at the location having a profound effect on counts (Hunter et al., 1988). In consequence, air sampling cannot satisfactorily be used to assess exposure to fungi in indoor air environments.

Some sampling techniques employed in aerobiological studies give total counts of all airborne particles, viable and non-viable, whereas others only give counts of viable fungal particles, i.e. propagules or colony forming units (CFU). Samplers do not collect all particles with equal efficiency; they differ in cut-off point, i.e. the particle size above which 50% or more of the particles are collected. At present, there is no standardised method for the sampling of airborne fungi, and therefore a range of samplers which are commonly used is

TABLE 27.7

Methods of sampling for airborne bacteria and fungi (after ECA, 1993)

Method, with examples	Sampling rate and time	Remarks
Total count, non-volumetric		
– gravity slide	–	semi-quantitative, over-representation of larger particles
Total count, non-volumetric		
– Burkard trap	10 l/min; 7 days	cut-off unknown
– rotating arm impactors	47 l/min; 15–60 sec	cut-off unknown
– filter methods[a]	1–4 l/min; ca. 4 h	
Viable count, non-volumetric		
– open Petri dish	–	semi-quantitative, over-representation of larger particles
Viable count, non volumetric		
– Andersen 6-stage impactor	28.3 l/min; 1–30 min	cut-off 0.65 μm[b]
– Andersen 2-stage impactor	28.3 l/min; 1–30 min	cut-off 0.65 μm[b]
– Andersen 1-stage impactor (N6)	28.3 l/min; 1–30 min	cut-off 0.65 μm[b]
– Surface Air System impactor (RCS)	180 l/min; 20 sec–6 min	cut-off 1.9 μm[c]
– Reuter Centrifugal impactor (RCS)	ca. 40 l/min; 30 sec–8 min	cut-off 3.8 μm[d]
– Reuter Centrifugal Plus impactor (RCS Plus)	ca. 50 l/min; 30 sec–8 min	cut-off unknown
– slit samplers	10–30 l/min	cut-off 0.7 μm[e]
– liquid impingers	12.5 l/min	cut-off 0.3 μm[e]

[a] May also be used for viable counts (see text).
[b] Andersen (1958).
[c] Lach (1985).
[d] Macher and First (1983).
[e] Nevalainen et al. (1992).

presented in Table 27.7.

Methods that assess the total number of particles, e.g. the Burkard samplers which collect onto adhesive-coated glass slides or tape, have the major disadvantage that only fungi with distinctive spores can be identified. However, where collection is on sterile membrane filters, e.g. in the CAMNEA method of Palmgren et al. (1986), as well as being used for total counts, washings from the filters can be used to obtain viable counts by plating out on agar medium.

Because of differences in cut-off point (Table 27.7), sampling time and sampling volume (Verhoeff et al., 1990), counts of viable fungal particles and the species isolated depend on the sampler used. Provided its characteristics and limitations are fully understood by the operator and it is correctly operated, any one of the available sampling devices can be used for internal comparisons. Standardisation is needed, however, if valid comparison is to be made between different investigations. Most published data on viable fungal particles in indoor air have come from investigations employing the Andersen 6-stage Impactor. In such a sampler, particles impact directly onto agar medium, but in correctly operated liquid impingers airborne clusters of spores may be broken into smaller units or individual spores. Consequently, higher counts may be obtained than with an impactor. Settle plates, or open Petri dishes, are generally considered to give the least reliable results, but recently highly significant correlations with 1-stage (N6) Andersen impactor counts have been obtained (Verhoeff et al., 1990, 1992). Although the number of species isolated is significantly lower on settle plates, they can be used to obtain semiquantitative information (Verhoeff et al., 1992).

The counts of viable fungi obtained depend on the collection medium used, and it has to be stressed that no single medium will enable the entire range of airborne fungi to be isolated. Sabouraud agar and other nutrient-rich media which favour vegetative growth of fungi are not suitable. They allow many different fungi to grow, but the rapidly growing fungi swiftly overgrow slower growing fungi on the plates, making counting difficult and inaccurate. One such medium is the malt extract agar (MEA) recommended by ACGIH (1989). Although it is widely used, it has not been endorsed as an isolation medium by the EC working group (ECA, 1993). A more satisfactory medium is obtained by omitting the dextrose and peptone from the MEA (Hunter et al., 1988), to which antibiotics such as penicillin and streptomycin, or chloramphenicol, should be added to prevent bacterial growth. V8 vegetable juice agar, which favours sporulation, can be also be used (Gravesen, 1972). Neither the ACGIH-recommended MEA nor the other media mentioned will truly reflect members of xerophilic fungi, some of which may not appear on these media at all. Recently, dichloran: 18% glycerol agar (DG18), a medium which restricts growth and facilitates counting (Hocking and Pitt, 1980), has been found to be very satisfactory for isolating xerophiles and useful for many other fungi (Verhoeff et al., 1990), although some hydrophilic fungi may not be detected on it.

Because of the problems associated with air-sampling, it has been suggested that settled house dust should be sampled for viable fungi, as dust may be a depository for and generator of airborne fungal particles. It can be sampled in exactly the same way as for mites. Samples can be stored at room temperature, but the analysis should be performed within a few days. House

dust samples can be plated by spreading duplicate representative 30 mg portions of dust directly onto the same media as are used for air sampling, or by suspending 100 mg portions in peptone or sucrose solution and spreading aliquots of dilution prepared from this over agar plates (COST 613, 1993).

Surfaces with or without visible fungal growth can be sampled to obtain qualitative information about the presence of fungi which may generate spore aerosols or may have been deposited from such aerosols. The spores and mycelium on Sellotape (or Scotch tape) pressed gently on the surface can be stained in lactophenol cotton blue and examined under the optical microscope. To assess what viable organisms are present, swabs can be swept over the surface and either streaked over an agar plate, or washed in peptone and the washings and/or dilution plated out. Alternatively, contact or pressure (Rodac) plates containing appropriate agar medium can be pressed gently onto the surface, and after incubation the resulting colonies can be transferred onto appropriate medium for identification (ECA, 1993).

To make total counts of spores and other particles, collection tapes or slides from samplers are examined microscopically under visible light. In the CAMNEA method, micro-organisms in the washings from the collection filter are fluorochrome-stained *in situ* on a second membrane, through which the washings are passed, and examined by direct epifluorescence microscopy (Palmgren et al., 1986). Recently, it has been shown that it is possible to count fungi on membrane filters by examination under the scanning electron microscope (Heikkilä et al., 1988).

Agar plates used to collect fungi from air, dust or surfaces are usually incubated at 25°C for at least 4 days. The colonies are then counted by the naked eye, and expressed as cafs/m^3 air or CFU/g dust as appropriate. If the colonial characteristics seen by the naked eye or the microscopical characteristics of sporing structures and spores on the original isolation plates do not enable identification to species, spores and/or mycelium from the colonies are transferred onto appropriate media for further incubation and identification to species level. The original plates may also be re-incubated for several days to allow further development of colonial and sporing characteristics which assist in identification.

It should perhaps be noted here that methods are likely to change over the next few years. There will still usually be a need to know which viable fungi are present, but new identification methods are appearing and continually being refined. For example, Zwick et al. (1991) have used an immunofluorescent test in conjunction with a Burkard personal air sampler to explore the homes of patients for airborne mould spores which cause their extrinsic allergic alveolitis, other immunological identification kits developed for use in the food industry may come to be used in this field. It is also possible that

analysis for ergosterol or β-glucan fungals spore and hyphae may replace total count methods. Glucan has been implicated in increased reporting of symptoms by occupants of buildings where IAQ complaints had been made (Rylander et al., 1991).

Recommended approach to investigations

For case studies in homes, it is recommended (ECA, 1993) that factors of possible relevance, e.g. mould odour, visible dampness, leakage, penetration, rising damp, condensation, mould patches and the HVAC system if present, be assessed in a walk through. Where mould growth (and associated dampness) is observed and remedial action is clearly indicated, it might appear that sampling of fungi is not necessary, but it has to be remembered that there may be other hidden sources of fungi which could be involved.

Where fungi are under investigation, it is recommended that surfaces should be investigated as indicated above, and that the air should be sampled to determine the range and preponderance of different species in the indoor air spora. Parallel duplicate air samples should be taken under normal living conditions in at least the living room and one bedroom, and parallel outdoor samples must also be taken for comparative purposes.

Sampling for fungi may be of particular value, and is therefore recommended (ECA, 1993), in the following instances:

(1) If a HVAC system is present and is a possible source of bioaerosols, samples should be taken at the air inlet from outdoors and at egress to the room, both when the ventilation system is operative and not.

(2) If damp filters in the ventilation system are a possible source of fungi, again samples should be taken at the inlet from outdoors and at egress to the room, when the ventilation system is in use and when not.

(3) If no mould growth is visible although an odour indicates the presence of fungi, samples should be taken at those locations where the odour is detected and also in a non-odorous room.

(4) If a specific organism has been indicated as a cause of observed allergy among occupants, samples should be taken to assess the prevalence of that organism in the indoor environment.

(5) If extrinsic allergic alveolitis (hypersensitivity pneumonitis), rhinitis, asthma or other recurrent respiratory problems are associated with the indoor environment, samples should be taken to assess its mycoflora.

(6) If the efficacy of remedial action for dampness or mould growth is to be assessed, sampling should be carried out before and after the action has been taken.

Mycological investigation should also be contemplated when no physical and/or chemical factors can be found to explain complaints. Additional infor-

mation about building characteristics and the behaviour of the occupants (ECA, 1993), which are the principal influences on the presence of fungi in the homes, should be recorded.

In view of the problems arising with air sampling, dust sampling should be considered for large-scale epidemiological studies of the relationship between fungi and health effects. Samples should be collected as for mites from the mattress and the bedroom and the living room floors. The type of flooring as well as the material of the mattress should be recorded.

Since each problem demands its own sampling strategy, no general sampling strategy can be given for large buildings. However, in order to evaluate the presence of fungi in buildings where IAQ problems are possibly related to central or local HVAC systems (ECA, 1989a), the following approach is recommended:

(1) Conduct a walk-through for visual and olfactory assessment of factors of possible relevance to microbiological problems.

(2) Determine whether there is recirculation of air in the ventilation system, since dust may accumulate in recirculation systems, making microbial growth more likely.

(3) Determine whether there is condensation in the system, as this is likely to promote microbial growth.

(4) Sample deposited dust and/or visible fungal growth, which are sources of airborne organisms.

(5) Pay particular attention to the inspection of humidifiers, and collect water and surface samples for evaluation.

(6) If sources of micro-organisms are found within the system, sample air and/or surfaces in rooms supplied by the system to determine whether the organisms are spreading from the system.

27.5.4 Bacteria

Most of the samplers used for viable fungi can also be used for bacteria, but, unless collecting into liquid (Table 27.6), sampling times should be relatively short, because many bacteria are rather susceptible to the effects of desiccation. For general purposes, tryptone soya agar (TSA) or tryptone yeast glucose agar (TYGA), containing an antimycotic such as cycloheximide to prevent fungal growth can be used as a collection medium. For specific groups of bacteria, selective media can be employed, e.g. Pseudomonas agar base supplemented with cetrimide and antibiotics for isolation of *Pseudomonas aeruginosa* or half-strength nutrient agar for thermophilic actinomycetes, but highly selective media may inhibit sublethally injured bacteria. The liquid used for trapping bacteria in a liquid impinger, e.g. 0.1% peptone, should be neither

unfavourable to them nor sufficiently nutritive to support their rapid multiplication. Ideally, aliquots of the collection liquid should be plated out on agar medium or added to enrichment medium within 1 h of sampling. If not, the liquid should be kept at 3–5°C for no longer than 24 h.

The sampling of dust for bacteria can be conducted in the same way as for house dust mites and their allergens and surfaces may be sampled using swabs or contact plates as described for fungi. Because of the greater number of species likely to be isolated and improvement in resuscitation of stressed bacteria (Hyvärinen et al., 1991), it is recommended that plates should be incubated at 2–25°C, rather than 37°C, and examined daily for several days. For thermophilic actinomycetes, plates should be incubated at 55°C.

Recommended approach to investigations

For case studies in homes, the same approach should be adopted as for fungi. Where an HVAC system is present, samples should be taken from surfaces, water, sludge, slime and sediments within the humidifier to obtain qualitative information about the bacteria present. For large-scale epidemiological studies on the relationship between the exposure to bacteria and health effects, a standardised sampling protocol should be used throughout. To evaluate the presence of bacteria in other non-industrial environments with central or local HVAC systems the same approach can be adopted as for fungi.

Legionella

Routine sampling of *Legionella* in indoor air or water is not recommended; it is more important that legionellosis is rapidly and correctly diagnosed (ECA, 1993). Sampling is however useful in helping to explain outbreaks of legionellosis. It can also be used to monitor the efficacy of maintenance practices and control measures taken to guarantee the safe use of hot water supplies and humidified ventilation systems.

For any investigation of *Legionella* in water services, it is essential to have a schematic diagram of the services. The entire route of the water supply should be traced and the condition of the pipes, the jointing methods used, the presence of lagging, sources of heat and the standard of protection afforded to cisterns noted. Attention should also be given to disconnected fittings, "dead-ends" and cross-connections with other services (Dennis, 1990).

Samples of water should be collected from the incoming supply; cisterns and calorifiers; an outlet close to, but downstream of, each system and calorifier; the distant point of each service and the water entering and leaving any fitting under particular suspicion (Dennis, 1990). In addition, swab samples should be taken from shower heads, pipes and taps (faucets), and samples of

any sludge, slime or sediments within water services or humidifiers should also be collected.

Samples can be stored at room temperature in the dark, but should be plated out within 48 h onto a semi-selective medium, BCYE agar, which is based on charcoal-yeast extract medium with the addition of various supplements (WHO, 1990).

Although it is not usual to sample indoor air for *Legionella,* Andersen samplers or liquid impingers have occasionally been used to collect 5–30 min samples (Seidel et al., 1987; Breiman et al., 1990). Distilled water or Page's saline is the recommended collection fluid in impingers (Dennis, 1990), and BCYE agar is used as isolation medium with both types of sampler.

PART VI — REFERENCES

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PART VII
Control of Indoor Air Quality and Climate

This part is concerned with the technical aspects of building construction and operation and the tools used by architects and engineers to control indoor air quality and climate. The basic criteria of building design and construction in order to obtain good indoor air quality are first presented and discussed. A chapter then deals with the heating, ventilation and air conditioning systems (HVAC), providing directions for a proper design and operation and for testing, balancing and maintaining HVAC systems.

General and specific rules for building operation and maintenance are then discussed together with ways of mitigating indoor air quality problems. Specific aspects of indoor air quality and climate control, namely moisture control, remedial and preventive measures to reduce indoor radon, combustion product control, air cleaning, ventilation, selection and evaluation of building materials and furnishing, and noise and lighting control, are dealt with in a final chapter.

Chapter 28 has been written by H. Levin; Chapters 29 and 30 by R. Axelrad. Chapter 31 has been prepared by M. Jantunen, with contributions from D. Walkinshaw, O. Seppänen, T. Lindvall, F. Bochicchio and J. McLaughlin.

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Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 28

Building Design for Good Indoor Air Quality

28.1 INTRODUCTION

The basic function of a building is to shelter occupants from outdoor elements and provide a healthy, comfortable environment for productive activity.

This statement is deceptive in its simplicity, because the definition of a "healthy, comfortable environment" evolves over time. For example, features such as artificial lighting and indoor plumbing were once considered luxuries. Accepted building design practices change in response to emerging expectations.

When most buildings relied on operable windows for ventilation, occupants faced a trade-off between tolerating odours or ventilating with untempered outdoor air. The ability to understand and choose between these relatively undesirable alternatives provided building occupants with a measure of control, while limiting their comfort expectations. The declining use of operable windows has reduced occupant control over ventilation. At the same time, increasingly sophisticated heating, ventilation, and air-conditioning designs have raised expectations. Building occupants have become less tolerant of fluctuating temperatures, unpleasant odours, or other departures from ideal conditions. People are coming to expect good indoor air quality (IAQ) as well as consistent thermal comfort, and they are holding designers, builders, owners, and operating staff responsible for meeting those expectations.

The causes of poor IAQ are complex; changes in building technologies, energy costs, and operator and occupant ignorance all contribute. In recent years, the sources and quantities of pollutants within buildings have proliferated, increasing the likelihood of problems. For good IAQ, air contaminants must be limited to concentrations that do not cause adverse reactions such as discomfort or health symptoms, while temperature, relative humidity, and air movement are maintained within the comfort range.

With an increasing frequency of IAQ-related complaints, control of indoor air quality is a rising priority for designers, builders, owners, and operating staff as well as building occupants. IAQ in the finished building can be significantly improved by conscientiously addressing IAQ concerns at all stages of the design process.

TABLE 28.1

Representative impacts of indoor air quality problems

Target of impact	Potential effects
Designers, builders, product manufacturers	Legal expenses, judgements and settlements Damage to reputation and sales
Building owners	Lost time and revenue Expense of repairs Legal expenses, judgements and settlements
Employers	Lost productivity and revenue Deteriorating institutional environment Workers compensation claims Damage to public image of reputation
Building occupants	Health effects, illness, lost work time Discomfort, dissatisfaction
Building equipment	Deterioration, material damage Malfunction from fouling Breakdown Destruction from electrical arcing
Building fabric	Deterioration Soiling Increased maintenance costs

28.1.1 The effects of poor indoor air quality

The costs of poor indoor air quality to the tenant include increased absenteeism and lower productivity linked to adverse health effects. Potential costs to the building designer and owner include strained relations between landlords and tenants (and between employers and employees), damage to reputation and liability expenses. Many insurance policies do not cover pollution-related personal or property damage. Soiling of surfaces and equipment and damage to appliances and office machines incur both aesthetic and economic costs (Weschler and Shields, 1991; Woods, 1989; EPA, 1989).

Table 28.1 summarizes the negative impacts of poor IAQ.

Occupant well-being and productivity are influenced by physical health and comfort, individual characteristics, and psychosocial factors. Because individuals differ in their tolerance for chemical contaminants and other stressors and in their requirements for thermal comfort, air that is considered acceptable by most building occupants may be unacceptable to a minority.

Exposure to poor IAQ can cause health disorders with effects that may be either short-term or permanent and that range in severity from discomfort or irritation to permanent disability or even death (Woods, 1989; EPA, 1989; Cone and Hodgson, 1989; Hodgson, 1988; Hodgson and Kreiss, 1984; Samet, et al., 1988; NRC, 1986). These effects are extensively dealt with in other parts of this book.

28.1.2 Factors affecting indoor air quality

Indoor air quality is produced by the interaction of many variables and processes. Some of these variables and processes are controlled or determined by the designer, some by the building operator, some by occupants, and some are external to the building itself. Numerous daily occurrences are involved, such as: turning building ventilation on and off, occupant activities (e.g., food preparation or graphic arts work that generate pollutants), and variations in outdoor air quality caused by rush hour traffic or exhaust plumes from nearby industrial facilities. This section will help an architect enhance the success of his or her designs by using these interdependent variables to promote good indoor air quality.

Table 28.2 lists the five major factors that interact to produce the indoor environment: the operation and maintenance of the building and HVAC system, the occupants of the building and their activities, the building contents, the outdoor environment, and the building fabric.

TABLE 28.2

Elements of a building that affect indoor air quality

Operation and maintenance of the building	Ventilation system operational routines and schedules; housekeeping and cleaning; equipment maintenance
Occupants of the building and their activities	Occupant activities; metabolism; personal hygiene
Building contents	Equipment; materials; furnishings; appliances
Outdoor environment	Climate; air quality; soil; water
Building fabric	Envelope; structure; floors and partitions

As Figure 28.1 illustrates, IAQ at a given moment and in a given location reflects the dynamic relationships of variables that fluctuate over time.

Some examples follow to show the interdependence of some factors.

Example 1: The influence of weather conditions (e.g., wind speed and direction) on indoor air quality is strongest when buildings are constructed with high infiltration and exfiltration rates. Although "tight buildings" are sometimes blamed for IAQ problems, natural ventilation through leakage sites is not a practical strategy for controlling indoor air quality. Infiltration can raise or lower indoor contaminant concentrations depending on outdoor air quality and on pressure relationships in the building. High infiltration rates also make it more difficult to control energy consumption and indoor temperatures and humidities.

Example 2: Building maintenance and housekeeping is performed during "off-hours" when the normal occupants are not present and ventilation systems are not operating. Floor waxes, furniture polishes, pesticides, equipment lubricants, and other maintenance and housekeeping supplies are applied. Vapours accumulate as these products dry, cure, and evaporate. Building ventilation is re-started just before occupants re-enter, and the odours are strong. Irritation of the eyes and of the mucous membranes in the nose and throat are felt by many occupants. These events are routine and demonstrate the importance of relating ventilation to the presence of pollutant sources.

Example 3: Increasing the volume of outdoor air supplied to a building tends to dilute indoor contaminants, depending on the distribution patterns of contaminants and of ventilation air. Increasing outdoor airflow also tends to increase the amount of energy required to maintain comfortable temperatures and humidities, but can reduce energy consumption when outdoor conditions allow untreated outdoor air to be used for "economizer" cooling.

It is important to know what pollutant sources will be in a building so that effective control can be planned. Cooking, floor waxing, furniture polishing, photocopying, and a host of other common activities create local air contaminants. Emissions of volatile organic compounds from building materials, equipment, appliances, and furnishings continue even when a building is vacant. The dispersal of these contaminants depends on conditions (e.g., occupant activities, air circulation patterns, building contents) that vary among buildings and within a single building (Tucker, 1988).

28.1.3 The economics of indoor air quality

Economic forces have produced a trend toward frugality in building construction and operation. There are pressures to use less material and less expensive materials, to build and occupy more rapidly, and to spend less on operation and maintenance.

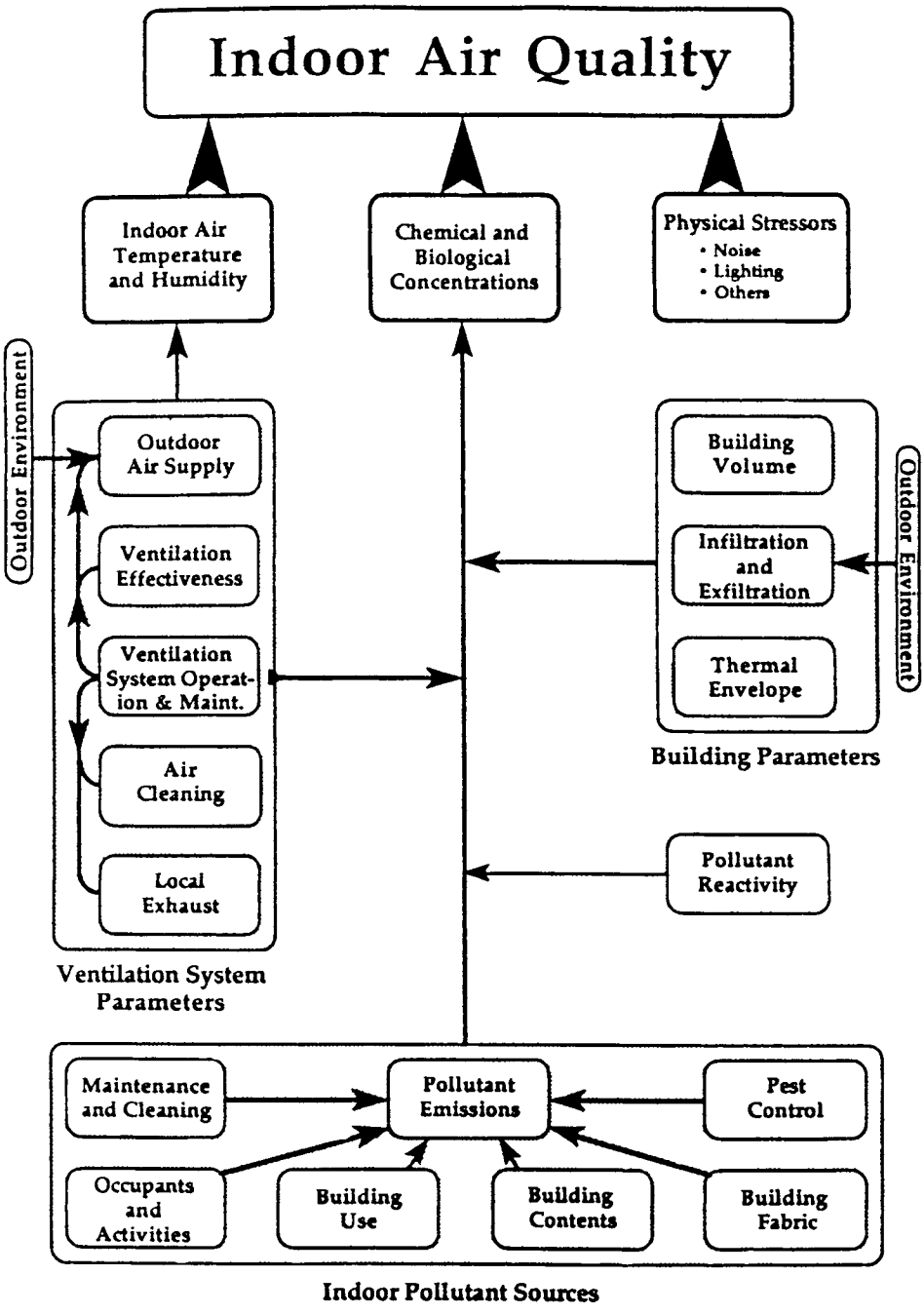


Fig. 28.1: Relationships of IAQ variables.

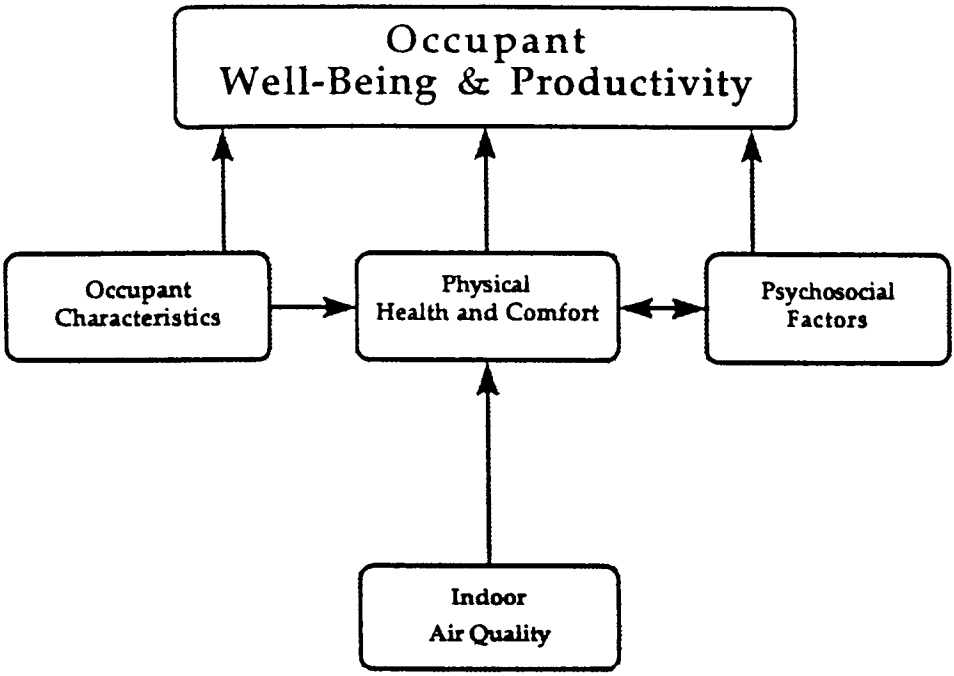


Fig. 28.2: Determinants of occupant well-being.

Attempts to minimize initial costs can lead to higher life-cycle costs. IAQ experts and many others believe that the benefits of a healthier building environment are likely to offset any additional costs that might be required to provide good indoor air quality. Reducing indoor air pollution will improve occupant health, reduce medical costs and lost work time, increase workplace productivity, and, in schools, improve educational opportunities. Figure 28.2 illustrates the cost of IAQ-related design considerations in relation to the overall cost of operating a business.

Since the oil embargo of 1973, efforts to improve the energy efficiency of buildings have resulted in conservation measures which often decrease outdoor air supply rates. Although these practices lower HVAC operating costs, they can have a negative impact on occupant health and comfort (Levin, 1981).

The reduced ventilation associated with energy conservation is not the sole cause of indoor air quality problems. Buildings have become more complex as technology has developed. Changes in design, construction, furnishings, operation, and maintenance have all contributed to IAQ problems. New products and processes have introduced new contaminant sources. For example, individual offices with walls and doors have largely given way to cubicle offices with fabric-covered partitions, because open office planning results in lower

construction costs and more flexibility to re-arrange work stations. However, the fabrics, frame, and insulation materials that make up the partitions sometimes emit odorous or irritating volatile organic chemicals (VOCs) and fibres (Levin, 1989).

Economic pressures (particularly in private, speculative buildings) often lead to building occupancy before construction is fully completed. Occupants can be exposed to large concentrations of vapours, fumes, and dusts from construction at a time when building ventilation may not yet be adjusted properly. Even if the ventilation system has been properly adjusted to serve a fully occupied building, it can become a delivery mechanism, distributing contaminants from the construction area to the occupied area. Special ventilation is required to prevent IAQ problems if construction activity takes place concurrent with normal occupancy (ASHRAE, Guideline 1-1989).

Some authorities have recommended using a process known as HVAC system commissioning to ensure the proper performance of the ventilation system according to the building design. Some architects and engineers report that this process can provide considerable savings to the building owner by reducing the costs associated with poorly functioning ventilation systems. The cost of contractor call-backs and disrupted building occupants is likely to be greater than the cost of proper commissioning.

Good IAQ saves money by avoiding problems such as reduced productivity, absenteeism, damage to reputation, liability expenses, and shortened life of equipment and furnishings. These savings are difficult to quantify for use in cost/benefit analyses. However, experts agree that investing in good IAQ requires only minor increases in design and construction cost, an investment that is easily recovered over the life cycle of the building and equipment (Levin, 1991; ASHRAE, 1989b).

28.1.4 Designing for good indoor air quality

A modern building is the result of a complex set of technologies. A large, diverse group of people must work in a coordinated, consistent fashion to make the resultant building useful, economical, safe, and attractive. IAQ is a relatively new and interdisciplinary concern for modern building design. Designing for good indoor air quality involves much detailed technical information in a wide variety of subjects, most of which are not yet familiar to many building design professionals. The architect (or other lead building design professional) must assemble a team capable of designing with IAQ in mind and must direct the members of the team throughout the process to maintain their focus on the goal of achieving good IAQ (Levin, 1991).

TABLE 28.3

Five major aspects of building design for good IAQ

Site planning and design	Ambient air quality; local source control; site design
Overall architectural design	Pollutant generating activities; envelope and structural materials; smoking lounges; building openings; vehicle access; operable windows; basement dehumidification
Ventilation and climate control	Dilution by outdoor air; outdoor air intake locations; exhaust locations; air cleaning and filtration; space air distribution; building pressure control; microbial control
Materials selection and specification	Low-emitting materials; preventive installation procedures; in-place curing
Construction process and initial occupancy	Design documentation and commissioning; special ventilation during finish work, furnishing, and initial occupancy period

Table 28.3 lists five stages of building design that must be addressed when designing for good IAQ. Data from each stage affects decisions in other stages. For example, prevailing wind speeds and directions are used in selecting locations for outdoor air intakes, just as soil and bedrock data are used during foundation design. The following sections of this chapter address these stages in detail.

Designers can simplify the problem of dealing with indoor environmental quality by considering it at every step of their work: during site evaluation and design, building schematic design, design development, and the preparation of construction documents. Some IAQ problems, especially those caused by unusual occupancy, cannot be anticipated adequately by designers. However, if a building is designed and constructed with the capacity to control contaminants in indoor air, trained operators can then use that capacity when it is needed.

28.1.5 Project documentation

From the outset of a project, thorough documentation improves communication among members of the design team and between the designers and

clients, construction contractors, and building operators. Design documentation is a requirement of the ASHRAE ventilation standard and is also necessary for performance evaluation during HVAC commissioning. It is desirable to appoint a member of the design team as the lead person and central contact for project documentation issues (ASHRAE, Standard 62-1989).

Documentation clarifies design objectives, system performance assumptions, loads, control logic, and other aspects of the HVAC system and its operation. It is a valuable tool for training operating staff and can help resolve occupant complaints quickly if they arise. In the event of litigation, thorough project documentation helps to establish the owners' and designers' intentions and efforts to produce and maintain good indoor air quality.

Separate portions of this chapter suggest elements to include in the project documentation for each major project phase as follows:

- documentation during project planning
- documentation during site evaluation
- documentation during design
- documentation during construction and commissioning
- documentation during initial occupancy

As project documentation accumulates, it should be organized and assembled in durable, moisture-resistant binders. Supplemented by operating and maintenance recommendations, the project documentation creates an owner's manual for the building. Architects and engineers should retain copies of the project documentation in their files and distribute multiple copies to building owners and operators.

28.1.6 Codes and standards

Building codes and standards establish minimum criteria for design and construction. In addition, in USA most states require that state-licensed professional designers prepare designs for buildings intended for public occupancy. These requirements imply that merely meeting building codes is not sufficient. If it were, then plans could be prepared by people who are not licensed design professionals, and permits could be issued on the basis of conformance to the codes. However, state legislation requires that designs be prepared by professionals whose competence has been reviewed and tested by state authorities.

Few existing standards specifically address indoor air quality concerns. However, the American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc. (ASHRAE) has developed ventilation and thermal comfort standards, respectively entitled "Ventilation for Acceptable Indoor Air Qual-

ity" and "Thermal Environmental Conditions for Human Occupancy", both of which can have a significant impact on IAQ. A discussion of these standards will be found later in this part of the book. Designers should be familiar with these ASHRAE standards and carefully consider all of their provisions during design (ASHRAE, Standard 62-1989; ASHRAE, Standard 55-1981).

ASHRAE Guideline 1-1989 for commissioning HVAC systems is intended to reduce the problems often associated with new buildings. Ventilation systems that are not fully functioning according to the design intent can be remedied before initial occupancy by following this guideline (ASHRAE, Guideline 1-1989).

28.2 PROJECT PLANNING

Planning for good IAQ cannot be a "tacked-on" extra or afterthought. It must be an integral part of the design process from the very start. Owners and architects often think IAQ control can be managed by simply reviewing plans and specifications shortly before issuing a bid package or by providing extra ventilation before and during initial start-up and occupancy. Those activities can help reduce some IAQ problems. However, many IAQ decisions should be made before determining schematic design and beginning design development. Later in the process, decisions that can affect IAQ have already been made and may be too difficult, time-consuming, and costly to revise.

This section discusses how to incorporate IAQ into the following project planning tasks:

- Programming
  - establish IAQ control as a priority and define IAQ goals and objectives
  - consider the type(s) of occupants and their susceptibility to indoor air contamination
  - consider occupant activities, devices, equipment, chemicals, and supplies associated with the principal, routine, and even occasional activities in the building
  - identify probable contaminant sources in each space or portion of the building
- Budgeting
  - specialized services during design, construction, and initial occupancy
  - time required for thorough commissioning and testing.
  - Selecting a design team.

28.2.1 Programming

The designer's first task is to convince owners and builders that "an ounce of prevention is worth a pound of cure". Work with the owner to define IAQ goals and objectives for the projected building. These goals should include strong, explicit statements regarding the delivery of quality air to occupants. As the project progresses, verify that the actual building use will conform to the assumptions used in the design.

The owner and designer should discuss the type(s) of occupants and their susceptibility to indoor air contaminants. Any group of people may include one or more individuals with asthma, allergies, or other conditions that predispose them to develop health symptoms from exposure to airborne contaminants. Some building areas may be intended for occupancy by groups that require special consideration, such as children or the elderly.

The dialogue between designer and client during the programming phase is valuable for identifying much of the information needed to address potential indoor air contaminant sources. Anticipate occupant activities and their potential to generate indoor air pollutants. Printing, copying, food preparation, and tobacco smoking are significant sources of indoor pollution. Document the activity analysis and review it during each of the design and contract document production phases that follow; update it as necessary.

Identify probable contaminant sources in each space or portion of the building and consider control strategies for each source. For example, space planning can separate incompatible functions, isolate pollution-generating activities, and buffer activities that are sensitive to air pollution.

28.2.2 Documentation during project planning

IAQ-related documentation during project planning should include items such as the following:

- building size and location
- types of occupants: note any special concerns over susceptibility to indoor air contaminants (e.g., children, elderly, asthmatics)
- anticipated occupant densities, activities, and use patterns for each space or type of space in the building.Include occupant densities for both "current" tenants and higher occupant densities of future tenants, if the potential for increased density exists
- activities with potential to generate indoor air pollutants (e.g., food preparation, tobacco smoking) including notes on types of pollutants and anticipated timing

  - building uses that require special environmental controls (e.g., libraries, laboratories, computer centres)
- project budget
  - budget for IAQ-related design activities
  - IAQ-related construction and commissioning costs. Note that there are additional considerations and costs if occupancy is to begin before construction has been completed throughout the building.
  - IAQ-related operating costs

28.2.3 Space planning

One of the most effective design approaches to indoor air quality control is to locate contaminant-generating activities where they will not affect other areas or where the contaminants can easily be exhausted. In large, multi-story buildings, this often requires careful program analysis and attention to space planning options early in the process. Collect sufficient information about intended activities and contaminant generation before space planning decisions are made. Later, when tenant improvements are designed, the specific use of the space and its air quality implications can be effectively considered in light of the design assumptions.

With leased and speculative buildings, details of the ultimate use are normally not available before construction of the base building. Therefore, plan a combination of flexible designs based on a realistic assessment of potential occupant activities that will result in acceptable IAQ. Where more than one area of a building or tenant space will be served by an air handling unit, make provisions to isolate completely the spaces during construction of tenant improvements or remodelling work. Consider installing a loop of exhaust ductwork to simplify later hook-ups if they become necessary. Some compromise is necessary. Unused flexibility results in unnecessary expense. However, inflexibility might eliminate potential tenants and be costly for a landlord or building owner in lost revenues or high costs for building modifications.

Identifying the range of necessary environmental control system requirements at the planning stage will accommodate both the owner's needs and the design requirements for acceptable air quality. Examples of specialized requirements include the special temperatures or humidities for computers or library collections and the special exhaust required for food preparation or graphic arts work.

28.2.4 Contaminants and their sources

Effective planning and design depend on knowing which sources release contaminants that can cause undesired effects. Each building is unique and its environment, design, construction, and use will vary over time; gaining the necessary knowledge about the effects of contaminants requires looking at the IAQ implications of many factors. Table 28.4 contains a summary of some major indoor air contaminants and their sources. These contaminants are related to typical building occupant complaints and adverse health effects.More information on them can be found in preceding chapters of this book.

TABLE 28.4

Sources of representative chemical and physical pollutants in non-industrial buildings

Examples of possible sources	Potential chemical or physical pollutant
Paint, glue, plastic	Organic gases and vapors
Paint, acid hardening lacquer, glue, Environmental Tobacco Smoke (ETS)	Formaldehyde and other aldehydes
Paint	Amines
Plastic	Phthalates
Impregnated wood	Fluorides
Gas cooker, unvented gas or petroleum oven	Nitrogen dioxide
Acid washed brick walls	Hydrogen chloride
People, unvented combustion	Carbon dioxide
ETS, unvented combustion, motor vehicle exhaust	Carbon monoxide
Copying machines, electronic cleaners	Ozone
Dirt from outside and from building materials	Inorganic dust
Paper, textiles	Organic dust
Acoustic ceilings, insulation	Man-made mineral fibres (MMMF)
House dust mites, animal dander, mould	Biological dust
Mould	Metabolic products of microorganisms
Solar radiation, lighting, heat-emitting apparatus	High temperature
Cold wall and window radiation, high air velocity	Cold discomfort
Machines, ventilation system, compressors	Low frequency sound, vibration
Machines, ventilation system, traffic	Noise
Daylight, electrical lighting, VDTs	Lighting glare and reflection, low contrast

28.2.5 Time factors of pollutant generation

The timing of pollutant generation significantly impacts the means for controlling the emissions. The schedule of pollutant-generating and pollutant-sensitive activities determines whether and how the activities affect each other. Consider these time factors when designing the ventilation system. Table 28.5 identifies some pollutant-generating activities according to location and timing or frequency of occurrence.

Designers can plan for temporally variable contaminant sources by providing flexible, easily operated control systems for HVAC. Flexibility is also achieved by using several units in parallel or series rather than a single large unit, such as a heat wheel or fan system.

TABLE 28.5

Contaminant sources and their spatial and temporal distribution

Type or location of source	Timing or frequency	General description or characteristics
Outdoor air	Cyclical	Daily traffic patterns Diurnal thermal patterns Seasonal thermal patterns Seasonal air quality variations Daily or seasonal releases from neighbouring structures or land
	Episodic	Extreme weather conditions Accidental releases
Base building	Continuous and episodic	Building materials Exposed to interior Exposed to air distribution system Concealed
Occupants and their activities	Continuous and episodic	Metabolic activity Work, study, rest, and recreation Food preparation Personal hygiene Operation of machines and equipment
	Periodic	Building maintenance Routine cleaning Dusting, vacuuming, waxing, and polishing Repair of building equipment Treatment of pest, odours

28.2.6 Budgeting

Plan for IAQ expenses during the budgeting process. The architect (or other prime design professional) should take responsibility for advising the client regarding IAQ expenses as well as the substantial costs of not incorporating good IAQ (Gainen, 1985).

Develop an estimate of the potential costs of addressing IAQ during design, construction and operation of the building. Include IAQ costs in the overall project feasibility analysis and project planning. Early attention to front-end costs can reduce or eliminate the considerable expense of post-occupancy IAQ problems (Levin, 1989).

The major IAQ budget considerations are funding for specialized services during design, construction, and initial occupancy and allowing sufficient time after construction for a thorough commissioning and testing of building performance.

The principal components of an IAQ project budget are shown in Table 28.6.

TABLE 28.6

The principal components of an IAQ project budget

– Advise the client of the costs of not addressing IAQ
– Develop an estimate of the potential costs of addressing IAQ during design, construction and operation of the building
– Include IAQ costs in the overall project feasibility analysis and project planning

28.2.7 Selecting a design team

The architect, as the lead designer, has the ultimate responsibility for a building's design and for determining that the completed building fulfils the design intent. The design team normally consists of architects, engineers, interior designers, specifications writers, specialized consultants, and construction experts. If the architect's own staff does not possess the necessary expertise, then specialized consultants are located and included in the design team. By utilizing in-house training or selecting consultants with demonstrated IAQ knowledge and project experience, architects will improve the working team's ability to avoid IAQ problems or resolve such problems as they arise.

When selecting contractors, consultants, and others with a critical role in creating the indoor environment, remember that indoor air quality is a developing field of knowledge. Most IAQ consultants received their primary training in other areas, making the selection process more complicated. Ask about training: attendance at IAQ conferences or seminars, familiarity with IAQ books or articles, IAQ self-study. Ask for a description of their experience with IAQ problems and with designing for good IAQ. Research their references: determine what service was provided and whether the consultant was knowledgeable and effective. Ask what they consider to be the most important considerations for IAQ based on their experience and the specific project under discussion.

28.3 SITE EVALUATION

Clients considering a purchase or planning a project often call on their architects and engineers to evaluate potential building sites. Many site characteristics can affect a building's ultimate IAQ and the measures necessary to control it. Site evaluation should be a part of feasibility studies, site planning and design, and building design.

This section describes the process of performing a site evaluation by gathering and analyzing information about the following:

- Climate — both the local climate and the building site's microclimate
- Outdoor air quality
  - local ambient air quality
  - nearby contaminant sources
- Soil and groundwater quality — on-site sources of contamination in soil and groundwater

28.3.1 Documentation during site evaluation

- IAQ-related documentation during site evaluation should include items such as the following:
- climate data
  - temperature/relative humidity: seasonal norms and extremes
  - wind: seasonal prevailing wind directions and speeds, local wind patterns
- outdoor air quality data
  - ambient air quality data from EPA NAAQS, state, or local sources
  - distance plot of nearby air pollutant sources, supplemented by detailed source information where available

- site data
  - prior site history
  - inventory of on-site sources (e.g., underground fuel tanks)
  - soil and groundwater test data

28.3.2 Climate/wind

Climate can affect IAQ in various ways. For example, weather conditions determine the amount of heating, cooling, and humidity treatment required for the outdoor air that is used for ventilation. Architects and engineers customarily evaluate temperature and humidity data when selecting HVAC equipment and designing for thermal comfort and energy efficiency. These factors and design decisions have important indoor air quality implications. Temperature and humidity extremes limit ventilation based on thermal conditioning capacity of the heating and cooling systems. If these extremes occur during periods of strong pollutant-generation indoors which typically includes warm weather periods, air quality can suffer from insufficient dilution and removal of pollutants.

Rather than review material that should be familiar to design professionals, the climate discussion in the remainder of this chapter has been limited to a consideration of wind patterns. Wind direction, speed, and timing determine the transport of airborne contaminants from areas surrounding the building site. Wind patterns also affect the potential for re-entry of exhausts and other emissions into the building. A wind rose is a useful tool for graphically displaying wind patterns at a building site. Figure 28.3 shows a typical wind rose, which visually displays the distribution of wind direction and velocity over the course of a year.

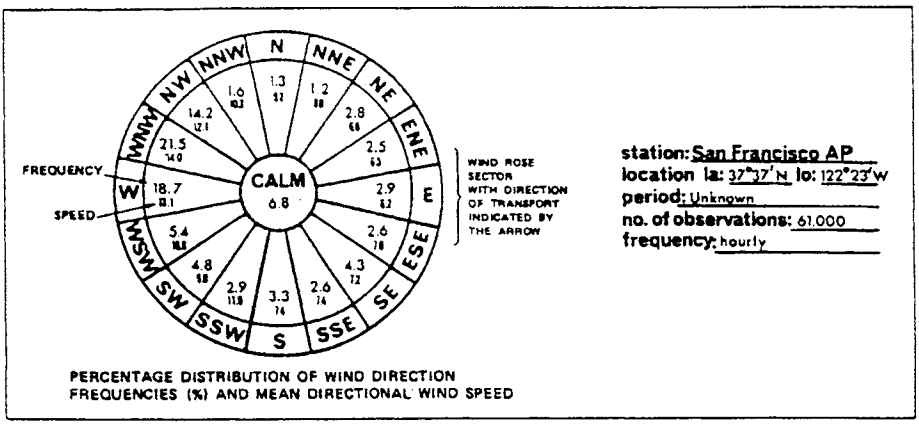


Fig. 28.3: Typical wind rose (Source: Thullier, 1970)

The wind roses in Figure 28.4 illustrate that annual distributions of wind direction can vary greatly from one locale to another. In addition, wind in an urban area can show turbulence and local wind shifts that are not revealed by airport or other regional data. Diurnal or seasonal variations in wind patterns may occur, often caused by sun angles or by topographic features such as hillside locations or proximity to bodies of water. On-site observations can be used to confirm or correct wind data obtained from locations remote from the building site.

Climate data for most U.S. metropolitan areas are available from the National Oceanic and Atmospheric Administration (NOAA). Additional data sources include airport administrators, harbor masters, academic and scientific institutions, hospitals, and some recreational facilities or equipment providers. Extensive on-site meteorology may also be available from industry and utilities because of past or present (regulatory) modelling requirements. In addition to the above sources, local newspapers often contain at least some if not all of the necessary data. Newspaper staffs often know individuals who monitor and record local weather in small communities.

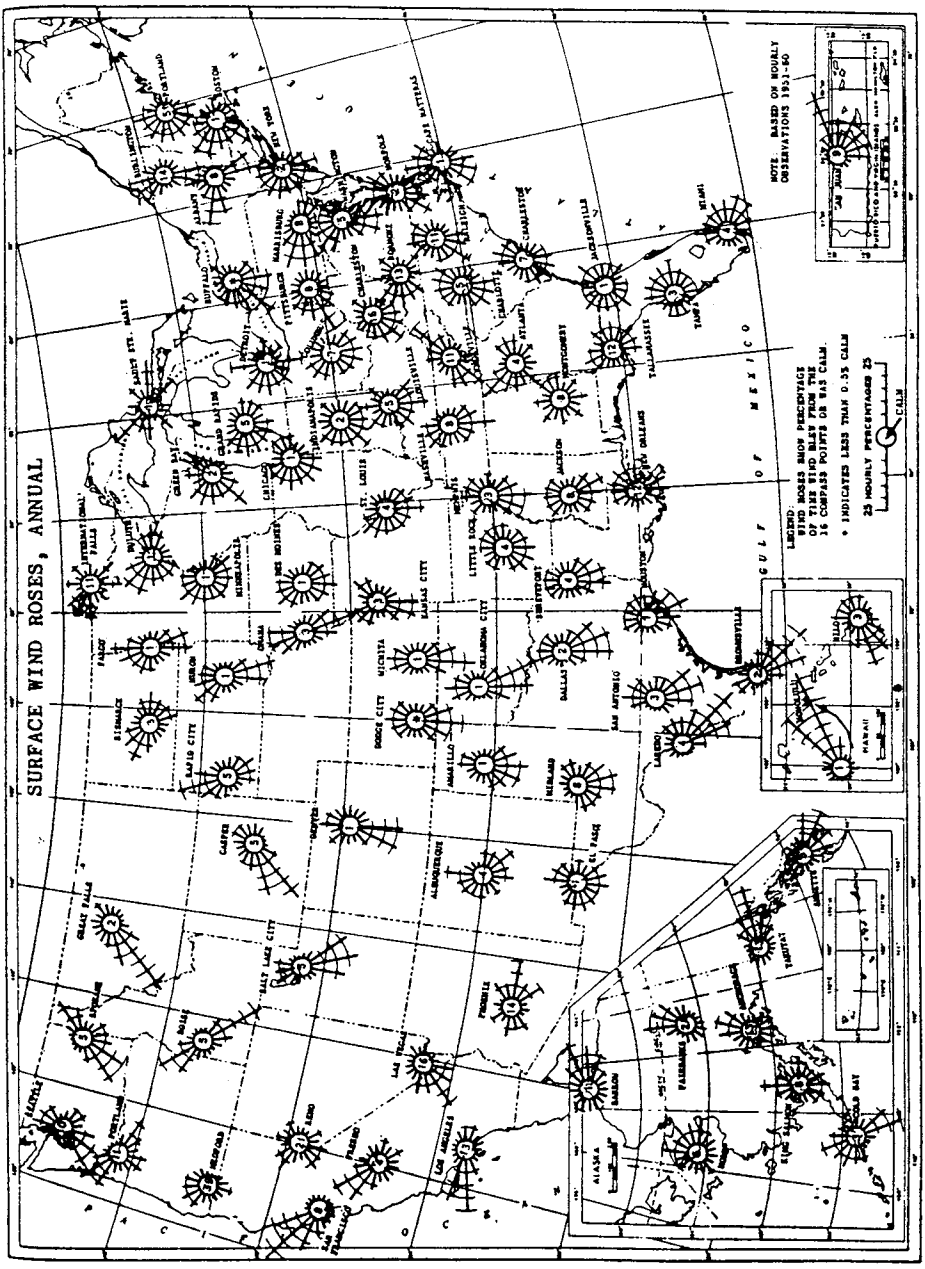
- Contact the nearest office of the National Oceanic and Atmospheric Administration (NOAA) or other sources for information on local weather for at least one complete year and historical weather extremes. The National Climate Data Center (NCDC), Department of Commerce, Asheville, NC, can provide weather data from the nearest airport.
- Obtain and plot hourly data for evaluating patterns before and during daily building occupancy periods. Plot data in a graphic form and analyze for prevalent patterns and variations.

Data plotted in visual form works best for site analysis. Wind data can be used to identify the best locations for outdoor air intakes, exhaust outlets, parking facilities, loading docks, and other features of the building and site that will affect indoor air quality. Wind pattern diagrams also help to clarify which potential contaminant sources near the site are most likely to influence IAQ in your building.

28.3.3 Outdoor air quality

Contaminated outdoor air results in occupant exposure to many of the same contaminants indoors. Since the ASHRAE ventilation standard and common sense suggest that outdoor air used for ventilation should be reasonably clean, it is important to determine the air quality in the community and (to the degree practicable) at the site, in order to design appropriate air cleaning and

Fig. 28.4 (Opposite page): Wind roses at selected sites throughout the U.S.


SURFACE WIND ROSES, ANNUAL
GULF OF MEXICO
NOTE BASED ON HOURLY OBSERVATIONS 1951-60
LEGEND
WIND ROSES SHOW PERCENTAGE FREQUENCY OF WIND FROM 16 COMPASS POINTS OR WAS CALM.
• INDICATES LESS THAN 0.55 CALM
25 HOURLY PERCENTAGES 25
CALM
HAWAII
ALASKA

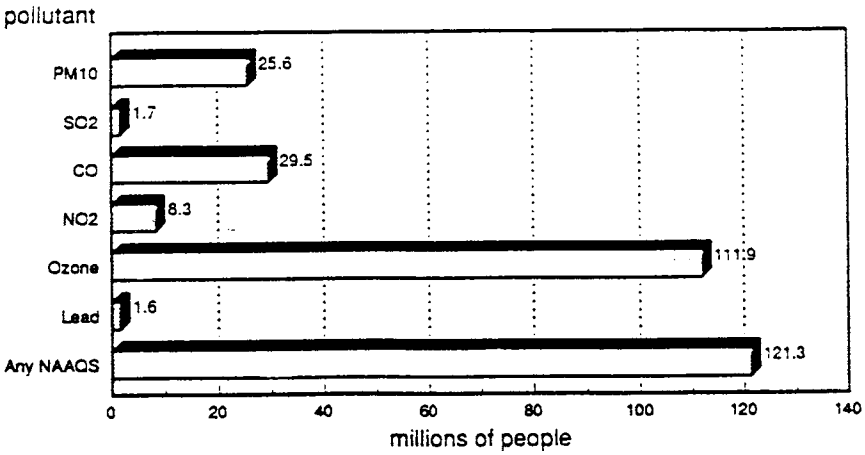


Fig. 28.5: People in counties with 1988 air pollutants exceeding the primary national ambient air quality standards.

filtration. Outdoor air is contaminated not only in most urban areas, but also in many suburban and rural parts. Figure 28.5 shows that in 1988, nearly half of all Americans lived in counties which had air ambient pollution levels exceeding EPA's National Ambient Air Quality Standards (NAAQS). Most of these exposures involve ozone, but other contaminants are also involved.

Ambient air quality data should be obtained for at least the most recent available year or two. Hourly data are most helpful, because diurnal and hourly variations are significant for most regulated outdoor air contaminants. The flow-chart in Figure 28.6 shows the process of assessing outdoor air quality at the site. It is important to begin assembling this information as early as possible in the design process so that missing data can be tracked down in time to utilize it during design.

In USA, each year, EPA's Office of Air Quality Planning and Standards publishes a national emissions report covering major metropolitan areas. NAAQS data is assembled by state and major metropolitan area. To obtain more site-specific data, contact the local or state agency responsible for ambient air quality monitoring in your area or the EPA office with jurisdiction. If you do not know how to identify the pertinent agency, the EPA Regional Office will be able to help you identify it.

28.3.4 Adjacent and nearby site uses

Data available from regional or community sources is generally appropriate for a site analysis, but may not be sufficient if there are nearby contaminant sources such as local industry or if the local topography is irregular (Thullier, 1978; EPA, 1977).

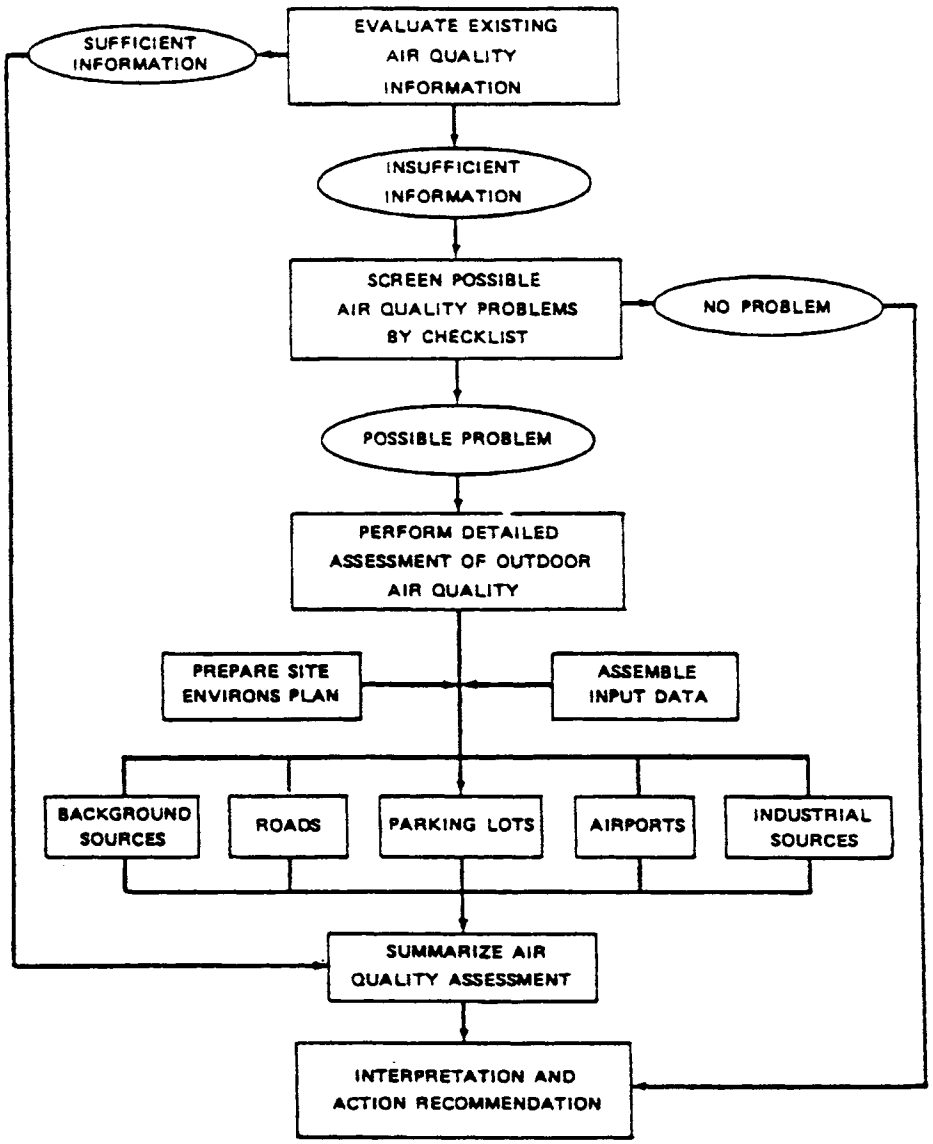


Fig. 28.6: Flowchart for the outdoor air quality assessment process.

Contaminants generated on adjacent sites may be a greater concern than regional ambient air quality. The potential for this type of contamination is based on factors such as:

- Types and quantities of contaminants generated on adjacent sites
- Adjacent building design characteristics
- Prevailing wind patterns and velocities

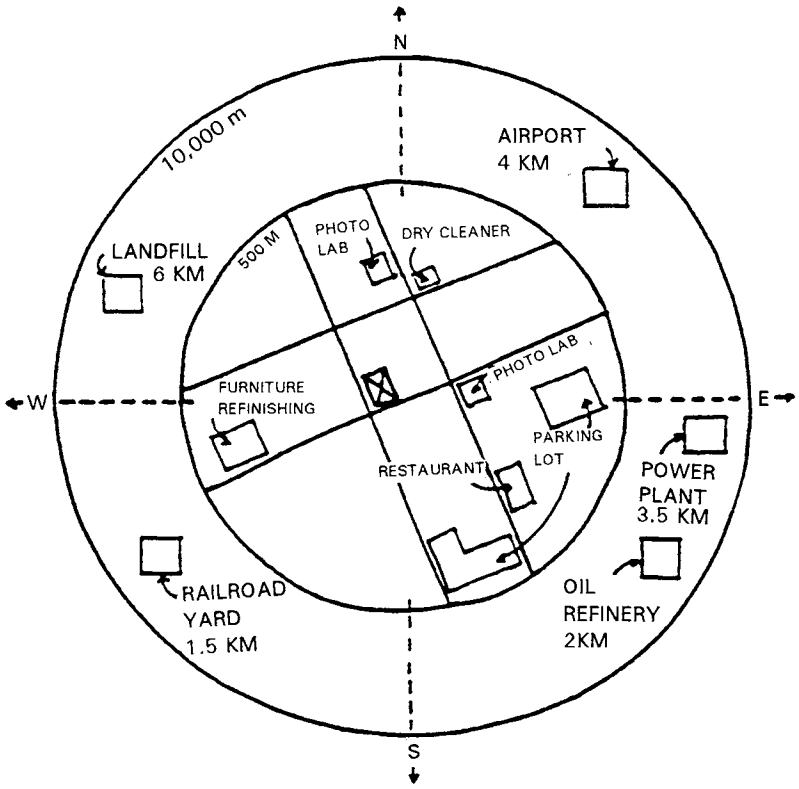


Fig. 28.7: Sample distance plot of air pollutant sources.

- Configuration of neighbouring buildings which could affect the dilution or escape of contaminant plumes
- General topography

Prepare a map of the area surrounding the site showing potential and actual sources of contaminants. An example of a potential source is land that is currently vacant, but is zoned for industrial use. Include both constant and intermittent sources.

After identifying existing and potential pollutant sources on adjacent and nearby sites, plot them on a scaled distance plan similar to Figure 28.7. This facilitates assessment of potential plume strength at the building site. EPA publishes general information on emissions from various types of industrial and commercial activities.

Identify each potential source and assess the potential for contamination of building intake air. Figure 28.8 shows an example of a map prepared for a

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Fig. 28.8 (Opposite page): Map of a housing project, Oakland, CA.

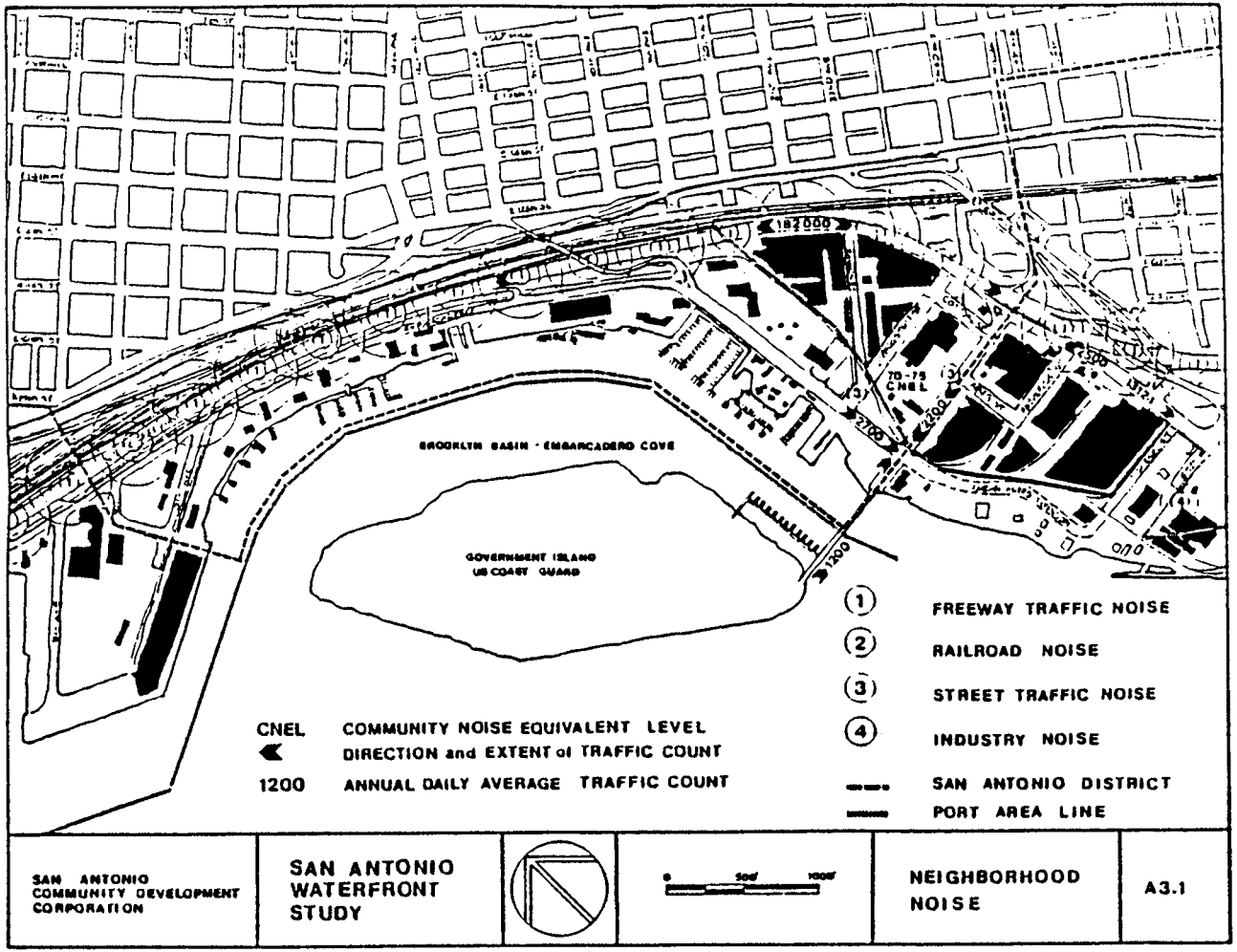
182000
70-75 CNEL
2700
2500
1500
BROOKLYN BASIN - EMBARCADERO COVE
GOVERNMENT ISLAND
US COAST GUARD
CNEL COMMUNITY NOISE EQUIVALENT LEVEL
DIRECTION and EXTENT of TRAFFIC COUNT
1200 ANNUAL DAILY AVERAGE TRAFFIC COUNT
1 FREEWAY TRAFFIC NOISE
2 RAILROAD NOISE
3 STREET TRAFFIC NOISE
4 INDUSTRY NOISE
SAN ANTONIO DISTRICT
PORT AREA LINE
SAN ANTONIO COMMUNITY DEVELOPMENT CORPORATION
SAN ANTONIO WATERFRONT STUDY
NEIGHBORHOOD NOISE
A3.1

TABLE 28.7

Activities that may be sources of ambient air contaminants. A partial list of some of the more common urban and suburban activities which produce ambient air contaminants

Commerical facilities	Laundry and dry cleaning Restaurants Photo-processing shops and laboratories Automobile repair shops and body shops Paint shops
Manufacturing	Electronics manufacturing and assembly Wood products, wood preservative treatment plants Paper and pulp
Utilities	Electric power plants Central steam plants
Agricultural facilities	Greenhouses Orchards Processing and packing plants

housing project in Oakland, California. The map combines information about potential sources of air contaminants with information about wind patterns (i.e., prevailing winds and vertical air mixing).

Table 28.7 provides some examples of some of the more common urban and suburban activities that produce ambient air contaminants, including odours.

Detailed information about contaminants and emissions near the site may be available from the owners or operators of facilities that are contaminant sources or from permit-issuing agencies or fire districts. If one cannot obtain reliable data to characterize the ambient air quality or emissions from specific sources, an air quality consultant can be hired to develop and implement an air quality monitoring program. Contact local or state agencies to obtain a list of firms qualified to perform the required services.

Obtain ambient air quality data for at least the most recent available 1–2 years. Identify current and zoning-approved uses for adjacent and other nearby sites. Contact owners or operators of facilities that may be contamination sources to request information on processes and emissions.

If information is not available directly from suspect properties, inquire at air quality districts for permits related to emissions, enforcement actions, or other information. Contact fire districts for hazardous materials lists from suspected sources.

Outdoor air quality may affect the relative desirability of alternative sites. After site acquisition, outdoor air quality data can be used in selecting equip-

ment for air filtration or air cleaning and in determining the best location for outdoor air intakes, exhaust outlets, parking facilities, loading docks, and other features of the building and site that will affect indoor air quality (Thullier, 1978).

A three-volume manual published by the U.S. Department of Housing and Urban Development (HUD) in 1978 presents a procedure for evaluating outdoor air quality at the site by combining wind data with ambient air quality data. Figure 28.9 presents an area-wide input data summary form from the HUD manual. The form also serves as a guide to identifying critical weather data.

28.3.5 Soil and groundwater quality

Soil and groundwater can be sources of indoor air contaminants. Gases such as methane are released underground as man-made and naturally occurring organic materials degrade. Some earthen materials also release gases; for example, radon is produced by the decay of radium. The radon potential of the ground depends on the local geological features, geochemistry and soil porosity. This information is typically available from local or national geological survey offices. Radon potential can also be tested.

The presence of contaminants in the soil or groundwater can indicate a need to reconsider the purchase of a site or to include design and construction features to prevent contaminant entry. Cracks, utility penetrations, and other openings in the foundation or basement walls can be pathways for contaminant entry. Soil gas can penetrate walls, depending on the permeability of the construction materials and the quality of the construction. Less commonly, airborne contaminants from soil gas can be drawn into the building after they are emitted into the ambient air, or groundwater contaminants can enter the building by way of the domestic water supply.

Because of the stack effect in most buildings (especially tall buildings), air pressures in lower levels and sub-grade spaces are often lower than in the surrounding soil. As the building sucks on the earth, gases can be drawn into the building.

Fractures and fissures can greatly influence final building design considerations. In USA fracture data are available from the United States Geological Service (USGS) and fissure data are available from ground-penetrating radar surveys done by utility companies.

28.3.6 Site history

Prior site uses such as agriculture, on-site processes, or waste dumping may have contaminated soil and groundwater. Some building areas were

OBSERVED AIR QUALITY DATA

Pollutant	Carbon Monoxide (mg/m^3)		Sulfur Dioxide ($\mu g/m^3$)		Suspended Particulate ($\mu g/m^3$)		Oxidant ($\mu g/m^3$)	Nitrogen Dioxide ($\mu g/m^3$)
Averging Time	1-hr	8-hr	24-hr	Annual Average	24-hr	Annual Geom. Mean	1-hr	Annual Average
Second-Highest Observation								

DEMOGRAPHIC DATA

Place Name	Elevation Above Sea Level	Population	Area (km^2)	Percent Use of Coal or Oil for Space Heat

AREA SOURCE POLLUTANT EMISSIONS DATA

Source Area	Emissions (metric tons per day)		
	CO	SO_2	TSP

County Emissions (Mg/Day) CO_____ SO_2_____ TSP_____

METEOROLOGICAL DATA

Annual average wind speed _____ (m/s)

Annual average mixing height _____ (m)

Annual average heating degree days _____

Local wind rose:

Direction	N	NE	E	SE	S	SW	W	NW
Frequency (%)								
Speed (m/s)								

Fig. 28.9: Sample air quality and weather data summary form.

formerly agricultural lands and could contain high concentrations of potent pesticides, herbicides, and fertilizers. Even recreational lands may have been treated with toxic chemical fertilizers or pesticides. Run-off from nearby sites may also deposit contaminants.

Prior construction may have been improperly or imprudently demolished, leaving hazardous residues. Contaminants such as lead, arsenic, and asbestos have been found at building sites. If older buildings were sandblasted, residual sand may contain high arsenic levels. Improperly disposed solvents, adhesives, paints, oils, and paint thinners are potential soil contaminants from former commercial or industrial activity.

Local planning agencies may have records of historical land uses. If not, the county recorder's office will have records of the previous owners of the land. Fire departments and local environmental health agencies may also have useful records. Abstract services have a responsibility to identify prior owners and uses. Flyover photographs (1:200 scale) are available nationwide through the Soil Conservation Service and through local planning agencies or surveyors.

If investigations reveal that prior uses may have contaminated soil or ground water, sample for chemical or physical contaminants before the final purchase. These testing requirements can be combined with routine soil and other geological testing. Cores extracted from different depths will be necessary where fill has been dumped.

Contact a qualified consultant with knowledge and experience in hazardous materials for advice and testing as required. Local air quality, environmental health, and hazardous materials control agencies can also provide assistance or identify qualified consulting firms.

On-site soil and water quality problems may affect the relative desirability of alternative sites. If a site has already been acquired, the building design, excavation, utilities plans, or other design and construction elements can incorporate measures to prevent contaminant entry. Local air quality, environmental health, and hazardous materials control agencies may be able to recommend appropriate actions. A substantial amount of guidance on radon-resistant construction features for one and two family dwellings is available from EPA. Some of this guidance can be applied to larger buildings. EPA is currently developing guidance on preventing entry of hazardous soil gases.

28.3.7 Other environmental considerations

While noise, lighting, and other environmental considerations are outside the scope of this primer, it is important to consider these factors during the site evaluation. Noise may affect the ability to use windows for ventilation or thermal control. Glare from adjacent buildings may influence the sizing and

placement of windows or the selection of glazing shading coefficients. Sound-transmitting or -carrying equipment locations should also reflect potential noise sources outdoors. For example, avoid locating air intakes near busy roadways or cooling towers or incorporate design considerations that will minimize problems (e.g., orientation, shielding).

28.4 DESIGN SERVICES AND GOOD INDOOR AIR QUALITY

28.4.1 Introduction

Earlier sections have described how to incorporate IAQ concerns into project planning and show the client the long-term benefits of maintaining good indoor air quality. During programming, the designer and client establish the goals and assumptions of the project. Once good indoor air quality has been adopted as a goal, it is necessary to identify the special features of the project that affect IAQ. Another section discussed data-gathering to gain a better understanding of potential outdoor air quality problems at the building site.

This section discusses the design phase, in which the goals, objectives, and assumptions that govern the project are translated into decisions about building form, space planning, environmental control systems, and the materials used to construct and furnish the building.

Design services vary greatly from project to project depending upon the client, the building, and the division of responsibilities. Construction managers, clients' representatives, and design professionals all play varying roles. Regardless of the distribution of responsibilities, IAQ-conscious designers should include the following items as part of each project:

- IAQ criteria: define explicit criteria that will direct design decisions and allow later evaluation of IAQ in the building;
- Site planning: address outdoor air and ground contaminants;
- Designing the building and mechanical systems;
  - design environmental control system to provide for both IAQ control and thermal comfort;
  - include flexibility to allow for future changes;
  - consider IAQ during selection of building materials and furnishings.

Most of the material in this section is also applicable to renovating activities. It is equally important to consider indoor air quality in designing for changes in existing buildings, although there may be less flexibility (i.e., higher cost) to respond to many IAQ concerns. This is an important reason to bring IAQ considerations into play from the start of a new construction. The design should be planned from the beginning to include flexibility to meet future needs (e.g., increased occupant population, installation of additional

emissions-producing equipment) and maintain good IAQ throughout the lifetime of the building.

IAQ-related documentation during the design phase should include items such as those listed in Table 28.8.

TABLE 28.8

Details of design documentation

Design criteria	– Indoor design conditions (all seasons): temperature, relative humidity, air movement – Outdoor design conditions (all seasons): temperature, humidity, wind direction and velocity, location and timing of outdoor sources of air pollutants of concern. – Assumed or anticipated occupant densities, activities and use patterns for each space or type of space in the building. – Assumed electrical load for light and power. – Any special loads which might exist. – Outside air supply rates under various operating conditions and loads. – Assumed ventilation effectiveness for each type of space under HVAC system mode of operation which results in different air supply design conditions. Definition of building envelope, including type and characteristics of materials and assumed infiltration. – Air quality design criteria. – Code requirements. – Noise criteria. – Fire and safety requirements. – Energy efficiency and projected operating cost.
HVAC system description	– Basic system types. – Major components. – Capacity and sizing requirements. – Redundancy provisions. – Intended operation in each seasonal model, including designed changeover conditions. – Changeover procedures. – Part-load operational strategies for each season. – Occupied/unoccupied operation modes for each season. – Design setpoints for control system, including permissible limits of adjustments. – Operation of system components in life safety modes. – Energy conservation procedures. – Any other engineered operational model of the system.

Some of the items to be included in IAQ documentation during the design phase already appear in standard bid documents. For example, the site plan necessarily shows the location of the building on the site, along with such features as roadways, parking areas, and landscape plantings. Architectural and mechanical plans show the locations of building openings and HVAC components. However, it is advisable to generate duplicate architectural/mechanical plans that can be annotated with additional IAQ-related information. For each item to be documented, consider what presentation format will be most useful to the building owner and operators. For example, air circulation patterns and pressure relationships are most clearly represented using floor plans, while manufacturers' test data on VOC and SVOC emissions might supplement a schedule of building materials.

28.4.2 Indoor air quality criteria

IAQ criteria provide an operating definition of "good air quality". The use of clearly-stated criteria allows decisions that affect IAQ to be made with the concurrence of the client and the designer and facilitates later evaluation of air quality in the building. When incorporated into the building design, IAQ criteria can provide for occupant health and comfort and protect the building fabric and systems.

One may determine that applicable building codes and standards, if actually implemented, will serve as adequate IAQ criteria for a given project. Even so, IAQ will be better protected if the applicable code provisions and related standards are clearly identified rather than being left implicit.

Table 28.9 shows three basic IAQ criteria and related design parameters. Experience has shown that additional or differing criteria are sometimes desirable. IAQ guidance established by governmental bodies, professional /societies, and consensus standards development groups can serve as an additional source of IAQ criteria. One can also develop special criteria for certain spaces in the building. Recommend IAQ criteria that can be incorporated into design assumptions, evaluated by mathematical modelling during design, and tested empirically during construction and in the completed building.

Criterion: thermal comfort needs are met per ASHRAE Standard 55-1981

Many IAQ complaints have been traced to thermal control problems involving building mechanical equipment. ASHRAE Standard 55-1981 "Thermal Environmental Conditions for Human Occupancy" identifies the range of design values for temperature, humidity and air movement that will provide a satisfactory thermal environment for 80% or more of the building occupants.

TABLE 28.9

Three basic IAQ criteria and related design parameters

Indicators	Occupant responses:
	Odour
	Irritation
	Illness rate
	Acceptability
	Carbon dioxide concentrations
	Occupant density
Characterization	Total VOC concentrations
	Particulate matter concentrations
	Formaldehyde concentrations
	Humidity levels
Factors	Outside air ventilation rate
	Source characterization
	Air filtration and cleaning performance
	Building pressures

The interaction of temperature and relative humidity

Figure 28.10 illustrates the "comfort envelope" of acceptable temperatures and relative humidities for summer and winter conditions (ASHRAE Standard 55-1981). Note that as the relative humidity rises cold temperatures become more tolerable, but high temperatures become less tolerable.

The activity level, age, dress, and physiology of each person affect that person's thermal comfort requirements. Standard 55-1981 assumes indoor clothing that is normal for the season.

Table 28.10 shows the operative temperature for thermal acceptability of sedentary or slightly active persons at 50% relative humidity recommended from ASHRAE Standard 55-1981.

Table 28.11 shows thermal environmental performance criteria for maintenance of comfort conditions (Woods et al., 1989a).

The effect of air movement on thermal comfort

According to Standard 55-1981, comfort can be maintained at air temperatures between 79°F and 82.5°F if the average air movement is increased 30 fpm (feet per minute) for each degree F of increased temperature. This strategy reaches its limit at 82.5°F and 160 fpm of air movement (see Fig. 28.11). Loose paper, hair, and other light objects may start to be blown about at air movements in excess of 160 fpm. However, recent laboratory research has

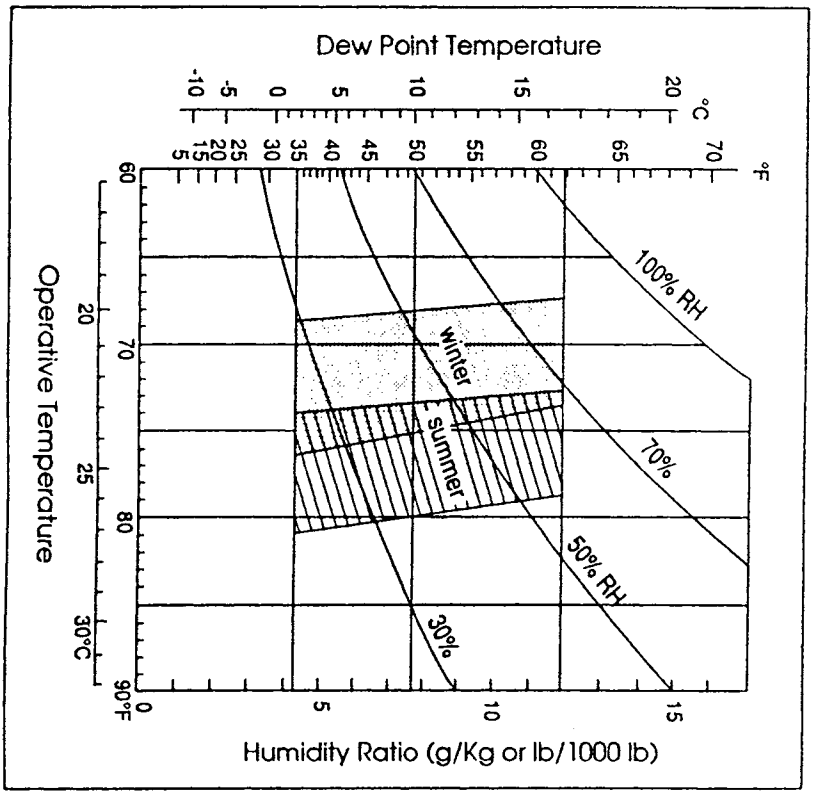

Fig. 28.10: Temperature/humidity ratios in the "comfort envelope".

TABLE 28.10

Operative temperatures for thermal acceptability of sedentary or slightly active persons at 50% relative humidity

Season	Description of typical clothing	clo*	Optimum operative temperature (°F)	Operative temperature range for 80% thermal acceptability (°F)
Winter	heavy slacks, long-sleeved shirt and sweater	0.9	71	68–74.5
Summer	light slacks and short-sleeved shirt	0.5	76	73–79

* clo value is a numerical representation of a clothing ensemble's thermal resistance.

TABLE 28.11

Thermal environmental performance criteria for maintenance of comfort conditions

Parameter	Guideline
Operative temperature (winter)	68.5 to 76°F (at 30% humidity)
Operative temperature (summer)	73 to 79°F (at 50% relative humidity)
Dew point	>35°F (winter); <62°F (summer)
Air movement	≤30 FPM (winter); ≤50 FPM (summer)
Vertical temperature gradient	Shall not exceed 5°F at 4 inch and 67 inch levels
Plane radiant asymmetry	<18°F in the horizontal direction; <9°F in the vertical direction

Source: Woods et al., 1987.

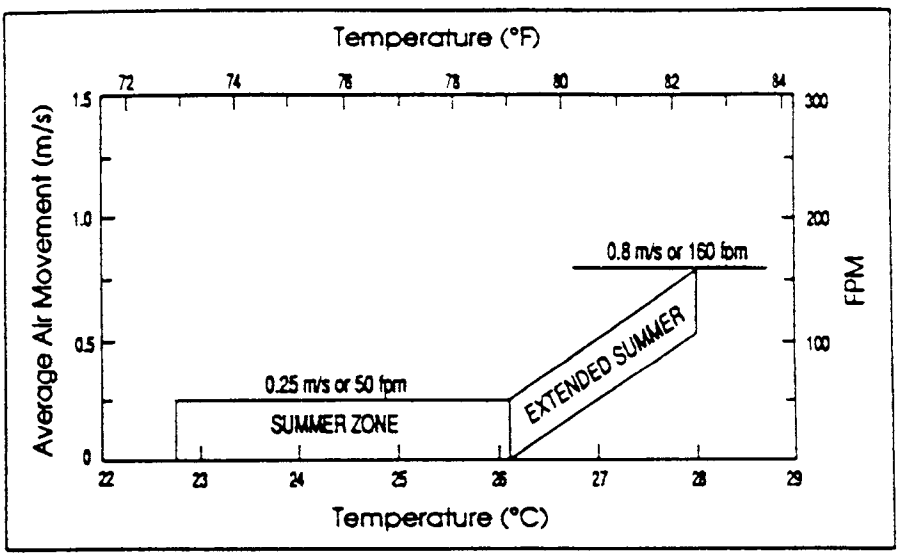

Fig. 28.11: Average air movement and temperature.

clearly shown that air quality is deemed less acceptable at temperatures above 76°F, even when the actual air quality is held constant. If tolerance for contaminants decreases at the upper end of the comfort range, control of contaminants is especially important during warmer periods. Most office buildings require cooling most of the time suggesting that temperatures are often near the upper end of the comfort range and that air quality control is particularly important under these conditions.

Radiant heat gain and loss

Radiant heat gain and loss also affect occupant comfort. Radiant heat transfer occurs between surfaces that are not touching. When windows and uninsulated walls are significantly hotter or colder than the room air, people located near these surfaces may be uncomfortable even if the measured air temperature is within the comfort range. Adding insulation or reducing solar gains can help to reduce these radiant heat effects.

Relevant design parameters

The design of HVAC zones, the selection of heating and cooling equipment, and the use of humidification or dehumidification equipment all affect whether or not conditions in the building meet the thermal comfort criterion. These design features will be discussed later.

Evaluating the success.

The following measurements can be used to evaluate whether the criterion of providing thermal comfort has been:

- indoor temperature and relative humidity (e.g.compare to ASHRAE Standard 55-1981); and
- number of occupant thermal comfort complaints.

Criterion: Outdoor air ventilation meets or exceeds ASHRAE Standard 62-1989

Standard building codes generally prescribe minimum outdoor air ventilation rates for mechanically-ventilated spaces. In most cases, these rates are adopted from ASHRAE Standard 62. However, because of time lags inherent in code amendment, the building codes that apply to a project may not reflect the most recent version, Standard 62-1989.

The design guidelines in ASHRAE Standard 62-1989 "Ventilation for Acceptable Indoor Air Quality" are intended to produce indoor air quality that is acceptable to at least 80% of a building's occupants. The standard provides alternative approaches to demonstrate compliance. One approach is to provide the prescribed outdoor air ventilation rate for each occupancy type. An alternative "air quality" procedure contains a number of criteria, including a subjective evaluation of air quality as well as maximum concentration limits for certain chemicals.

Most designers use ASHRAE Standard 62 only to obtain values for the outdoor air ventilation rates (as shown in Table 28.9). However, other requirements such as the documentation of design assumptions and the removal of contaminants from unusual sources are equally important. Designers should

be familiar with this ASHRAE standard and carefully consider all of its provisions and assumptions. Specific provisions which are particularly important to IAQ should be clearly articulated in the architect–engineer agreement. These provisions include but are not limited to: quality assurance, outdoor air quality, occupant density, and treatment of high contaminant areas such as smoking lounges.

Limitations of Standard 62-1989

It may be important to prescribe higher ventilation rates than those recommended in ASHRAE 62-1989. Early in the design process, designers should discuss this possibility with their clients and agree on the design objective. For example, if more than 80% of the occupants are to be satisfied with the indoor air quality, it will probably be necessary to supply more than the minimum ventilation rates prescribed by ASHRAE or to improve source control.

The ASHRAE ventilation standard is primarily based on occupant comfort rather than on the health effects of indoor air contaminants. Although widely accepted health-based guidelines are not available for most indoor air contaminants, there are clearly situations in which the prescribed minimum outdoor air ventilation rates would not provide essential health protection. The committee that drafted the standard agreed that controlling ventilation to the prescribed minimums would protect against adverse health effects from most contaminants, but included the caveat that the standard is intended for spaces where no unusual sources of contamination exist.

Although the standard does not define "unusual sources", its authors made assumptions about the types of activities usually found in each type of space for which the standard specifies outside air ventilation rates. It is especially important that designers identify possible sources of unusual or major contaminants in any building they are designing and provide specific means for controlling them. Evaluate sources based on the following considerations:

- Is the source normally found in this type of occupancy?
- Are there contaminants emitted from the source that will not be perceived by the occupants (i.e., will the sources emit non-odorous or non-irritating substances that are capable of causing acute or chronic health effects)? This type of contaminant is not directly addressed by the ASHRAE standard. Examples are radon, certain pesticides and carcinogens, and possibly ozone.

The minimum ventilation rates and estimated maximum occupancy values shown in Table 28.12 are used as assumptions when actual occupancy plans are not known at the design stage. Actual occupancies vary considerably both above and below those listed in the table. It is preferable to use occupancy estimates based on information from the building owner or occupant. Consider

TABLE 28.12

Extract from Table 2, ASHRAE Standard 62-1989

Application	Estimated maximum occupancy (P/1000 ft^2)	cfm/person	cfm/ft^2
Food and beverage service			
Dining rooms	70	20	
Cafeteria, fast food	100	20	
Bars, cocktail lounges	100	30	
Kitchen (cooking)	20	15	
Offices			
Office space	7	20	
Reception areas	60	15	
Conference rooms	50	20	
Public spaces			
Smoking lounge	70	60	
Elevators			1.00
Retail stores, sales floors, and show room floors			
Basement and street	30		0.30
Upper floors	20		0.20
Malls and arcades	20		0.20
Smoking lounge	70	60	
Sports and Amusement			
Spectator areas	150	15	
Game rooms	70	25	
Playing floors	30	20	
Ballrooms and discos	100	25	
Theatres			
Lobbies	150	20	
Auditorium	150	15	
Transportation			
Vehicles	150	15	
Education			
Classroom	50	15	
Music rooms	50	15	
Libraries	20	15	
Auditoria	150	15	
Hotels, motels, resorts, dormitories			cfm/room
Bedrooms			30
Living rooms			30
Lobbies	30	15	
Conference rooms	50	20	
Assembly rooms	120	15	

the potential for increased occupant density in the future, so that flexibility can be designed into the HVAC system.

Relevant design parameters

The ventilation recommendations of ASHRAE Standard 62-1989 have a direct effect on the selection of outdoor air ventilation rates for each type of occupancy and on the design of the supply air distribution system.

Note that, in addition to meeting the respiration needs of building occupants, outdoor air must be provided to replace the air removed by exhaust fans and to supply combustion air for boilers, water heaters, and cooking appliances. In some buildings, exhaust fans and combustion appliances are the dominant factors in determining the quantity of outdoor air required for proper operation.

Evaluating the success

The following measurements can be used to evaluate how well outdoor air ventilation needs have been met in the design:

- occupant judgments of the indoor air's acceptability;
- measured outdoor air ventilation rate;
- peak carbon dioxide concentration (the standard is designed to maintain CO_2 concentrations at or below 1000 ppm)

Criterion: Indoor contaminant levels are minimized

The outdoor air ventilation rates recommended in ASHRAE Standard 62-1989 are intended to lower contaminant concentrations through dilution. However, it is preferable to control contaminant sources, rather than increasing general ventilation beyond minimum recommended rates.

IAQ criteria can be established for specific contaminants such as VOCs (volatile organic compounds), formaldehyde, ozone, particulates, or radon. This can be valuable if activities and processes identified during programming are potential strong sources of specific contaminants. However, it may be difficult to select target concentrations, because most of the existing guidance is geared to industrial settings. Indoor air exposures in non-industrial environments usually involve exposure to multiple chemicals and may not be adequately addressed by the exposure limits developed for healthy industrial workers. Many organizations including the World Health Organization, ASHRAE, and NIOSH have recommended exposure limits substantially below the occupational guidelines of the private, non-profit American Conference of Governmental Industrial Hygienists (ACGIH) or the federal Occupational Safety and Health Administration (OSHA) (see Part VIII). When no other

guidance exists, ASHRAE recommends using 1/100 of the occupational exposure limit as a guideline value (WHO, 1987; ASHRAE Standard 62-1989).

It is often more practical to adopt a general criterion of minimizing indoor contaminant concentrations than to establish target concentrations for specific substances or classes of contaminants.

The general criterion of minimizing indoor contaminant concentrations can be subdivided into three elements:

- minimize the production of indoor air contaminants;
- isolate and remove indoor air contaminants;
- minimize the entry of outdoor air contaminants.

Relevant design parameters

To minimize the production of indoor air contaminants, the designer should review each class of building materials and select the materials and installation procedures that offer the lowest emission rates. The design and operation of building mechanical systems can isolate and remove air contaminants through the control of building pressures and the use of local exhaust. The entry of outdoor air contaminants can be minimized through careful location of building openings (e.g., outdoor air intakes, loading docks, and doors) and on-site contaminant sources such as dumpsters, as well as through selection of appropriate air filtration/air cleaning equipment. These topics will be discussed at greater length later in this chapter.

Evaluating the success

The following measurements can be used to evaluate how well indoor contaminant concentrations have been minimized:

- frequency of occupant complaints about odours or health symptoms;
- measured concentrations of specific target contaminants;
- pressure differentials measured between different functional areas within the building (e.g., between bathrooms and adjoining hallways) and between the building and outdoors.

28.4.3 Site planning

Section 28.3 provided guidance for site assessment and evaluation. Using the information in that section and the resulting analysis, the design team can develop a site plan that will minimize the impact of outdoor air pollution of IAQ. Major elements of site design that can improve IAQ are setbacks, bird-proofing, landscaping, the shape and orientation of the building shell, parking and vehicular circulation planning, and management of other on-site contaminant sources.

Setbacks

Setbacks protect structures from vehicle emissions and other nearby off-site sources. For sites near heavily travelled roadways, a small increase in setbacks can mean a large decrease in contaminant concentrations.

In reasonably calm air, contaminant concentrations decrease quickly with increasing distance from the source. This is because contaminants tend to disperse in three dimensions, producing significant dilution as distance increases. Where winds are constant and not too strong, little dispersion may occur and contaminant plumes can be entrained in building ventilation air.

Landscaping

Landscaping buffers buildings from off-site contaminant sources. Plants can break up air flows that contain contaminants. Be careful not to place planting materials and the soil supporting them too close to air intakes. Microbial activity, decay products in the soil, chemical fertilizers, pesticides, and pollen can become indoor air contaminants. Plant material can also physically block air flow. Consider not only the original size of each planting, but also its potential dimensions at maturity.

Bird-proofing

Perching, roosting, and nesting locations that attract birds can result in accumulated wastes that could promote microbial growth and lead to serious illness. For example, a suburban office with an adjacent lake can attract many birds that may roost on air intake grilles, leaving their faeces. Grilles protecting air intakes should be "bird-proof" to prohibit perching or roosting as well as entry. Horizontal grilles create the most serious problems, because droppings fall into the outdoor air intake plenums.

Shape and orientation of building shell

Arrange structures to allow for the movement of prevailing winds, avoiding stagnant air and the trapping of pollutants. Locate exhausts so that prevailing winds will sweep exhaust plumes away from the building. The locations and orientation of outdoor air intakes should be designed to avoid the entry of contaminants from your own site or the surrounding area. If one is not certain of the air flows through his site design, it may be necessary to conduct wind tunnel testing or mathematical modelling to determine the air flow around and through the building masses (ASHRAE, 1989).

Parking and vehicular circulation planning

Large masses of on-site parking areas, with concomitant vehicle idling or running at low speeds, generate considerable amounts of emissions. Locate

vehicle parking, loading, and roadway areas away from building openings or air outdoor supply intakes, or consider orientation and shielding options that will minimize the potential for contaminant entry.

Management of other on-site contaminant sources

In addition to roadways, parking and loading areas, your site design may include other features that could be sources of indoor air contaminants. Examples include decorative elements such as flower beds or fountains, as well as functional items such as dumpsters, incinerators, and underground fuel tanks. Select locations and designs that fulfil the intended function while minimizing the potential for contaminant entry.

28.4.4 Building envelope

As the program and design criteria are translated into a design concept, it is important to consider the impact of alternative designs on IAQ. The locations of major elements and the way they fit into the general building scheme affects IAQ far more than the details developed in subsequent architectural phases. Some of the most important elements are: openings in the building shell, thermal characteristics of the building shell, the relationships of interior spaces, the environmental control scheme, and the dominant surface materials.

Critical components of the ventilation system such as air-handling equipment, air intakes and exhausts, and cooling towers must be planned in conjunction with the overall building shape. Do not omit these elements from drawings or models of preliminary design schemes and proposals. Some designs have even made a design element of the ventilation components. The Larkin Soap Company Administration Building, designed by Frank Lloyd Wright and built in 1906, was the first sealed, air-conditioned office building in the United States. Wright expressed the brick-clad ventilation system shafts as major design elements.

Openings in the building shell

The number and location of openings in the building shell affect the ability to provide thermal comfort and control air contaminants. Section 28.3 of this chapter discuss how to evaluate the building site and identify potential sources of outdoor air contaminants. The locations of operable windows (if any), doors, and outdoor air intakes should reflect prevailing wind patterns and contaminant sources outside the building.

Naturally-ventilated buildings use openings in the building shell such as windows, doors, and skylights to supply outdoor air and cause air to circulate. This strategy can be used in almost any type of building, but is most practical

in buildings that are relatively narrow. Operable windows can significantly enhance the occupants' sense of well-being.

However, the potential for frequent use of operable windows in a mechanically ventilated building imposes additional design considerations on the HVAC system. Even under ideal circumstances, it is difficult to maintain good control over natural ventilation under all climate and indoor thermal conditions. ASHRAE Standard 62-1989 states that, if natural ventilation is insufficient to meet design requirements, mechanical ventilation must be provided. Many buildings therefore rely exclusively on mechanical ventilation.

In some situations a sealed building can provide better IAQ than a building with operable windows. Uncontrolled infiltration allows outdoor air contaminants to bypass filters and air cleaning equipment; it can also disrupt the balance of the mechanical ventilation system. This is not to suggest that natural ventilation is undesirable or impossible. In certain climates and for certain building types, natural ventilation may be very effective and comfortable.

Thermal characteristics of the building shell

The ability to provide thermal comfort is affected by the size, location, and orientation of windows and skylights. Radiant heat gains and losses through glass (and other uninsulated surfaces) complicates HVAC zone design. In addition, glare from windows and skylights can produce health symptoms and complaints.

Thermal insulation not only reduces energy consumption, but also affects the surface temperature of the building interior. In cold climate zones, walls or ceilings may have "cold spots" where structural members or other factors produce voids in the insulation. Condensation on the cold surface can lead to mould or mildew growth, even though the relative humidity of room air remains within the range prescribed by ASHRAE 55-1981.

Spatial relationships between functional areas

During programming, the designer and client discuss the intended use(s) of the building, types and probable timing of activities that may generate odours or contaminants, and any special needs of the anticipated occupants. Decisions about the layout of functional areas within the building can simplify or complicate the work of the HVAC system designer. Factors to consider include: isolating the odours produced by different activities, providing adequate space and service access for mechanical equipment, and locating storage areas for food, rubbish, and chemicals to facilitate good sanitation and minimize hazards from spills.

Mixed occupancies can present a particular challenge for mechanical designers. For example: An office building also contains a beauty salon and a

cafeteria. To confine odours, both businesses are maintained under negative pressure with respect to the central corridor of the building. The exhaust rate in the beauty salon remains constant; however, the exhaust rate in the cafeteria kitchen changes as staff operate the hoods over various cooking appliances. When all of the kitchen exhaust fans are operating, the cafeteria is at significantly lower pressure than the beauty salon. If the two businesses are located near each other (particularly if they share a common wall), odours from the beauty salon could be drawn into the cafeteria.

28.4.5 Environmental control scheme

The environmental control scheme is critical to meeting IAQ criteria. As shown in Table 28.9, thermal comfort is affected by the size and layout of HVAC zones, the capacity of heating and cooling equipment, and humidity control. Outdoor air ventilation needs are addressed by selecting appropriate outdoor air ventilation rates and providing effective air distribution. Indoor contaminant levels are minimized with appropriate air filtration/air cleaning equipment, control of building pressures/use of local exhaust, and careful location of HVAC components.

A more complete discussion on HVAC will be found in Chapter 29.

HVAC zones

The area regulated by a single thermostat is referred to as a temperature control "zone". The term "zone" is also used to describe the areas served by individual pieces of equipment such as air handling units. Temperature control zones vary greatly in size, depending on the building design and usage. A single zone can encompass several rooms, or even an entire building. On the other hand, a single room can contain more than one zone.

The use of multiple zones increases the ability to accommodate individual preferences and to compensate for changing thermal loads. Spaces bounded by exterior walls or ceilings may require either heating or cooling as the HVAC system compensates for the indoor-outdoor temperature difference, the effects of solar gain, and internal heat gains from lighting, equipment, and the human occupants. Interior ("core") spaces generally require year-round cooling. If two rooms have very different thermal loads due to differences in the number of occupants, the amount of heat-generating equipment, exterior wall area, or window orientation, they cannot be well served by a single thermostat.

To function properly, a thermostat must be able to sense the temperature in the space it is intended to regulate. Avoid placing them in direct sunlight, against exterior walls, or near heat-generating equipment.

Thermostats are sometimes installed in the return airstream to avoid

occupant interference. If this strategy is used, it is critically important to provide good air mixing in the occupied space.

Heating / cooling equipment capacity

Heating and cooling equipment capacity is selected with the goal of maintaining comfortable indoor temperatures under extreme ("design") outdoor conditions. Heating equipment is relatively easy to select by combining weather information with data on the thermal resistance of the building shell and projected outdoor air ventilation rates. It is common to select heating equipment with excess capacity as a "safety factor".

The process of sizing cooling equipment is more complicated. The cooling plant must accommodate both the sensible heat load (the need to lower the dry-bulb temperature of the air) and the latent heat load (heat generated by condensation as moisture is removed from the air). The sensible heat load is the dominant factor in hot, dry climates, but in hot, humid areas the latent heat load may be more important. The additional latent heat load that results when airflows are increased to meet ASHRAE 62-1989 could make it difficult to provide indoor comfort with conventional HVAC equipment. Increasing the total cooling capacity without considering the additional humidity might result in indoor moisture problems such as mould growth and potential damage to the building structure and interior finishes. Various approaches are available that can dehumidify the air before it is cooled, including heat recovery ventilation that transfers both sensible and latent heat from the incoming airstream to the outgoing airstream.

Humidity control

Indoor humidity should be maintained within the comfort limits defined in ASHRAE Standard 55-1981. Microorganism growth can be a problem if surface condensation occurs due to high relative humidity or cold surface temperatures. ASHRAE Standard 62-1989 recommends that humidities should be kept low enough throughout the building so that water does not routinely condense anywhere except on the cooling coil. Insulation can be installed on cold water piping and other cold surfaces to prevent condensation.

Humidification may be needed in cold climate zones to prevent winter complaints of dry skin and mucous membranes. Indoor relative humidities as low as 10% have been reported in Northern regions. Steam is preferred as a moisture source in humidifiers, but care should be taken to avoid contamination from additives to boiler or steam water supplies. Cold water humidification should only use water from filtered and de-ionized potable sources. Avoid particulate contamination from water sprays.

TABLE 28.13

Various recommended and adopted ventilation rates

(L/s-p)*	CFM/p	Basis or recommending/adopting group and year
>0.3	>0.6	2% CO_2 (respiration)
>0.5	>1.0	1% CO_2 (performance)
>1	>2.0	0.5% CO_2 (TLV)
>3.5	>7.0	0.15% CO_2 (Pettenkofer Rule, 1858; body odour)
2.5	5.0	ASHRAE Standard 62-1981
3.5	7.0	Swedish Building Code 1980
4	8.0	Nordic Building Regulation Committee 1981
5–7	10–15	Berglund et al. (body odour)
8	16.0	Fanger et al. (body odour)
7.5–30	15–60	ASHRAE Standard 62-1989
5–10	10–20	Swedish Building Code 1988
10–30	20–60	Swedish Allergy Committee 1989
10, 20	20–40	Nordic Building Regulation Comm., preliminary 1989
16–20	32–40	Weber et al., Cain et al. (tobacco smoke annoyance)
14–50	28–100	Fanger et al. (total odour)

*1 litre per second per person – 2 cubic feet per minute per person.

Outdoor air ventilation rate

Table 28.13 illustrates the wide range of ventilation rates recommended by various agencies and researchers to meet a variety of specific criteria. Remember the criteria used in developing ASHRAE 62-1989. Its recommended minimum ventilation rates are intended to maintain an acceptable level (i.e., >80% of occupants will rate air quality as "acceptable") of occupant comfort when no unusual sources of contaminants are present. The ASHRAE standard assumes that all outdoor air supplied reaches the occupied zone and mixes well with the room air.

ASHRAE's recommended ventilation rates have evolved over time, reflecting changes in society as well as in construction materials and mechanical systems. Outside air is essential to dilute contaminants, and early standards were largely based on maintaining body odour at an acceptable level. In 1973, concern over energy conservation prompted ASHRAE to reduce its recommended minimum rate to 5 cubic feet per minute (cfm) per occupant. However, it was found that 5 cfm per occupant generally did not provide an adequate outside air supply to achieve comfort and avoid IAQ-related complaints. Under certain conditions, contaminant levels were found to rise above accepted or

recommended levels when outside air supply levels were less than 15 cfm per occupant. ASHRAE Standard 62-1989 increased minimum outside air ventilation rates from 5 to 15 cubic feet per minute (cfm) per occupant.

Higher design ventilation rates tend to increase system capacity, energy consumption, and associated costs. It is important to achieve a balance between energy conservation and indoor air quality. However, analysis of the energy and capital costs associated with increasing ventilation rates from 5 to 15 cfm/person have shown that for most occupancy types the increases in annual costs are fairly small, approximately 5–10%, even in the most extreme climate conditions studied (Minnesota and Florida) (Eto and Meyer, 1990).

The use(s) of the building, the contaminant sources, and the needs and expectations of the owner and occupants all affect the requirements for outdoor air ventilation. Consider factors such as: occupant density; health status of occupants; susceptibility of the occupants to odours, allergens, irritants, and toxins; the scheduling and duration of occupancy; the type of activities taking place in the space; the thermal environment; and air supply distribution and circulation patterns. Incorporate flexibility into the ventilation design to account for factors that are likely to change over the life of the building.

By establishing ventilation rates without regard to the type and number of contaminant sources present, the ASHRAE standard places the burden on the designer to identify potential sources and provide for their control. Any point sources of pollution such as copiers, cooking areas, or other obvious sources should be directly exhausted to the outdoors. This principle is reflected in building codes for exhaust from toilet rooms.

Provide adequate make-up air for fuel-burning appliances, clothes dryers, and exhaust fans. In buildings with low occupant density and large quantities of powered exhaust (e.g., laboratories), the requirement for make-up air may exceed the outdoor air ventilation rates recommended in ASHRAE Standard 62-1989. Note that combustion appliances, clothes dryers, and exhaust fans should be vented to unrestricted outdoor air, not into attics, crawl spaces, or basements.

Table 28.14 translates ventilation rates from cubic feet per minute per person into air changes per hour for three ceiling heights: 11.5 ft, 9.75 ft, and 8 ft. The table is based on an assumption of 7 persons per 1000 ft^2 of floor area. Concealed spaces above suspended ceilings should be included in the height calculation if they are used as return air plenums. Interior space occupied by solid elements (such as furnishings, equipment, floors, and walls) that reduce the amount of air volume should be considered in modifying the volume downward. A general figure for reduction might be 10% less than the total floor to ceiling height, plus the volume above the ceiling if applicable.

TABLE 28.14

Ventilation levels in air changes per hour

Ventilation per person		Air exchange rate (ach)		
CFM	L/s	Ceiling height		
		11.5 ft	9.75 ft	8 ft
35	17.5	1.26	1.52	1.80
20	10.0	0.72	0.87	1.03
15	7.5	0.54	0.65	0.77
10	5.0	0.36	0.45	0.52
5	2.5	0.18	0.21	0.26

Air filtration / air cleaning equipment

Air cleaning systems should address both contaminants generated outdoors and contaminants that originate inside the building. Media filters or electrostatic precipitators are used to remove particles, while adsorbents or scrubbers are used to remove gases. Some air cleaning devices combine these technologies (e.g., by embedding activated charcoal in the filter media); however, this discussion will consider the treatment of particulates and gaseous pollutants as separate issues.

Particle filtration can be an effective prevention tool, cleaning the outdoor air supply and return air before it circulates through the HVAC equipment and is distributed to the occupants. Properly designed and maintained filters enhance the health and comfort properties of indoor air and the occupants' perception of indoor air quality. Effective filtration is essential to maintain HVAC system performance (EPA, 1990).

Acute health effects from exposure to elevated particle levels include irritation, allergies, and infection. In addition, some particles are carcinogenic (e.g., some of the substances contained in environmental tobacco smoke). Particles deposited in the HVAC equipment can soil or clog coils, reducing performance efficiency and airflow, increasing operating costs, and potentially damaging HVAC components. Particles circulated by the air distribution system cause soiling and staining of the building contents, increasing housekeeping costs.

Sub-micron particles (<1 μm in diameter) comprise only a tiny fraction of the total particulate matter mass, but constitute the overwhelming majority of the total number of particles. Although larger particles cause soiling far

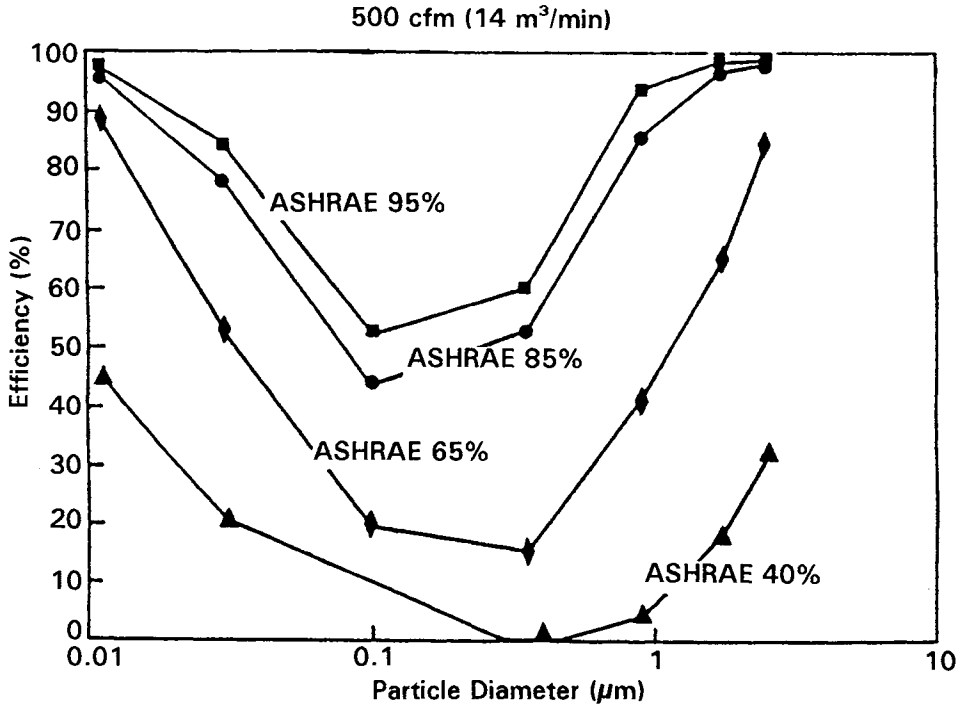

Fig. 28.12: Particle-size-dependent efficiency for various grades of ventilation filters.

more rapidly, fine particles are most capable of penetrating deep into the human respiratory tract where they can do the greatest health harm. Therefore, removal of particles in the 0.1–1.0 μm range is most important from a health perspective. Figure 28.12 shows the performance of rated particle filters for particles from 0.01 to 10 μm in diameter. As can be seen, even filters rated at 85 or 95% efficiency remove only about 45–55% of the particles in the 0.1–0.3 μm diameter range.

The concentration and toxicity of fine particles varies depending on the source. Tobacco smoke particles are small and hazardous while some fine particles from outdoor sources may not be as toxic. Some bacteria are in the size range less than 1 μm in diameter. The filtration method shown in Figure 28.13 can be very effective for fine particles if the collecting sections of the electrostatic precipitator are frequently cleaned. An electrostatic precipitator must be carefully designed and maintained to avoid generation of excess ozone that can reach the occupied space.

The alternative method for removing fine particles is with high efficiency particle arrestance (HEPA) filters. HEPA filters are used for clean rooms and other sensitive applications, but they are costly and impose a significant

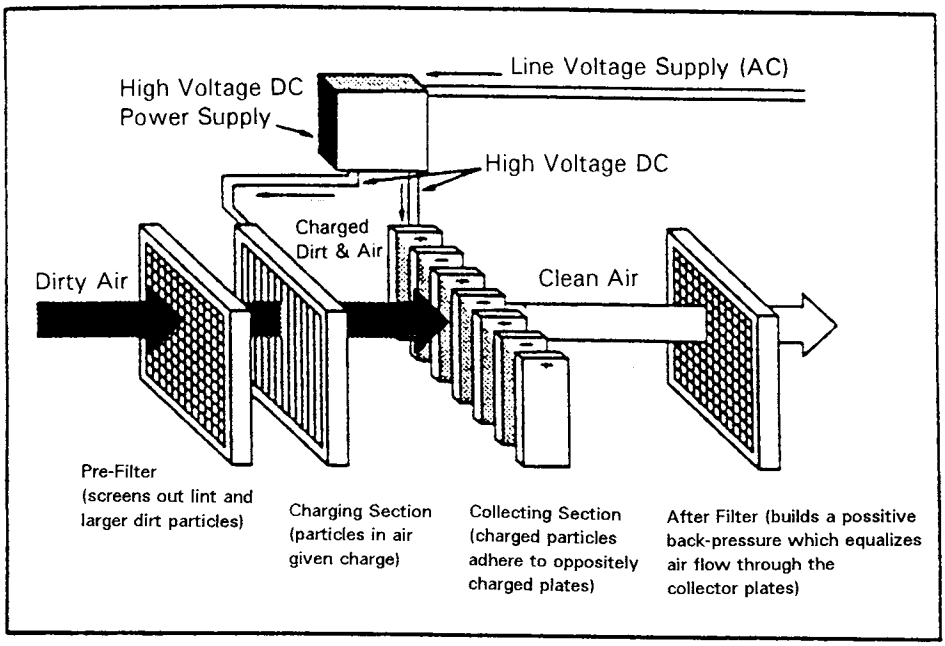

Fig. 28.13: Air cleaning and filtration by combination electrostatic precipitation and media filters.

pressure drop on the system. Thus, larger fans and additional energy to operate them are required to overcome the additional resistance imposed by HEPA filters. For this reason, they are not normally used in commercial applications. Extended surface filters provide improved efficiency while minimizing the increase in pressure drop.

Filter installations should be designed to minimize by-passing and facilitate filter inspection and replacement. While these may be obvious concerns, they are frequently overlooked during design. Failure to address them adequately results in significant deterioration of indoor air quality. Specifications should indicate the desired rating rather than relying on general terminology such as "medium efficiency" or "high efficiency". Evidence suggests that increased filter efficiency is a worthwhile investment to protect human health and building systems. In anticipation of this trend, consider upgrading to at least medium-efficiency filters, and design the system to handle higher efficiency filters in the future.

One may be able to design for lower outdoor airflows by using air cleaning equipment and following the "indoor air quality" procedure described in ASHRAE Standard 62-1989. Systems must be properly designed specifically for this purpose if it is to be accomplished. The trade-offs between cleaning and

recirculating return air and conditioning and circulating outside air vary greatly from case to case. The critical design factors are the thermal and humidity conditions and contaminant contents of both the outdoor air and the return air relative to the design conditions. The limiting gaseous or particulate contaminant not removed by the air cleaning system (sometimes CO_2) then becomes the basis for the minimum outdoor air flow rate.

The design and maintenance of air cleaning systems for the removal of gaseous contaminants should be carefully considered on a case-by-case basis. Air cleaning is typically considered a less practical approach to control of gaseous contaminants than either source control or ventilation, and may not be practical for residences, offices, and schools. However, sorbents or scrubbers may achieve an additional reduction in the levels of certain gases when source control and ventilation do not result in acceptable pollutant concentrations. Activated carbon is a commonly-used sorbent. Other useful sorbents include specially treated carbon or potassium permanganate. Vendors have made useful design information available to design professionals to select appropriate media.

Air circulation patterns and pressure relationships

No matter how effective are efforts to minimize indoor pollutant sources, IAQ depends in great part on airflow patterns into and within the building. These airflow patterns are affected by openings in the building shell and other architectural features. However, the design, construction, and operation of the HVAC system are the dominant influences controlling air circulation in most non-residential buildings.

ASHRAE Standard 62-1989 states that ventilation systems must supply air throughout the occupied zone. Whether a building is mechanically or naturally ventilated, the ventilation system design should be developed with a clearly articulated scheme of pressure relationships and overall system balance. Air movement from one compartment to another will depend on pressure differences and available flow paths.

Early in the design process, develop an analysis of the airflow into, through, and out of the building. This can begin with a simple conceptual diagram similar to the drawing in Figure 28.14. The airflow analysis should show the following items:

- HVAC system intake and exhaust locations;
- other air entry and exit points in the building shell;
- the path of the air through the system, into the spaces, and back to the mechanical equipment;
- air circulation in each typical space taking into account the type of furnishings and partitions that will actually be used.

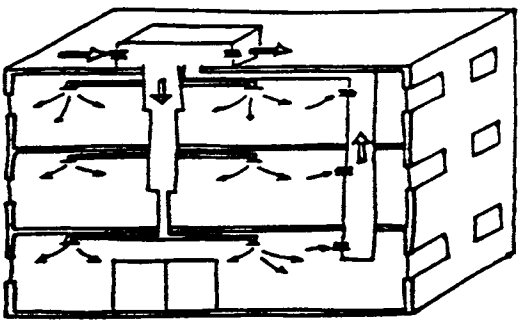

Fig. 28.14: Ventilation conceptual plan.

As the design is refined, the diagram may be expanded to show potential flows between contiguous spaces, through floor and wall penetrations, and from floor to floor. This design tool will facilitate careful consideration of pressure-driven airflows within the building that can often cause contaminant transfer (EPA, 1991).

Consider pressure relationships as you plan the airflows into and out of each space and between the building and outdoors. The basic flow scheme and actual pressure differences may have a greater impact on indoor air quality than the specific ventilation system components or air flow quantities that are chosen. If the entire building is designed to run positive (to prevent the entry of unconditioned, unfiltered air), the total outdoor airflow must exceed the total quantity of air that is withdrawn by powered exhaust, wind, and the stack effect. In addition to meeting the ventilation requirements spelled out in ASHRAE Standard 62-1989, the flow of outdoor air into the building should be sufficient to replace the air removed by exhaust fans and provide combustion air for appliances.

Consider the air pressures in unoccupied spaces as well as occupied areas of the building. If the air pressure in crawlspaces, basements, or underground ducts falls below the air pressure in the soil, radon and other soil gases could be drawn into those spaces and into the building. If ductwork is located in unconditioned spaces, leaks in supply ducts waste energy and complicate pressure management, while leaky return ducts could draw in contaminants.

The designer must provide for proper air distribution and mixing in each space by planning the locations of supply diffusers, return or exhaust registers and grilles. Supply air must be delivered to the occupants to be effective. Do not assume that all the air passing through the diffuser is delivered to the occupants' breathing zone. Short circuiting can occur if supply air is drawn into return or exhaust grilles before it has mixed with room air (note Fig. 28.15).

Return air is often routed through grilles or slots in the ceiling to return air

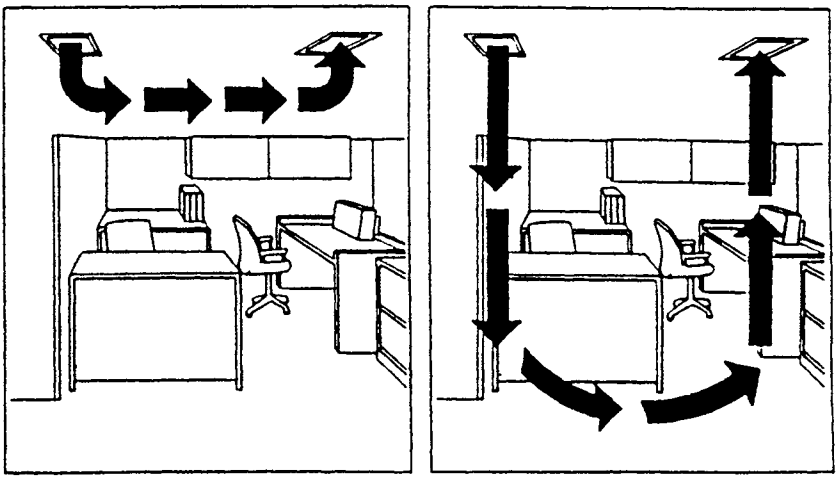

Fig. 28.15: Short circuiting.

plenums in the concealed spaces above suspended ceilings. Unducted returns complicate the task of balancing the system, adjusting supply, return, and exhaust quantities to meet design requirements. Unbalanced airflows produce uneven air distribution and can increase the potential for dead spots, stagnant air, or undesirable pressure relationships. Whenever possible, use ducted returns and distribute return air registers throughout the ventilated space.

Different interior layouts, ceiling heights, and operating conditions affect the delivery of supply air to the occupants' breathing zone. Supply air distribution is influenced by the location of supply and return (or exhaust) openings, the temperature and velocity of supply air, the "throw" of the diffusers, and by return and exhaust flow rates. Analyze the impact of air supply and return locations on air flows in typical and unique spaces to determine whether short circuiting, dead zones, or other space air distribution problems might occur (see Figs. 28.19 and 28.20). If there is concern about space air distribution, use physical models (either at reduced or full scale) to determine design air flows.

Supply air temperature affects distribution as warm air rises and cold air falls. Therefore, typical ceiling and high wall mounted diffusers work better with cold air than warm air to achieve good mixing. In variable air volume distribution systems, when supply volumes are minimal or low, there may be insufficient velocity (force) to create complete mixing.

Air mixing is affected by obstacles such as partitions, as illustrated in Figure 28.16. The layout of interior partitions and furnishings may not be known at the time of the overall HVAC design. The use of flexible duct (where permitted) enhances the ability to respond to changes in layout; however, the

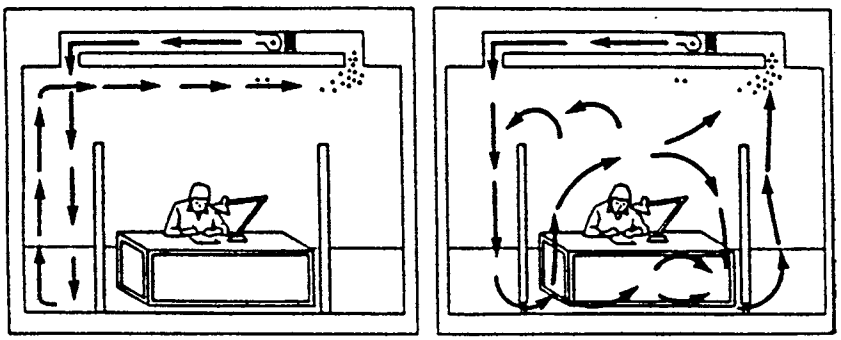

Fig. 28.16: Office air circulation.

system design (including fan selection) must reflect the increased pressure drops associated with flex duct. The base building HVAC designer should, therefore, require a detailed program statement that defines the range of possible occupant densities, activities, and layouts. Make this program statement part of the building design documentation, and document design assumptions and decisions accordingly. If changes are made in the original program when the local distribution system is designed, corresponding changes may be required in the base building HVAC system.

In Figure 28.16, air in the office on the left does not adequately reach the occupant. In the office on the right, raising partitions off the floor presumably improves circulation. Studies have not demonstrated that air circulation is significantly improved simply by raising partitions without other modifications to improve supply air distribution.

Figures 28.17 A,B,C, and D illustrate basic concepts of air distribution relating to contaminant control. Contaminants from stationary local sources should be exhausted as close to the source as practical. The control concept shown in Figure 28.17A is the best method, because the exhaust duct is connected directly to the machine and the contaminated air is be exhausted directly outdoors. The design shown in Figure 28.17B is slightly less desirable, although the exhaust inlet is very close to the machine (i.e., the source being controlled). In Figure 28.17C, the source is at least partially isolated from the rest of the space. Figure 28.17D shows the least desirable design, with minimal direct control of the machine's emissions.

28.4.6 Materials evaluation and selection

Building materials and furnishings can be important sources of indoor air contaminants. Designers can reduce indoor air pollutant levels by carefully

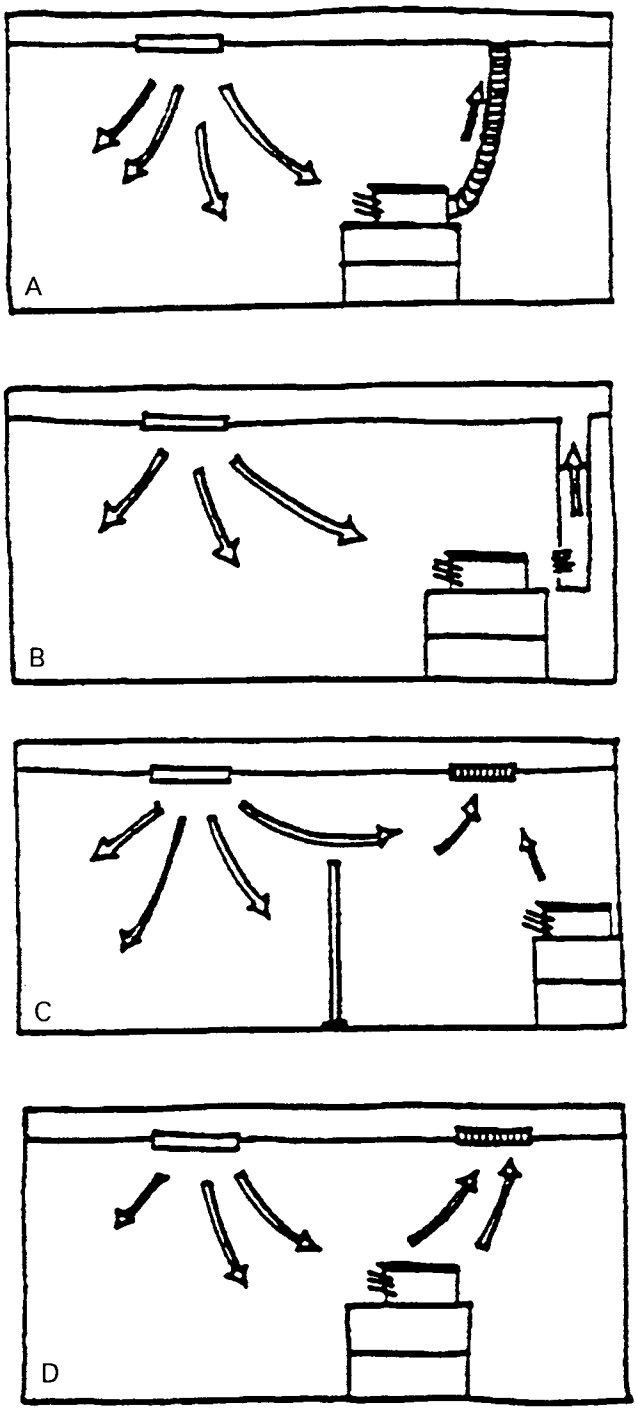

Fig. 28.18A–D: Control concepts. These four figures illustrate some possible control concepts where a photocopier is placed in an open office area. "A" is best.

selecting materials to minimize the potential for contamination from the following processes:

1. Emission of chemicals that are used or formed in the production of building materials and furnishings.
2. Release of particles from materials surfaces into the air.
3. Growth of microbial contaminants on material surfaces, especially moist surfaces.
4. "Sink effect" — chemical molecules in the air can adsorb (physically attach) to material surfaces (known as "sinks"), from which they may later be released into the air.
5. Maintenance and refinishing requirements of materials determine the need for additional chemicals that may become indoor air contaminants.

Designers can reduce the emission of chemicals that are used or formed in the production of building materials and furnishings by discussing their indoor air quality concerns with manufacturers and suppliers and selecting the lowest-emitting materials and processes available for each class of product. "Wet-applied" materials such as caulks, paints, and adhesives are of particular concern.

Particles may be released because binders or adhesives used to manufacture products deteriorate over time. Environmental factors such as moisture, heating, ultraviolet radiation from sunlight, and mechanical abrasion can all contribute to particle release. Certain particles are especially troublesome because they are irritants or even carcinogens. The massive effort to control asbestos and lead in buildings illustrates the importance of considering particles as a potential source of indoor air contamination. Fibreglass used in thermal insulations can be a skin irritant and causes itching in many people, so careful installation is warranted.

Biological contaminants such as fungi, bacteria, and viruses require favourable conditions to survive and multiply. Moisture and nutrients are essential. Moisture control is very important, because any carbonaceous material can provide the necessary nutrients. Special attention to material selection is warranted where high air moisture levels are expected (e.g., in kitchens, showers, or downstream from cooling coils in air handlers). In these high risk areas, easily-cleanable, smooth surfaces are recommended. Where insulations are used (as in air handlers for noise control), care should be taken to protect fibrous materials from soiling, as soiled fibreglass will take on moisture much more rapidly than clean fibreglass. This can be accomplished at least to some degree by adequate filtration of circulation air.

The potential for a material to act as a sink is a function, in large part, of the amount of surface area exposed to the circulating indoor air. Therefore, fabric upholstery, textile wall coverings, carpets, and other "fleecy" materials

should be used only when necessary for aesthetic or acoustic purposes. When used, attention should be given to the potential for sink effect, and ventilation should address the need to control airborne chemical concentrations.

Evaluating materials as chemical pollutant sources

Organic chemicals emitted into the indoor air after renovation or new construction are often blamed for sickness, irritation, and discomfort among building occupants. Most of the chemicals of concern are either volatile organic compounds (VOCs) or semi-volatile organic compounds (SVOCs). Much can be done to reduce building occupants' exposures to organic chemical emissions from building materials and products. Control measures include careful planning, specifications, selection, modification, and treatment of products as well as special installation procedures and proper ventilation system operation.

Materials evaluation during product selection

Materials evaluation is a valuable design tool for reducing occupant exposure to VOCs and SVOCs. The steps of the process are as follows:

- Identify target products that may be significant sources of emissions based on:
  - quantity: even products with relatively low emissions per unit area can be important sources of contaminants if used in quantity. Carpets and particleboard work stations are examples;
  - location: all other things being equal, occupants are most likely to be affected by nearby materials (e.g., work surfaces);
  - toxicity: some chemicals (e.g., pesticides) are much more toxic than others;
  - potential emission rates: different products that serve the same function may have dramatically different emission rates. In addition, emission rates for many materials vary over time and depend on temperature and humidity (see also Chapter 24).
- Review and evaluate manufacturers' test results, supplemented by additional emissions testing if necessary.
- Develop recommendations and specifications:
  - recommend products and installation procedures that minimize emissions while meeting performance requirements. Negotiate with product manufacturers, suppliers, and installers to modify products or their installation and use;
  - develop ventilation specifications to minimize adverse effects of contaminants on occupants, building contents, and the building fabric. Consider airing out the building before occupancy(see below).

Design professionals can require manufacturers to provide information

about chemical composition, emissions, and indoor air quality impacts of their products. Manufacturers are in the best position to evaluate their products and have both marketing and liability motivations to do so. Standardized test procedures are evolving, and many manufacturers are becoming accustomed to requirements that they submit the requested information. Architects, interior designers, and engineers cannot assume responsibility for overseeing the tests or interpreting the results, but should take responsibility for ensuring that the manufacturer has done so. Designers can also be responsible for selecting the lowest emitting products and installation techniques among the available alternatives for each class of materials.

Table 28.15 shows the categories of building materials that are typically considered as impacting indoor air quality. Chapters 2 and 24 provide an extensive discussion of these materials.

TABLE 28.15

Typical materials of concern, especially in office buildings

Site work and foundations	Insecticides and other soil treatments Waterproofing, particularly petroleum derivatives
Structure and envelope	Wood preservatives Concrete sealers, curing agents Caulking Sealants Joint fillers Glazing compounds or gasketry
Insulations	Thermal insulation Fire proofing Acoustic insulations
Interiors and finishes	Subfloor or underlayment Flooring or carpet adhesive Carpet backing or pad Carpet or resilient flooring Wall coverings Adhesives Paints, stains Panelling Partitions Furnishings Ceiling tiles
HVAC systems	Duct insulations Duct sealants Chemical water treatment

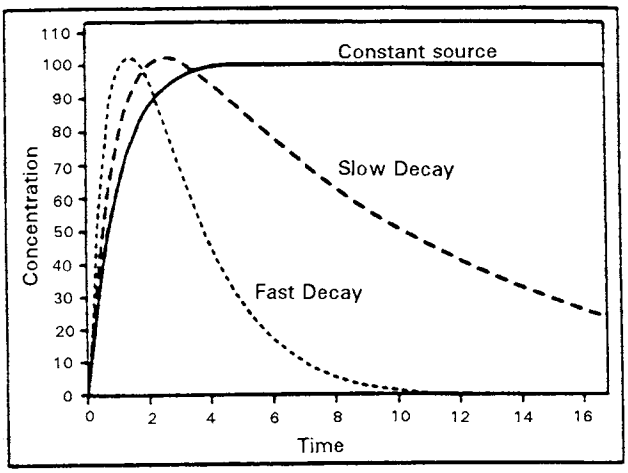

Fig. 28.18: Decay curves.

Figure 28.18 shows emission curves for three basic pollutant source types classified by the duration and course of their emissions. Constant sources are exemplified by items such as moth crystal, which are intended to emit a constant quantity of VOCs during their useful life. Slow decay sources are exemplified by particleboard or preservative-treated wood, which release formaldehyde and preservatives (respectively) at slowly decreasing rates, but for many months or even years after they are installed in a building. Fast decay is characteristic of products like paints and adhesives that are installed in a wet state and are expected to emit the majority of their emissions during the first few hours or days of their lives.

Figure 28.19 shows the effect of different ventilation rates on the emissions of VOCs from a caulking compound. The emissions decay much more rapidly with a higher ventilation rate (1.84 ach). This means that increased ventilation would be useful if a space is to be occupied shortly after use of the caulk. Figure 28.20 shows that much of the chemical emissions are deposited on the surface of the chamber, especially when the emissions are strong immediately after application of the caulk. This lowers peak concentrations immediately after the caulk is applied. The deposited chemicals are later re-emitted, effectively extending the period of potential exposure. Note that the cumulative emissions are the same, regardless of the ventilation rate or the sink effect; only the shape of the curves change.

Available emissions data for building materials are not comprehensive, nor are they necessarily comparable among various sources. Nevertheless, where available, these data can be helpful in determining the need to more carefully

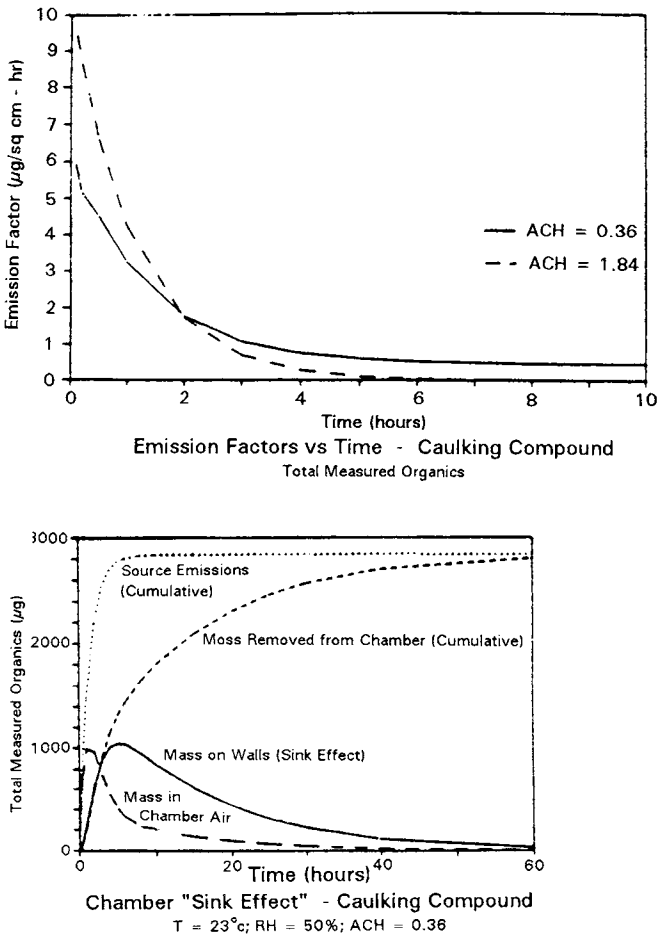

Fig. 28.19: Environmental chamber tests of emission rates. (A) Effect of different ventilation rates on emissions. (B) The "sink effect" — chemicals are adsorbed on chamber walls and then re-emitted. Source: Tichenor (1987).

evaluate products or obtain more information from manufacturers. Table 28.16 presents some representative data. Additional data are available in Chapter 24 (Levin, 1989, 1991; Tucker, 1988).

Test data on emission rates or source strengths of building products and materials are useful when making decisions regarding product selection and use. The information is also useful when prescribing ventilation system operating protocols to maintain acceptable IAQ and when assessing complaints associated with IAQ problems. For example, it might be possible to select materials with fast decay curves (taking particular care to protect workers from high level exposures during construction), use increased ventilation to

TABLE 28.16

Typical emission rates for sources in a 400 m^2 office area. Source: Tucker (1988)

Source*	Condition	Emission factor ($mg/m^2 \cdot h$)**	Assumed amount (m^2)	Emission rate (mg/h)
Silicone caulk	<10 hours	13	1	13
Silicone caulk	10–100 hours	<2	1	<2
Floor adhesive	<10 hours	220	30	6600
Floor adhesive	10–100 hours	<5	30	<150
Floor wax	<10 hours	80	100	8000
Floor wax	10–100 hours	<5	100	<500
Wood stain	<10 hours	10	100	1000
Wood stain	10–100 hours	<0.1	100	<10
Polyurethane wood finish	<10 hours	9	100	900
Polyurethane wood finish	10–100 hours	<0.1	10	<10
Floor varnish or lacquer	NA	1	100	100
Particleboard	2 years old	0.2	300	60
Particleboard (HCHO)	new	2	300	600
Plywood panelling (HCHO)	new	1	1000	1000
Chipboard	NA	0.13	300	39
Gypsum board	NA	0.026	1000	26
Wallpaper	NA	0.1	1000	100
Latex-backed carpet (4-PC)	1 week old	0.15	400	60
Latex-backed carpet (4-PC)	2 weeks old	0.08	400	32
Moth cake (para)	23C	14,000	0.1	1400
Dry-cleaned clothes (perc)	0–1 day	1	6	6
Dry-cleaned clothes (perc)	1–2 days	0.5	6	3

Para = paradichlorobenzene; HCHO = formaldehyde; perc = perchloroethylene(tetrachloroethylene); 4-PC = 4-phenylcyclohexene, an odorous constituent of some latex-backed carpets; NA = not available.

* Emissions data shown are typical only for the specific brands, models, or units that have been tested; the data do not represent all products of the source type listed. Product-to-product variability can be very high.

** Typical values selected by author based on data in "Database of Indoor Air Pollution Sources."

further accelerate the decay process, and delay the installation of fleecy materials until after the bulk of VOCs have been emitted from the materials and removed from the building. Information on emission rates and decay curves can also be useful in negotiating with manufacturers and suppliers to

minimize VOCs through pre-shipment storage practices and modified installation procedures.

Several factors limit the use of product selection to protect occupants from the toxic or irritating effects of VOCs and SVOCs. Not enough is known about the health and irritation effects of very low exposures. Low emissions of one compound may be more harmful than higher emissions of an alternative compound. In some cases, occupant activities or other sources outside the designer's control may be more important than product emissions as sources of VOCs.

Special procedures: ventilation and "bake out"

Special procedures may prevent or remedy IAQ problems that result from material emissions. Many office buildings have used special ventilation protocols such as increased ventilation during material installation and initial building occupancy in order to reduce residual airborne VOC concentrations during the early occupancy period.

An interesting approach known as a "bake out" has been used to accelerate the emissions of chemicals from newly installed materials, products, and furnishings. In this approach, the unoccupied building is heated to temperatures above 90°F for more than 24 hours, maintaining the maximum outdoor air ventilation that allows this elevated temperature. Then, the building temperature is lowered to the designed occupancy temperature, and the building is ventilated with maximum outdoor air for at least 24 hours before normal occupancy. The longer the period of elevated temperature and the higher the temperature achieved, the more effect the procedure is likely to have. The increased vapour pressure drives the chemicals of concern out of building materials and into the building air, from which it is removed by the increased ventilation (Girman et al., 1990; Levin, 1992).

This procedure is not completely validated. It is preferable to avoid the need for "bake-out" by using low-emitting materials, proper ventilation during construction, and thorough commissioning before occupancy. If "bake-out" is used, caution should be exercised to avoid damage to the building structure or contents during the bake-out as a result of excessive temperature or changes in humidity.

28.5 THE CONSTRUCTION PROCESS

Monitoring the construction process with an eye to IAQ will improve IAQ in the finished building. This chapter discusses five topics that require attention during construction:

- field orders, shop drawings, and change orders: monitor deviations from the construction documents;
- progress inspections: observe construction progress in the building regularly with an eye to IAQ;
- control of construction contaminants: use temporary ventilation during installation of pollutant sources such as carpeting and furnishings;
- commissioning: implement a thorough commissioning plan for the HVAC system and the building envelope;
- initial occupancy: use special ventilation protocols before and during the initial occupancy period.

The construction and commissioning process translates the design into reality. Few, if any, buildings are constructed precisely as they were designed. Documentation during construction, commissioning, and initial occupancy should record the progress of the project, any departures from the original design, and any events that might be expected to affect indoor air quality in the completed building. It should also include balance reports and other test results from the pre-occupancy and immediate post-occupancy period.

28.5.1 Documentation during construction and commissioning

IAQ-related documentation during construction and commissioning should include:

- submittals and shop drawings;
- description of temporary ventilation and other measures undertaken to minimize contaminant levels during construction;
- records of progress inspections;
- records of spills and responses;
- IAQ-related change orders, product substitutions, and other departures from the original design;
- "as-built" architectural and mechanical plans;
- test and balance reports;
- description of "bake out" procedure and any accompanying test results.

28.5.2 Field orders, shop drawings and change orders

Architects and their consultants should review, evaluate, and follow-up on contractor submittals and shop drawing approval requests as well as change orders and field orders. Many items covered by these documents, including HVAC system components, insulations, sealants, finish materials, and fur-

nishings, have a significant impact on indoor environmental quality. The architect and HVAC consultant should make a list of items requiring special attention with respect to IAQ, notify the appropriate parties, and monitor implementation during construction.

Changes made by contractors or designers during construction can significantly affect IAQ. These changes are often in response to previously unanticipated problems or events during construction. During the change order and shop drawing approval process, ensure that any changes meet the design intent and the performance criteria that have been established.

28.5.3 Progress inspections

Remember IAQ performance goals and criteria during all field visits and progress inspections. Architects can identify critical components to be inspected during construction and develop a plan of construction site monitoring or quality control related to indoor air. One critical element is the use of specified products that are important for air quality such as HVAC system components, finishes, insulations, composite wood products, and others. See Table 28.15 for a list of significant materials.

Check ventilation system air flow paths to be certain that there are no unexpected obstacles to proper air distribution, especially in concealed spaces such as shafts and return-air plenums above dropped ceilings. Check the locations of critical elements such as thermostats to see that they are not improperly located (e.g., in direct sunlight, above a heating element or a heat-emitting appliance, in an inaccessible location, or in a zone outside the area served by the air handling unit the thermostat controls).

Air supply and return system testing, adjusting, and balancing work should be monitored and verified as it occurs. This work is one of the most critical elements of the construction process; it makes an integrated system out of a collection of independent components. Evaluating individual components or subsystems does not ensure that they will operate properly when the HVAC system and building envelope are completed and the building is occupied.

Normal testing, balancing, and adjusting of the HVAC system does not reliably estimate the delivery of air or removal of pollutants in the breathing zone, nor does air balance work verify the performance of the system under heating or cooling load. Architects should require that system performance be tested under part and full load and that outside supply air ventilation in the breathing zone be measured as part of the HVAC testing and final approval process. This is covered in more detail in the discussion of Commissioning in Section 28.5.5.

28.5.4 Control of construction contaminants

Inspect temporary ventilation systems to confirm that they are in place and operate properly. The operating hours and air volumes should be sufficient to prohibit excessive contaminant build-up in enclosed spaces. Check pressure relationships to ensure that occupied spaces are isolated from construction areas. Specify installation of suitable physical barriers to achieve isolation of the contaminant generating areas.

Review contractors' reports of chemical and fuel spills and inspect contaminated areas. In some cases, building materials or furnishings that have been contaminated by spills must be removed and replaced.

28.5.5 Commissioning

The commissioning process involves comprehensive planning, thorough documentation, and systematic implementation. Proper commissioning means building it right in the first place and making sure that it works right before occupying the building.

The costs of discovering and remedying HVAC system defects after occupancy are substantial. In addition to the costs of investigating and resolving problems, they include costs attributable to occupant illness and absence from work; meeting time for owners, designers, contractors, employers, and occupants; litigation costs; and lost revenues for landlords and tenants. Researchers for the British Columbia Buildings Corporation concluded that HVAC system commissioning is cost effective, as noted in Table 28.17 (present value = $0.20

TABLE 28.17

Item		Savings based on HVAC ($/ft^2)
Costs for Commissioning	Designer	0.01–0.10
	Contractor	0.10–0.20
	Owner	0.025–0.10
	Subtotal	0.145–0.40
Avoided Costs	Energy	0.13–0.26
	Maintenance	0.15
	Construction	0.07
	Satisfied tenants	0.25
	Subtotal	0.60–0.76
Net Result		0.20–0.515 over first 5 years

to \$0.50 per ft^2 cost savings over the first five years).

In the design for the San Francisco Public Library, commissioning was funded out of monies originally set aside for contingencies. This accounting reflects the shift in costs from planned prevention to post-problem remedial action (Bernheim, 1992).

The HVAC system commissioning process requires clear descriptions of the following:

- the sequence of actions for commissioning
- the performance required by the system(s) to be commissioned
- the intended use and operation of the system(s)
- the responsibility of all personnel involved

Suggested components of an HVAC system commissioning program are described in Table 28.18.

TABLE 28.18

Suggested components of an HVAC system commissioning program

Design phase (by architect/engineer)	Establishment of clear design criteria
	Documentation of HVAC design criteria and systems description
	Preparation of a commissioning plan
	Description of verification procedures
	Documentation requirements for commissioning process including all reports, submittals, drawings, schematics, checklists, operating data, maintenance data, and as-built documentation
Construction phase	Pre-commissioning preparation for start-up
	Personnel selection
	Pre-commissioning meeting of designer, owner, and contractor representatives
	Actual system start-up: initial operation of all equipment
	Final start-up: complete performance inspection
	Temperature control system
	Facility automation system
	Testing and balancing
	Equipment documentation
Final commissioning	Meeting of all relevant parties to discuss system and answer any questions about system sequences, set points, operation: reviewal of all final documentation for submittal to owner
	Assembly of all documents for submittal to owner
	Training operational personnel in the following: system philosophy; system familiarization; system sequence; system maintenance; system diagnosis.

28.5.6 Initial occupancy

The initial occupancy period is difficult for new occupants. Moving an office is stressful in itself. Unfortunately, it can also be the most difficult period for maintaining acceptable IAQ. Problems arise due to off-gassing from new materials, incomplete or inadequate air balancing, and malfunctions in newly completed air handling or control equipment. However, proper building testing and special ventilation will minimize IAQ complaints (Girman, 1990; Levin, 1989).

If one has to troubleshoot an IAQ complaint, he has to remember that industrial standards for air contaminants are generally not useful for evaluating non-industrial settings. Industrial standards are intended to protect workers (usually adult males) from high concentrations of individual substances; they are not designed to protect the general population from the combinations of contaminants that are found in modern non-industrial buildings.

During the initial occupancy period, architects should recommend provisional ventilation system operating protocols that maximize outdoor air supply rate and hours of operation and minimize operating temperatures. They should also recommend procedures for quickly, systematically, and economically evaluating IAQ, including objective inspections, rating systems, and questionnaires.

IAQ-related documentation during initial occupancy should include items such as the following:

- provisional ventilation system operating protocols;
- evaluation procedures and results for any IAQ evaluation;
- reports on any other tests or inspections taking place before or during initial occupancy, such as:
  - inspections or tests of building envelope;
  - inspections or tests of mechanical system;
- reports of any IAQ-related complaints, including the response and resolution.

Building owners often occupy new buildings before construction is complete. This "constructive occupancy" can result in considerable ambiguity when IAQ problems occur. Whenever premature occupancy occurs, designers should notify owners, contractors, and tenants that special precautions must be taken to avoid IAQ problems. These precautions may include one or more of the following:

- extend hours of ventilation system operation;
- increase outdoor air fraction;
- operate at lower temperatures;
- perform construction work in occupied space only during unoccupied periods.

Increasing the supply of outdoor air minimizes odours and contaminant levels and accelerates the off-gassing process for surface-dominated emissions. Because emissions are a function of vapour pressure which increases with temperature, maintain the lowest comfortable temperatures in occupied portions of the building during initial occupancy (Levin, 1992).

When buildings are vacant prior to initial occupancy (or re-occupancy after renovation), consider airing out procedures to accelerate emissions from surface materials. Where super-heating (raising the indoor temperature above normal occupancy temperatures) is used for bake-out purposes, it is essential that adequate ventilation be maintained during the bake-out period — at least at the point when maximum temperature is reached. This is necessary to reduce airborne concentrations of the chemicals which are targeted in the bake-out, because as air concentrations increase, emissions decrease. It is also important to reduce the quantity of emitted chemicals that adsorb on sinks to be re-emitted later. However, the procedure of bake-out is not completely validated and caution should be exercised to avoid damage to the building structure or contents during the bake-out as a result of excessive temperature or humidity changes (Girman et al., 1990).

If bake-out procedures are not employed, it is important to run the HVAC system continuously during the first hot weather period after construction. This will prevent uncontrolled (inadvertent) bake-out without adequate ventilation.

28.5.7 Testing building performance

This section provides a listing of items that must be checked before building occupancy and discusses ventilation protocols for the initial occupancy period. Perform the following testing prior to building occupancy:

Building Envelope

- Flood test flat roof systems to test for water leaks. (Caution: do not exceed design live loads.)
- Inspect flashings for possible signs of water leakage.
- Inspect doors and windows for correct installation including application of weather-stripping materials.
- Inspect drain systems, including sump pumps, for correct installation and signs of back-up of water.
- Inspect windows, including any solar equipment (e.g., solar shades), to verify proper installation and correct solar angles.
- Verify that outdoor air is not being drawn into the building through openings in the envelope (e.g., doors and windows) located in service dock areas or other potentially polluted areas.

Mechanical Systems

- Verify that mechanical systems are operating correctly and that air distribution systems are properly balanced.
- Verify that all outdoor supply louvres are open and working correctly.
- Verify that all interior spaces are receiving the correct amount of outside air according to the guidelines provided in ASHRAE 62-1989 or other project IAQ criteria. For example, if the exact number of occupants is not known, an estimate of 7 persons per 1000 ft^2 may be used to calculate ventilation requirements for general office space.
- During occupancy periods, verify that outdoor dampers remain open to design set points and that fans in air handling units operate continuously. Caution: In extremely cold climates, protect heat exchange equipment from freezing. Also, in humid climates, make sure that the building does not become excessively humid.
- Verify that all air supply registers/diffusers and return grilles are open and unobstructed, providing for adequate mixing in each supply zone. Supply diffusers should be adjusted so that occupants are not sitting in a direct stream of air and so that short circulating from supply to return does not occur.
- Verify that local exhaust grilles and hoods are correctly installed and operating.
- Verify appropriate negative or positive pressures in all interior zones.
- Check for back drafting at all combustion vents.

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Indoor Air Quality
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Chapter 29

Heating, Ventilation and Air Conditioning (HVAC) Systems and Indoor Air Quality[1]

This chapter provides information about specific HVAC system designs and components in relation to indoor air quality. It also serves as introductory material for building owners and managers who may be unfamiliar with the terminology and concepts associated with HVAC (heating, ventilating, and air conditioning) system design. Further detailed information can be found in ASHRAE manuals and guides and in some of the guidance developed by other trade and professional associations.

29.1 INTRODUCTION

All occupied buildings require a supply of outdoor air. Depending on outdoor conditions, the air may need to be heated or cooled before it is distributed into the occupied space. As outdoor air is drawn into the building, indoor air is exhausted or allowed to escape (passive relief), thus removing air contaminants.

The term "HVAC system" is used to refer to the equipment that can provide heating, cooling, filtered outdoor air, and humidity control to maintain comfort conditions in a building. Not all HVAC systems are designed to accomplish all of these functions. Some buildings rely only on natural ventilation. Others lack mechanical cooling equipment (AC), and many function with little or no humidity control. The features of the HVAC system in a given building will depend on several variables, including:

- age of the design;
- climate;
- building codes in effect at the time of the design;
- budget that was available for the project;

1 A part of the text of this chapter has been derived from EPA, DHHS, CDC; NIOSH. Building Air Quality: A guide for building owners and facility managers, Appendix B: HVAC systems and Indoor Air Quality, EPA/400/1-91/033, Washington, D.C.: U.S. Environmental Protection Agency. December 1991.

- planned use of the building;
- owners' and designers' individual preferences;
- subsequent modifications.

HVAC systems range in complexity from stand-alone units that serve individual rooms to large, centrally controlled systems serving multiple zones in a building. In large modern office buildings with heat gains from lighting, people, and equipment, interior spaces often require year-round cooling. Rooms at the perimeter of the same building (i.e., rooms with exterior walls, floors, or roof surfaces) may need to be heated and/or cooled as hourly or daily outdoor weather conditions change. In buildings over one story in height, perimeter areas at the lower levels also tend to experience the greatest uncontrolled air infiltration.

Some buildings use only natural ventilation or exhaust fans to remove odours and contaminants. In these buildings, thermal discomfort and unacceptable indoor air quality are particularly likely when occupants keep the windows closed because of extreme hot or cold temperatures. Problems related to underventilation are also likely when infiltration forces are weakest (i.e., during the "swing seasons" and summer months).

Modern public and commercial buildings generally use mechanical ventilation systems to introduce outdoor air during the occupied mode. Thermal comfort is commonly maintained by mechanically distributing conditioned (heated or cooled) air throughout the building. In some designs, air systems are supplemented by piping systems that carry steam or water to the building perimeter zones. As this chapter is concerned with HVAC systems in relation to indoor air quality, the remainder of this discussion will focus on systems that distribute conditioned air to maintain occupant comfort.

29.1.1 Roles of the HVAC system operator and facility manager

The system operator(s) and facility manager(s) (or IAQ manager) are among the most significant factors in determining whether IAQ problems will occur in a properly designed, constructed, and commissioned HVAC system. HVAC systems require preventive maintenance and prompt repairs if they are to operate correctly and provide comfortable conditions. The operator(s) must have an adequate understanding of the overall system design and its limitations. The HVAC system capacity and distribution characteristics should be evaluated before renovations to the building, changes in its occupancy, or changes in the use of an area. System operators must be able to respond appropriately to occupant complaints. For example, if an occupant complains that it is too cold or too hot and the observed (measured) conditions are outside of the ASHRAE comfort zone, the HVAC system needs to be evaluated.

Sometimes the problem can be relieved by fine tuning or repairing the HVAC system, but in some cases the system cannot perform as expected, and a long-term solution must be investigated.

29.2 TYPES OF HVAC SYSTEMS

29.2.1 Single zone

A single air handling unit can only serve more than one building area if the areas served have similar heating, cooling, and ventilation requirements, or if the control system compensates for differences in heating, cooling, and ventilation needs among the spaces served. Areas regulated by a common control (e.g., a single thermostat) are referred to as zones. Thermal comfort problems can result if the design does not adequately account for differences in heating and cooling loads between rooms that are in the same zone.

This can easily occur if:

- the cooling load in some area(s) with in a zone changes due to an increased occupant population, increased lighting, or the introduction of new heat-producing equipment (e.g., computers, copiers);
- areas within a zone have different solar exposures. This can produce radiant heat gains and losses that, in turn, create unevenly distributed heating or cooling needs (e.g., as the sun angle changes daily and seasonally).

29.2.2 Multiple zone

Multiple zone systems can provide each zone with air at a different temperature by heating or cooling the airstream in each zone. Alternative design strategies involve delivering air at a constant temperature while varying the volume of airflow, or modulating room temperature with a supplementary system (e.g., perimeter hot water piping).

29.2.3 Constant volume

Constant volume systems, as their name suggests, generally deliver a constant airflow to each space. Changes in space temperatures are made by heating or cooling the air or switching the air handling unit on and off, not by modulating the volume of air supplied. These systems often operate with a fixed minimum percentage of outdoor air or with an "air economizer" feature (described in the Outdoor Air Control discussion that follows).

29.2.4 Variable air volume

Variable air volume (VAV) systems maintain thermal comfort by varying the amount of heated or cooled air delivered to each space, rather than by changing the air temperature. However, many VAV systems also have provisions for resetting the temperature of the delivery air on a seasonal basis, depending on the severity of the weather. Overcooling or overheating can occur within a given zone if the system is not adjusted to respond to the load. Underventilation frequently occurs if the system is not arranged to introduce at least a minimum quantity (as opposed to percentage) of outdoor air as the VAV system throttles back from full airflow, or if the system supply air temperature is set too low for the loads present in the zone.

29.3 BASIC COMPONENTS OF AN HVAC SYSTEM

The basic components of an HVAC system that delivers conditioned air to maintain thermal comfort and indoor air quality are (see Fig. 29.1):

- outdoor air intake;
- mixed-air plenum and outdoor air control;
- air filter;
- heating and cooling coils;
- humidification and/or de-humidification equipment
- supply fan
- ducts
- terminal device
- return air system
- exhaust or relief fans and air outlet
- self-contained heating or cooling unit
- control
- boiler
- cooling tower
- water chiller

29.3.1 Outdoor air intake

Building codes require the introduction of outdoor air for ventilation in most buildings. Most non-residential air handlers are designed with an outdoor air intake on the return side of the ductwork. Outdoor air introduced through the air handler can be filtered and conditioned (heated or cooled) before distribution. Other designs may introduce outdoor air through air-to-air heat exchangers and operable windows.

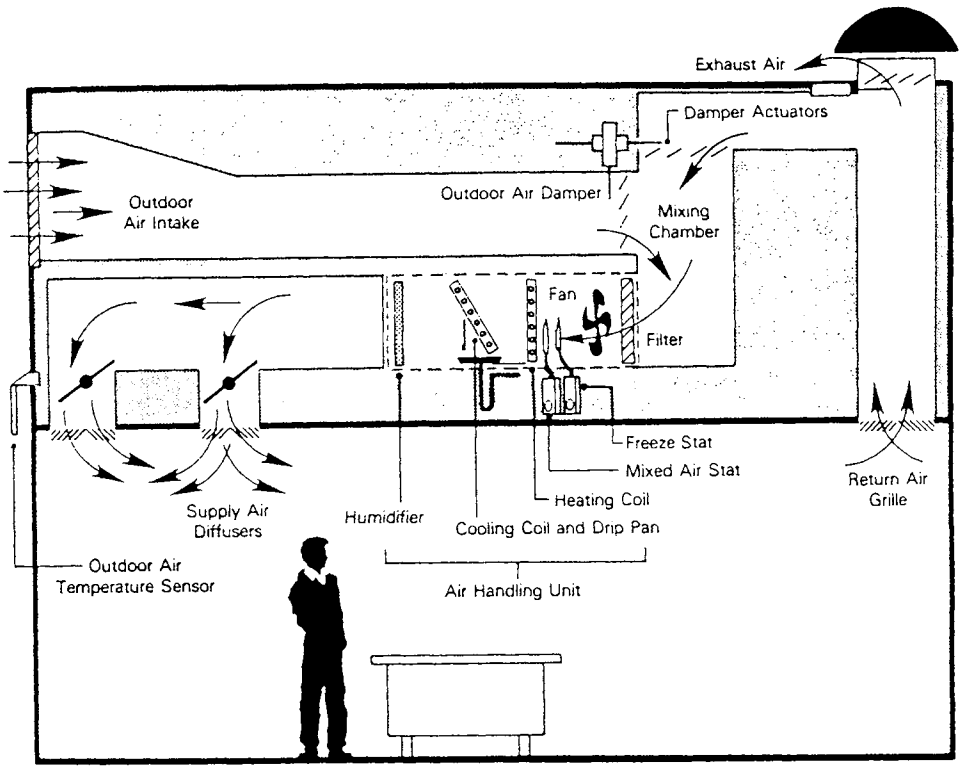

Fig. 29.1: Components of HVAC.

Indoor air quality problems can be produced when contaminants enter a building with the outdoor air. Rooftop or wall-mounted air intakes are sometimes located adjacent to or downwind of building exhaust outlets or other contaminant sources. Problems can also result if debris (e.g., bird droppings) accumulates at the intake, obstructing airflow and potentially introducing microbiological contaminants.

If more air is exhausted than is introduced through the outdoor air intake, then outdoor air will enter the building at any leakage sites in the shell. Indoor air quality problems can occur if the leakage site is a door to a loading dock, parking garage, or some other area associated with pollutants.

29.3.2 Mixed-air plenum and outdoor air controls

Outdoor air is mixed with return air (air that has already circulated through the HVAC system) in the mixed-air plenum of an air handling unit. Indoor air quality problems frequently result if the outdoor air damper is not

operating properly, e.g., if the system is not designed or adjusted to allow the introduction of sufficient outdoor air for the current use of the building. The amount of outdoor air introduced in the occupied mode should be sufficient to meet needs for ventilation and exhaust make-up. It may be fixed at a constant volume or may vary with the outdoor temperature.

When dampers that regulate the flow of outdoor air are arranged to modulate, they are usually designed to bring in a minimum amount of outdoor air (in the occupied mode) under extreme outdoor temperature conditions and to open as outdoor temperatures approach the desired indoor temperature. Systems that use outdoor air for cooling are called "air economizer cooling" systems. Air economizer systems have a mixed air temperature controller and thermostat that are used to blend return air (typically at 74°F) with outdoor air to reach a mixed air temperature of 55–65°F. Mixed air temperature settings above 65°F may lead to the introduction of insufficient quantities of outdoor air for office space use.

The mixed air is then further heated or cooled for delivery to the occupied spaces.

Air economizer systems have a sensible or enthalpy control that signals the outdoor air damper to go to the minimum position when it is too warm or humid outdoors.

Note that economizer cycles which do not provide dehumidification may produce discomfort even when the indoor temperature is the same as the thermostat setting.

If outdoor air make-up and exhaust are balanced, and the zones served by each air handler are separated and well defined, it is possible to estimate the minimum flow of outdoor air to each space and compare it to ventilation standards such as ASHRAE 62-1989. Techniques used for this evaluation include the direct measurement of the outdoor air at the intake and the calculation of the percentage of outdoor air by a temperature or CO_2 balance. Carbon dioxide measured in an occupied space is also an indicator of ventilation adequacy. Some investigators use tracer gases to assess ventilation quantities and airflow patterns. There are specific methods for each of these assessments. See Chapter 26 for more information.

Many HVAC designs protect the coils by closing the outdoor air damper if the airstream temperature falls below the set-point of a freezestat. Inadequate ventilation can occur if a freezestat trips and is not reset, or if a freezestat is set to trip at an excessively high temperature. Stratification of a cold outdoor air and warmer return air in the mixing plenums is a common situation, causing nuisance tripping of the freezestat. Unfortunately, the remedy often employed to prevent this problem is to close the outdoor air damper. Obviously, solving the problem in this way can quickly lead to inadequate outdoor air in occupied parts of the building.

29.3.3 Air filters

Filters are primarily used to remove particles from the air. The type and design of a filter determine its efficiency at removing particles of a given size and the amount of energy needed to pull or push air through the filter. Filters are rated by different standards and test methods such as dust spot and arrestance which measure different aspects of performance.

Low efficiency filters (ASHRAE Dust Spot rating of 10–20% or less) are often used to keep lint and dust from clogging the heating and cooling coils of a system. In order to maintain clean air in occupied spaces, filters must also remove bacteria, pollens. insects, soot, dust and dirt with an efficiency suited to the use of the building. Medium efficiency filters (ASHRAE Dust Spot rating of 30–60%) can provide much better filtration than low efficiency filters. To maintain the proper airflow and minimize the amount of additional energy required to move air through these higher efficiency filters, pleated-type extended surface filters are recommended. In buildings that are designed to be exceptionally clean, the designers may specify the equipment to utilize both a medium efficiency pre-filter and a high efficiency extended surface filter (ASHRAE Dust Spot rating of 85–95%). Some manufactures recommend high efficiency extended surface filters (ASHRAE Dust Spot rating of 85%) without pre-filters as the most cost effective approach to minimizing energy consumption and maximizing air quality in modern HVAC VAV systems that serve office environments.

Air filters, whatever their design or efficiency rating, require regular maintenance (cleaning for some and replacement for most). As a filter loads up with particles, it becomes more efficient at particle removal but increases the pressure drop through the system, therefore reducing airflow. Filter manufactures can provide information on the pressure drop through their products under different conditions. Low efficiency filters, if loads to excess, will become deformed and even "blow out" of their filter rack. When filters blow out, bypassing of unfiltered air can lead to clogged coils and dirty ducts. Filtration efficiency can be seriously reduced if the filter cells are not properly sealed to prevent air from bypassing.

Filters should be selected for their ability to protect both the HVAC system components and general indoor air quality. In many buildings, the best choice is a medium efficiency, pleated filter because these filters have a higher removal efficiency than low efficiency filters, yet they will last without clogging for longer than high efficiency filters.

Choice of an appropriate filter and proper maintenance are important to keeping the ductwork clean. If dirt accumulates in ductwork and if the relative humidity reaches the dew-point (so that condensation occurs), then the nutri-

ents and moisture may support the growth of microbiologicals. Attention to air filters is particularly important in HVAC systems with acoustical duct liner, which is frequently used in air handler fan housings and supply ducts to reduce sound transmission and provide thermal insulation. Areas of duct lining that have become contaminated with microbiological growth must be replaced. (See later discussion of ducts and duct cleaning). Sound reduction can also be accomplished with the use of special duct-mounted devices such as attenuators or with active electronic noise control.

Air handlers that are located in difficult-to-access places (e.g., in places which require ladders for access, have inconvenient access doors to unbolt, or are located on roofs with no roof hatch access) will be more likely to suffer from poor air filter maintenance and overall poor maintenance. Quick release and hinged access doors for maintenance are more desirable than bolted access panels.

Filters are available to remove gases and volatile organic contaminants from ventilation air; however, these systems are not generally used in normal occupancy buildings. In specially designed HVAC systems, permanganate oxidizers and activated charcoal may be used for gaseous removal filters. Some manufacturers offer "partial bypass" carbon filters and carbon impregnated filters to reduce volatile organics in the ventilation air of office environments. Gaseous filters must be regularly maintained (replaced or regenerated) in order for the system to continue to operate effectively.

29.3.4 Heating and cooling coils

Heating and cooling coils are placed in the airstream to regulate the temperature of the air delivered to the space. Malfunction of the coil controls can result in thermal discomfort. Condensation on underinsulated pipes and leakage in piped systems will often create moist conditions conducive to the growth of moulds, fungus, and bacteria.

During the cooling mode (air conditioning), the cooling coil provides dehumidification as water condenses from the airstream. Dehumidification can only take place if the chilled fluid is maintained at a cold enough (generally below 45°F for water). Condensate collects in the drain pan under the cooling coil and exits via a deep seal trap. Standing water will accumulate if the drain pan system has not been designed to drain completely under all operating conditions (sloped toward the drain and properly trapped). Under these conditions, moulds and bacteria will proliferate unless the pan is cleaned frequently.

It is important to verify that condensate lines have been properly trapped and are charged with liquid. An improperly trapped line can be a source of contamination, depending on where the line terminates. A properly installed

trap could also be a source, if the water in the trap evaporates and allows air to flow through the trap into the conditioned air.

During the heating mode, problems can occur if the hot water temperature in the heating coil has been set too low in an attempt to reduce energy consumption. If enough outdoor air to provide sufficient ventilation is brought in, that air may not be heated sufficiently to maintain thermal comfort or, in order to adequately condition the outdoor air, the amount of outdoor air may be reduced so that there is insufficient outdoor air to meet ventilation needs.

29.3.5 Humidification and dehumidification equipment

In some buildings and in zones within buildings, there are special needs that warrant the strict control of humidity (e.g., operating rooms, computer rooms). This control is most often accomplished by adding humidification or dehumidification equipment and controls. In office facilities, it is generally preferable to keep relative humidities above 20 or 30% during the heating season and below 60% during the cooling season. ASHRAE Standard 55-1981 provides guidance on acceptable temperature and humidity conditions (see also Chapter 31).

The use of a properly designed and operated air conditioning system will generally keep relative humidities below 60% RH during the cooling season, in office facilities with normal densities and loads.

Office buildings in cool climates that have high interior heat gains, thermally efficient envelopes (e.g., insulation), and economizer cooling may require humidification to maintain relative humidity within the comfort zone. When humidification is needed, it must be added in a manner that prevents the growth of microbiologicals within the ductwork and air handlers.

Steam humidifiers should utilize clean steam, rather than treated boiler water, so that occupants will not be exposed to chemicals. Systems using media other than clean steam must be rigorously maintained in accordance with the manufacturer's recommended procedures to reduce the likelihood of microbiological growth.

Mould growth problems are more likely if the humidistat set-point located in the occupied space is above 45%. The high limit humidistat, typically located in the ductwork downstream of the point at which water vapour is added, is generally set at 70% to avoid condensation (with a potential for subsequent mould growth) in the ductwork. Adding water vapour to a building that was not designed for humidification can have a negative impact on the building structure and the occupants' health, if condensation occurs on cold surfaces or in wall or roof cavities.

29.4 TESTING, BALANCING AND MAINTAINING

Modern HVAC systems typically use sophisticated, automatic controls to supply the proper amounts of air for heating, cooling, and ventilation in commercial buildings. Problems during installation, operation, maintenance, and servicing of the HVAC system could prevent it from operating as designed. Each system should be tested to ensure its initial and continued performance. In addition to providing acceptable thermal conditions and ventilation air, a properly adjusted and balanced system can also reduce operating costs and increase equipment life.

Testing and balancing involves the testing, adjusting, and balancing of HVAC system components so that the entire system provides airflows that are in accordance with the design specifications. Typical components and system parameters tested include:

- all supply, return, exhaust, and outdoor airflow rates;
- control settings and operation;
- air temperatures;
- fan speeds and power consumption;
- filter or collector resistance.

The typical test and balance agency or contractor coordinates with the control contractor to accomplish three goals: verify and ensure the most effective system operation within the design specifications, identify and correct any problems, and ensure the safety of the system.

A test and balance report should provide a complete record of the design, preliminary measurements, and final test data. The report should include any discrepancies between the test data and the design specifications, along with reason for those discrepancies. To facilitate future performance checks and adjustments, appropriate records should be kept on all damper positions, equipment capacities, control types and locations, control settings and operating logic, airflow rates, static pressures, fan speeds, and horsepower.

Testing and balancing of existing building systems should be performed whenever there is reason to believe the system is not functioning as designed or when current records do not accurately reflect the actual operation of the system. The Associated Air Balance Council recommends the following guidelines in determining whether testing and balancing is required:

- when space has been renovated or changed to provide for new occupancy;
- when HVAC equipment has been replaced or modified;
- when control settings have been readjusted by maintenance or other personnel;
- after the air conveyance system has been cleaned;
- when accurate records are required to conduct an IAQ investigation;

- when the building owner is unable to obtain design documents or appropriate air exchange rates for compliance with IAQ standards or guidelines.

Because of the diversity of system types and the interrelationship of system components, effective balancing requires a skilled technician with the proper experience and instruments. Due to the nature of the work, which involves the detection and remediation of problems, it is recommended that an independent test and balance contractor be used and that this contractor report directly to the building owner, facility manager, or IAQ manager.

29.4.1 Duct leakage

Leakage of air from ducts can cause or exacerbate air quality problems, in addition to wasting energy. In general, sealed duct systems specified with a leakage rate of less than 3% will have a superior life cycle cost analysis and reduce the likelihood of problems associated with leaky ductwork. Examples of excessive duct leakage leading to problems include:

- leakage of light troffer-type diffusers at the diffuser/light fixture interface when they are installed in a return plenum. Such leakage has been known to cause gross short-circuiting between supply and return, wasting much of the conditioned air. If the "room" thermostat is located in the return plenum, the room can be very uncomfortable while the temperature in the plenum is operating at the control set-point;
- leakage of supply ductwork due to loose-fitting joints and connections or "blow outs" of improperly fabricated seams;
- leakage of return ducts located in crawl spaces or below slabs, allowing soil gases and moulds to enter the ductwork.

29.4.2 Supply fans

After passing through the coil section where heat is either added or extracted, air moves through the supply fan chamber and the distribution system. Air distribution systems commonly use ducts that are constructed to be relatively airtight. Elements of the building construction can also serve as part of the air distribution system (e.g., pressurized supply plenums or return air plenums located in the cavity space above the ceiling tiles and below the deck of the floor above). Proper coordination of fan selection and duct layout during the building design and construction phase and ongoing maintenance of mechanical components, filters, and controls are all necessary for effective air delivery.

Fan performance is expressed as the ability to move a given quantity of air (cubic feet per minute or cfm) at a given resistance or static pressure (meas-

ured in inches of water column). Airflow in ductwork is determined by the size of the duct opening, the resistance of the duct configuration, and the velocity of the air through the duct. The static pressure in a system is calculated using factors for duct length, speed of air movement an changes in the direction of air movement.

It is common to find some differences between the original design and the final installation, as ductwork must share limited space with structural members and other "hidden" elements of the building system (e.g., electrical conduit, plumbing pipes). Air distribution problems can occur, particularly at the end of duct runs, if departures from the original design increase the friction in the system to a point that approaches the limit of fan performance. Inappropriate use of long runs of flexible ducts with sharp bends also causes excessive friction. Poor system balancing (adjustment) is another common cause of air distribution problems.

Dampers are used as controls to restrict airflow. Damper positions may be relatively fixed (e.g., set manually during system testing and balancing) or may change in response to signals from the control system. Fire and smoke dampers can be triggered to respond to indicators such as high temperatures or signals from smoke detectors. If a damper is designed to modulate, it should be checked during inspection to see that it is at the proper setting. ASHRAE and the Associated Air Balance Council both provide guidance on proper intervals for testing and balancing.

29.4.3 Ducts

The same HVAC system that distributes conditioned air throughout a building can distribute dust and other pollutants, including biological contaminants. Dirt or dust accumulation on any components of an air handling system — its cooling coils, plenums, ducts, and equipment housing — may lead to contamination of the air supply.

There is widespread agreement that building owners and managers should take great precautions to prevent dirt, high humidity, or moisture from entering the ductwork; there is less agreement at present about when measures to clean up are appropriate or how effective cleaning techniques are at making long-term improvements to the air supply or at reducing occupant complaints.

The presence of dust in ductwork does not necessarily indicate a current microbiological problem. A small amount of dust on duct surfaces in normal and to be expected. Special attention should be given to trying to find out if ducts are contaminated only where specific problems are present, such as: water damage or biological growth observed in ducts, debris in ducts that

restricts airflow, or dust discharging from supply diffusers.

Problems with dust and other contamination in the ductwork are a function of filtration efficiency, regular HVAC system maintenance, the rate of airflow, and good housekeeping practices in the occupied space. Problems with biological pollutants can be prevented by minimizing dust and dirt build-up, promptly repairing leaks and water damage, preventing moisture accumulation in the components that are supposed to be dry, and cleaning the components such as the drip pans that collect and drain water.

In cases where sheet metal ductwork has become damaged or water-soaked, building owners will need to undertake clean-up or repair procedures. For example, in cases where the thermal liner or fibreboard has become water-soaked, building managers will need to replace the affected areas. These procedures should be scheduled and performed in a way that does not expose building occupants to increased levels of pollutants and should be carried out by experienced workers. Correcting problems that allowed the ductwork to become contaminated in the first place is important. Otherwise, the corrective action will only be temporary.

The porous surface of fibrous glass duct liner presents more surface area (which can trap dirt and subsequently collect water) than sheet metal ductwork. It is therefore particularly important to pay attention to the proper design, installation, filtration, humidity, and maintenance of ducts that contain porous materials. In addition, techniques developed for cleaning unlined metal ducts often are not suitable for use with fibrous glass thermal liner or fibreboard. Such ducts may require a special type of cleaning to maintain the integrity of the duct (i.e., no heavy brushing tools that might fray the inner lining) while removing dirt and debris.

More research on both the efficacy and the potential for unintended exposures to building occupants from various cleaning techniques is needed before firm guidance can be provided regarding duct cleaning.

Pay attention to worker safety when working with air handling systems including during duct cleaning. Any worker who may potentially breathe duct contaminants or biocides should wear suitable protective breathing apparatus. Workers who are doing the duct cleaning should be encouraged to also look for other types of problems, such as holes or gaps in the ducts that could allow contaminants to enter the ventilation airstream.

29.4.4 Recommendations on duct cleaning

– *Any duct cleaning should be scheduled during periods when the building is unoccupied to prevent exposure to chemicals and loosened particles.*

The air handling unit should not be used during the cleaning or as an air

movement device for the cleaning process. The National Air Duct Cleaning Association recommends that the system should be run to allow at least eight air changes in the occupied space when duct cleaning has been completed.

- *Negative air pressure that will draw pollutants to a vacuum collection system should be maintained at all times in the duct cleaning area to prevent migration of dust, dirt, and contaminants into occupied areas.*

Where possible, use vacuum equipment or fans during cleaning and sanitizing to make sure that cleaning vapours are exhausted to the outside and do not enter the occupied space.

- *If it is determined that the ductwork should be cleaned, careful attention must be given to protecting the ductwork.*

When gaining access to sheet metal ducts for cleaning purposes, it is essential to seal the access hole properly in order to maintain the integrity of the HVAC system. Access doors are recommended if the system is to be cleaned periodically, and all access holes should be identified on the building's mechanical plans.

Particular attention is warranted when cutting fibrous glass ducts, and manufacturers' recommended procedures for sealing should be followed stringently. Use existing duct system openings where possible because it is difficult to repair the damage caused by cutting new access entries into the ductwork. Large, high volume vacuum equipment should only be used with extreme care because high negative pressure together with limited airflow can collapse ducts.

- *Duct cleaning performed with high velocity airflow (i.e., greater than 6,000 cfm) should include gentle, well-controlled brushing of duct surfaces or other methods to dislodge dust and other particles.*

Duct cleaning that relies only on a high velocity airflow through the ducts is not likely to achieve satisfactory results because the flow rate at the duct surface remains too low to remove many particles.

- *Only HEPA filtered (high-efficiency particle arrestor) vacuuming equipment should be used if the vacuum collection unit is inside the occupied space.*

Conventional vacuuming equipment may discharge extremely fine particulate matter back into the atmosphere, rather than collecting it. Duct cleaning equipment that draws the dust and dirt into a collection unit outside the building is also available. People should not be allowed to remain in the immediate vicinity of these collection units.

- *If biocides are to be used, then select only products registered by EPA for such use, use the products according to the manufacturer's directions, and pay careful attention to the method of application.*

At present, EPA accepts claims and therefore registers antimicrobials for use only as sanitizers, not disinfectants or sterilizers in HVAC systems. (See

Appendix F for definitions of antimicrobials.) There is some question about whether there are any application techniques that will deposit a sufficient amount of the biocide to kill bacteria, germs, or other biologicals that may be present. Materials such as deodorizers that temporarily eliminate odours caused by microorganisms provide only a fresh smell, and are not intended to provide real control of microbiological contaminants.

– *Use of sealants to cover interior ductwork surfaces is not recommended.*

No application techniques have been demonstrated to provide a complete or long-term barrier to microbiological growth, nor have such materials been evaluated for their potential health effects on occupants. In addition, using sealants alters the surface burning characteristics of the duct material and may void the fire safety rating of the ductwork.

– *Careful cleaning and sanitizing of any parts of coils and drip pans can reduce microbiological pollutants.*

Prior to using sanitizers, deodorizers, or any cleansing agents, carefully read the directions on the product label. Once cleaned, these components should be thoroughly rinsed and dried to prevent exposure of building occupants to the cleaning chemicals.

– *Water-damaged or contaminated porous materials in the ductwork or other air handling system components should be removed and replaced.*

Even when such materials are thoroughly dried, there is no way to guarantee that all microbial growth has been eliminated.

– *After the duct system has been cleaned and restored to use, a preventive maintenance program will prevent the recurrence of problems.*

Such a program should include particular attention to the use and maintenance of adequate filters, control of moisture in the HVAC system, and periodic inspection and cleaning of HVAC system components.

29.4.5 Terminal devices

Thermal comfort and effective contaminant removal demand that air delivered into a conditioned space be properly distributed within that space. Terminal devices are the supply diffusers, return and exhaust grilles, and associated dampers and controls that are designed to distribute air within a space and collect it from that space. The number, design, and location (ceiling, wall, floor) of terminal devices are very important. They can cause a HVAC system with adequate capacity to produce unsatisfactory results, such as drafts, odour transport, stagnant areas, or short-circuiting.

Occupants who are uncomfortable because of distribution deficiencies (draughts, odour transport, stagnant air, or uneven temperatures) often try to compensate by adjusting or blocking the flow of air from supply outlets.

Adjusting system flows without any knowledge of the proper design frequently disrupts the proper supply of air to adjacent areas. Distribution problems can also be produced if the arrangement of movable partitions, shelving, or other furnishings interferes with airflow. Such problems often occur if walls are moved or added without evaluating the expected impact on airflows.

For ventilation efficiency testing see Chapter 26.

29.4.6 Return air systems

In many modern buildings the above-ceiling space is utilized for the unducted passage of return air. This type of system approach often reduces initial HVAC system costs, but requires that the designer, maintenance personnel, and contractors obey strict guidelines related to health and safety codes (e.g., building codes) that must be followed for materials and devices that are located in the plenum. In addition, if a ceiling plenum is used for the collection of return air, openings into the ceiling plenum created by the removal of ceiling tiles will disrupt airflow patterns. It is particularly important to maintain the integrity of the ceiling and adjacent walls in areas that are designed to be exhausted, such as supply closets, bathrooms, and chemical storage areas.

After return air enters either a ducted return air grille or a ceiling plenum, it is returned to the air handlers. Some systems utilize return fans in addition to supply fans in order to properly control the distribution of air. When a supply and return fan are utilized, especially in a VAV system, their operation must be coordinated in order to prevent under — or over — pressurization of the occupied space or overpressurization of the mixing plenum in the air handler.

29.4.7 Exhausts, exhaust fans, and pressure relief

Most buildings are required by law (e.g., building or plumbing codes) to provide for exhaust of areas where contaminant sources are strong, such as toilet facilities, janitorial closets, cooking facilities, and parking garages. Other areas where exhaust is frequently recommended but may not be legally required include: reprographics areas, graphic arts facilities, beauty salons, smoking lounges, shops, and any area where contaminants are known to originate.

For successful confinement and exhaust of identifiable sources, the exhausted area must be maintained at a lower overall pressure than surrounding areas. Any area that is designed to be exhausted must also be isolated (disconnected) from the return air system so that contaminants are not transported to another area of the building.

In order to exhaust air from the building, make-up air from outdoors must be brought into the HVAC system to keep the building from being run under negative pressure. This make-up air is typically drawn in at the mixed air plenum as described earlier and distributed within the building. For exhaust systems to function properly, the make-up air must have a clear path to the area that is being exhausted.

It is useful to compare the total cfm of powered exhaust to the minimum quantity of mechanical-introduced outdoor air.

To prevent operating the building under negative pressure (and limit the amount of unconditioned air brought into the building by infiltration), the amount of make-up air drawn in at the air handler should always be slightly greater than the total amount of relief air, exhaust air, and air exfiltrating through the building shell. Excess make-up air is generally relieved at an exhaust or relief outlet in the HVAC system, especially in air economizer systems. In addition to reducing the effects of unwanted infiltration, designing and operating a building at slightly positive or neutral pressures will reduce the rate of entry of soil gases when the systems are operating. For a building to actually operate at a slight positive pressure (e.g., specified at less than one-half air change per hour at 0.25 pascals). Otherwise unwanted exfiltration will prevent the building from ever achieving a neutral or slightly positive pressure.

29.4.8 Self-contained units

In some designs, small decentralized units are used to provide cooling or heating to interior or perimeter zones. With the exception of induction units, units of this type seldom supply outdoor air. They are typically considered a low priority maintenance item. If self-contained units are overlooked during maintenance, it is not unusual for them to become a significant source of contaminants, especially for the occupants located nearby.

29.4.9 Controls

HVAC systems can be controlled manually or automatically. Most systems are controlled by some combination of manual and automatic controls. The control system can be used to switch fans on and off, regulate the temperature of air within the conditioned space, or modulate airflow and pressures by controlling fan speed and damper settings. Most large buildings use automatic controls, and many have very complex and sophisticated systems. Regular maintenance and calibration are required to keep controls in good operating order. All programmable timers and switches should have "battery backup" to reset the controls in the event of a power failure.

Local controls such as room thermostats must be properly located in order to maintain thermal comfort. Problems can result from:

- thermostats located outside of the occupied space (e.g., in return plenum)
- poorly designed temperature control zones (e.g., single zones that combine areas with very different heating or cooling loads)
- thermostat locations subject to drafts or to radiant heat gain or loss (e.g., exposed to direct sunlight)
- thermostat locations affected by heat from nearby equipment

To test whether or not a thermostat is functioning properly, try setting it to an extreme temperature. This experiment will show whether or not the system is responding to the signal in the thermostat, and also provides information about how the HVAC system may perform under extreme conditions.

29.4.10 Boilers

Like any other part of the HVAC system, a boiler must be adequately maintained to operate properly. However, it is particularly important that combustion equipment operate properly to avoid hazardous conditions such as explosions or carbon monoxide leaks, as well as to provide good energy efficiency. Codes in most parts of the country require boiler operators to be properly trained and licensed.

ASHRAE has made recommendations of how much combustion air is needed for fuel burning appliances.

Elements of boiler operation that are particularly important to indoor air quality and thermal comfort include:

- Operation of the boiler and distribution loops at a high enough temperature to supply adequate heat in cold weather.
- Maintenance of gaskets and breaching to prevent carbon monoxide from escaping into the building.
- Maintenance of fuel lines to prevent any leaks that could emit odours into the building.
- Provision of adequate outdoor air for combustion.
- Design of the boiler combustion exhaust to prevent re- entrainment, especially from short boiler stacks or into multistorey buildings that where added after the boiler plant was installed.

Modern office buildings tend to have much smaller capacity boilers than older buildings because of advances in energy efficiency. In some buildings, the primary heat source is waste heat recovered from the chiller which operates year-round to cool the core of building.

29.4.11 Cooling towers

Maintenance of a cooling tower ensures proper operation and keeps the cooling tower from becoming a niche for breeding pathogenic bacteria, such as Legionella organisms. Cooling tower water quality must be properly monitored and chemical treatments used as necessary to minimize conditions that could support the growth of significant amounts of pathogens. Proper maintenance may also entail physical cleaning (by individuals using proper protection) to prevent sediment accumulation and installing drift eliminators.

29.4.12 Water chillers

Water chillers are frequently found in large building air conditioning systems because of the superior performance they offer. A water chiller must be maintained in proper working condition to perform its function of removing the heat from the building. Chilled water supply temperatures should operate in the range of 45°F or colder in order to provide proper moisture removal during humid weather. Piping should be insulated to prevent condensation.

Other than thermal comfort, IAQ concerns associated with water chillers involve potential release of the working fluids from the chiller system. The rupture disk (safety release) of the system should be piped to the outdoors, and refrigerant leaks should be located and repaired. Waste oils and spent refrigerant should be disposed of properly.

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Indoor Air Quality
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Chapter 30

Building Operations and Maintenance[1]

30.1 MANAGING BUILDINGS FOR GOOD INDOOR AIR QUALITY

The relationships among building owners, management, staff, and occupants are an important factor in decisions that affect indoor air quality. The objectives of the major players in these relationships may be very different. Occupants want the building to be pleasant, safe and attractive; if they are paying tenants, they also want to get the maximum use out of the space they rent for the least cost. Building owners and management want to maintain a reputation for providing quality property at reasonable cost, but also need to derive a profit. Facility staff are often caught in the middle, trying to control operating and maintenance costs while still keeping occupants satisfied.

Regardless of the points on which they may disagree, building occupants, staff, and management share the goal of providing a healthy indoor environment. Recognition of this common goal may help avoid conflict when discussing IAQ-related policies.

Any IAQ management system will be successful only if it is organized to fit your specific building. It would not be appropriate for this document to prescribe any single approach. However, the skills associated with IAQ management activities will be identified to help building management decide who will be best able to carry them out. Education and training programs for staff and building occupants should be provided to ensure that new procedures are understood and adopted.

Managing a building for good indoor air quality involves reviewing and amending current practice (and establishing new procedures, if necessary) to:

(a) Operate and maintain HVAC equipment
- keep all equipment and controls in proper working order
- keep interior of equipment and ductwork clean and dry

1 A part of the text of this chapter has been derived from EPA, DHHS, CDC, NIOSH. Building Air Quality: A guide for building owners and facility managers, Appendix B: HVAC systems and Indoor Air Quality, EPA/400/1-91/033, Washington, D.C.: U.S. Environmental Protection Agency. December 1991.

(b) Oversee activities of staff, tenants, contractors, and other building occupants that impact indoor air quality
- smoking;
- housekeeping;
- building maintenance;
- shipping and receiving;
- pest control;
- food preparation and other special uses.

(c) Maintain communications with occupants so that management will be informed of complaints about the indoor environment in a timely way
- identify building management and staff with IAQ responsibilities
- use health and safety committees

(d) Educate staff, occupants, and contractors about their responsibilities in relation to indoor air quality
- staff training;
- lease arrangements;
- contracts.

(e) Identify aspects of planned projects that could affect indoor air quality and manage projects so that good air quality is maintained
- redecorating, renovation, or remodelling
- relocation of personnel or functions within the building
- new construction

30.1.1 Developing an indoor air quality management plan

The elements of an IAQ management plan are summarized in the chart in Figure 30.1. Development of the management plan involves reviewing and revising staff responsibilities so that IAQ considerations become incorporated into routine procedures.

Organizations may assign responsibility for operations, record keeping, purchasing, communications, planning, and policy-making in many different ways. However, the key elements of good IAQ management remain the same:

(a) Reach an understanding of the fundamental influences that affect indoor air quality in your building by:
- becoming familiar with literature on IAQ;
- keeping abreast of new information.

(b) Select an IAQ manager with:
- clearly defined responsibilities;
- adequate authority and resources.

(c) Use the IAQ profile and other available information to:
- evaluate the design, operation, and usage of the building;

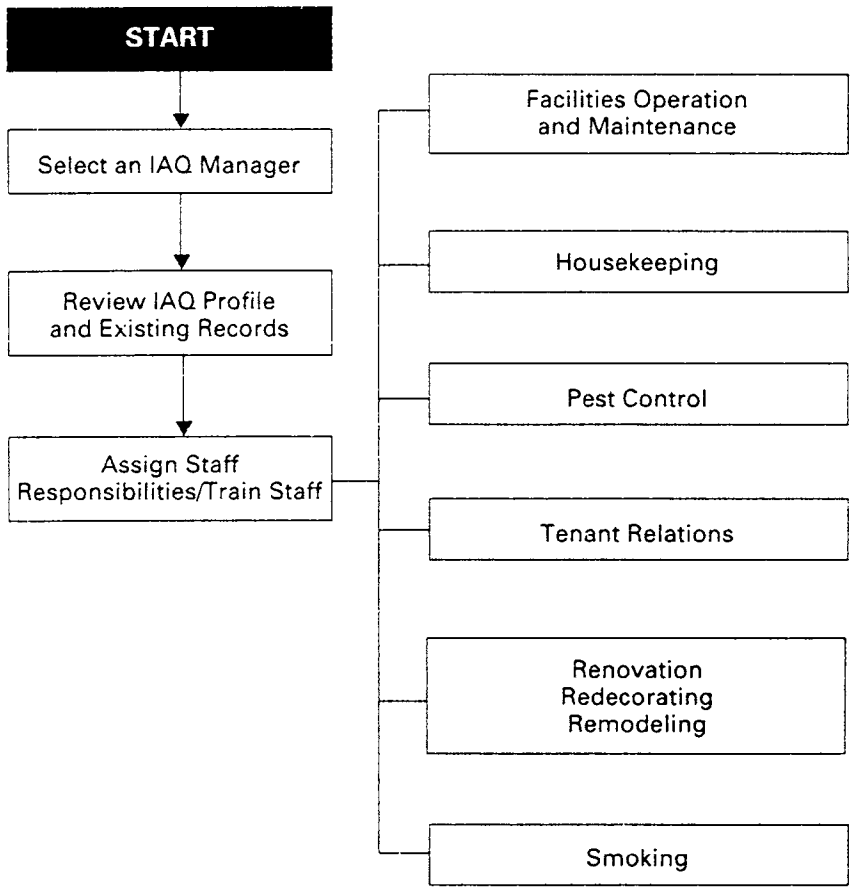


Fig. 30.1: Developing an IAQ management plan.

– identify potential IAQ problem locations;
– identify staff and contractors whose activities affect indoor air quality.

(d) Review and revise staff responsibilities to ensure that responsibilities that may affect indoor air quality are clearly assigned. In addition, establish lines of communication for sharing information pertaining to:

– equipment in need of repair or replacement;
– plans to remodel, renovate, or redecorate;
– new uses of building space or increases in occupant population;
– installation of new equipment.

(e) Review standard procedures and make necessary revisions to promote good indoor air quality, such as:

– terms of contracts (e.g., pest control, leases);
– scheduling of activities that produce dust, emissions, odours;

- scheduling of equipment operation, inspection, and maintenance;
- specifications for supplies (e.g., cleaning products, construction materials, furnishings);
- policy regarding tobacco smoking within the building.

(f) Review the existing record keeping system and make necessary revisions to:

- establish a system for logging-IAQ related complaints;
- obtain Material Safety Data Sheets for hazardous materials used and stored in the building.

(g) Educate building staff, occupants, and contractors about their influence on indoor air quality by:

- establishing a health and safety committee;
- instituting training programs as needed.

IAQ problems may occur even in buildings whose owners and managers conscientiously apply the best available information to avoid such problems. Those who can demonstrate their ongoing efforts to provide a safe indoor environment are in a strong legal and ethical position if problems do arise.

(h) Select an IAQ Manager

IAQ management will be facilitated if one individual is given overall responsibility for IAQ. Whether or not this person is given the title of "IAQ Manager," he or she should have a good understanding of the building's structure and function and should be able to communicate with tenants, facility personnel, and building owners or their representatives about IAQ issues.

The IAQ manager's ongoing responsibilities might include:

- developing the IAQ profile;
- overseeing the adoption of new procedures;
- establishing a system for communicating with occupants about IAQ issues;
- coordinating staff efforts that effect indoor air quality, and making sure that staff have the information (e.g., operating manuals, training) and authority to carry out their responsibilities;
- reviewing all major projects in the building for their IAQ implications;
- reviewing contracts and negotiating with contractors (e.g., cleaning services, pest control contractors) whose routine activities in the building could create IAQ problems;
- periodically inspecting the building for indicators of IAQ problems;
- managing IAQ-related records;
- responding to complaints or observations regarding potential IAQ problems;
- conducting an initial walk-through investigation of any IAQ complaints.

30.1.2 Product of the review of the indoor air quality profile and other existing records

The goal is to establish:

(a) a priority list of locations and activities within the building that will require special attention in order to prevent indoor air quality problems

(b) a list of staff and contractors whose responsibilities need to be included in the IAQ management plan

Review IAQ profile and other existing records

If the IAQ manager was not actively involved in developing the IAQ profile, one of the first tasks will be to review the profile carefully. The manager can start by also identifying building locations with a potential for IAQ problems, staff and contractors whose activities impact indoor air quality, and other building occupants whose activities impact indoor air quality.

In addition to information from the IAQ profile, it may be helpful to review lease forms and other contractual agreements for an understanding of the respective legal responsibilities of the building management, tenants, and contractors. Incorporation of IAQ concerns into legal documents helps to ensure the use of proper materials and procedures by contractors and can help to limit the load placed on ventilation equipment by occupant activities.

Assign responsibilities / train staff

The assignment of responsibilities varies widely between organizations, depending upon the routine activities to be carried out and the capabilities of the available personnel. It would not be appropriate for this document to suggest how IAQ-related responsibilities should be allocated in your organization. For example, issues of access in buildings with tenant-occupied space highlight the need for cooperation between building managers and the tenants' office managers. The building staff may be limited in its access to tenant spaces and tenants may not have access to building operations areas such as mechanical rooms, yet both tenants and building management have responsibilities for maintaining good indoor air quality.

Facility personnel are not generally trained to think about IAQ issues as they go about their work. Even though building staff may be observing events and conditions that would indicate potential problems to an experienced IAQ investigator, the staff member's attention may be directed elsewhere. As new practices are introduced to prevent indoor air quality problems, an organized system of record keeping will help those practices to become part of routine operations and to "flag" decisions that could affect IAQ (e.g., renovations, new tenants). The best results can be achieved by taking time to think about the

established channels of communication within your organization, so that new forms can be integrated into decision making with minimum disruption of normal procedures.

Using information from the IAQ profile, the IAQ manager should work with staff and contractors to ensure that building operations and planning processes incorporate a concern for indoor air quality. New procedures, record keeping requirements, or staff training programs may be needed. Growing interest in IAQ is stimulating government agencies and private sector organizations to develop training programs. The flow of information between the IAQ manager and staff, occupants, and contractors is particularly important. Good indoor air quality requires prompt attention to changing conditions that could cause IAQ problems, such as installation of new equipment or furnishings, increases in occupant population, or new uses of rooms.

Facility operation and maintenance

Indoor air quality can be affected both by the quality of maintenance and by the materials and procedures used in operating and maintaining the building components including the HVAC system.

Facility staff who are familiar with building systems in general and with the features of their building in particular are an important resource in preventing and resolving indoor air quality problems. Facility personnel can best respond to indoor air quality concerns if they understand how their activities affect indoor air quality. It may be necessary to change existing practices or introduce new procedures in relation to:

(a) *Equipment operating schedules*: Confirm that the timing of occupied and unoccupied cycles is compatible with actual occupied periods, and that the building is flushed by the ventilation system before occupants arrive. ASHRAE 62-1989 provides guidance on lead and lag times for HVAC equipment. In hot, humid climates, ventilation may be needed during long unoccupied periods to prevent mould growth.

(b) *Control of odours and contaminants*: Maintain appropriate pressure relationships between building usage areas. Avoid recirculating air from areas that are strong sources of contaminants (e.g., smoking lounges, chemical storage areas, beauty salons). Provide adequate local exhaust for activities that produce odours, dust, or contaminants, or confine those activities to locations that are maintained under negative pressure (relative to adjacent areas). For example, loading docks are a frequent source of combustion odours. Maintain the rooms surrounding loading docks under positive pressure to prevent vehicle exhaust from being drawn into the building. Make sure that paints, solvents, and other chemicals are stored and handled properly, with adequate (direct exhaust) ventilation provided. If local filter traps and adsor-

bents are used, they require regular maintenance. Have vendors provide Material Safety Data Sheets (MSDSs).

(c) *Ventilation quantities*: Compare outdoor air quantities to the building design goal and local and State building codes and make adjustments as necessary. It is also informative to see how your ventilation rate compares to ASHRAE 62-1989, because that guideline was developed with the goal of preventing IAQ problems. (Note: Increasing ventilation quantities to meet ASHRAE guidelines may exceed the capacity of HVAC equipment to condition the air.)

(d) *HVAC equipment maintenance schedules*: Inspect all equipment regularly (per recommended maintenance schedule) to ensure that is in good condition and is operating as designed (i.e., as close to the design set points for controls as possible). Most equipment manufacturers provide recommended maintenance schedules for their products. Components that are exposed to water (e.g., drainage pans, coils, cooling towers, and humidifiers) require scrupulous maintenance to prevent microbiological growth and the entry of undesired microbiologicals or chemicals into the indoor airstream.

(e) *HVAC inspections*: Modify the HVAC Checklists (see Fig. 30.2 at the end of this chapter) as necessary so that they are appropriate for inspection of the specific equipment in your building. Be thorough in conducting these inspections. Items such as small exhaust fans may operate independently from the rest of the HVAC system and are often ignored during inspections. As equipment is added, removed, or replaced, document any changes in function, capacity, or operating schedule for future reference. It may also be helpful to store equipment manuals and records of equipment operation and maintenance in the same location as records of occupant complaints for easy comparison if IAQ problems arise.

(f) *Building maintenance schedules*: Try to schedule maintenance activities that interfere with HVAC operation or produce odours and emissions (e.g., painting, roofing operations) so that they occur when the building is unoccupied. Inform occupants when such activities are scheduled and, if possible, use local ventilation to ensure that dust and odours are confined to the work area.

(g) *Purchasing*: Review the general information provided by MSDS and request information from suppliers about the chemical emissions of materials being considered for purchase.

At present there is no general system for certifying or labelling low-emission products nor is there a standard procedure for building managers to use in gathering emissions data on products they are considering for purchase. Limited information on some materials such as pressed-wood products is available, and more may be expected in the future.

Public and private sector organizations are working to develop product

testing procedures for acceptance by such organizations as the American Society for Testing and Materials (ASTM).

30.1.3 Preventive maintenance management

An HVAC system requires adequate preventive maintenance (PM) and prompt attention to repairs in order to operate correctly and provide suitable comfort conditions and good indoor air quality. The HVAC system operator(s) must have an adequate understanding of the overall system design, its intended function, and its limitations. The preventive maintenance program must be properly budgeted and implemented, not merely planned on paper.

A well-implemented PM plan will improve the functioning of the mechanical systems and usually save money when evaluated on a life-cycle basis. However, in some buildings, because of budgetary constraints, maintenance is put off until breakdowns occur or complaints arise, following the "if it isn't broken, don't fix it" philosophy. This type of program represents a false economy and often increases the eventual cost of repairs.

Poor filter maintenance is a common example of this phenomenon. Filters that are not changed regularly can become a bed for fungal growth and a major source of odours, sometimes allowing particles or microorganisms to be distributed within the building. When filters become clogged, the fans use more energy to operate and move less air. If the filters are an inexpensive, low-efficiency type that becomes clogged and then "blows out," the coils then accumulate dirt, causing another increase in energy consumption. Poor air filter efficiency and poor maintenance may cause dirt to build up in ducts and become contaminated with moulds, possibly requiring an expensive duct cleaning operation.

General elements of a PM plan include:

- periodic inspection, cleaning, and service as warranted;
- adjustment and calibration of control system components.
- maintenance equipment and replacement parts that are of good quality and properly selected for the intended function

Critical HVAC system components that require PM in order to maintain comfort and deliver adequate ventilation air include:

- outdoor air intake opening;
- damper controls;
- air filters;
- drip pans;
- cooling and heating coils;
- fan belts;
- humidification equipment and controls;

- distribution systems;
- exhaust fans.

Some private sector organizations have developed guidance on preventive maintenance.

Maintenance "indicators" are available to help facility staff determine when routine maintenance is required. For example, air filters are often neglected, sometimes due to a reasons such as difficult access, and fail to receive maintenance at proper intervals. Installation of an inexpensive manometer, an instrument used to monitor the pressure loss across a filter bank, can give an immediate indication of filter condition without having to open the unit to visually observe the actual filter.

Computerized systems are available that can prompt your staff to carry out maintenance activities at the proper intervals. Some of these programs can be connected to building equipment so that a signal is transmitted to your staff if a piece of equipment malfunctions. Individual areas can be monitored for temperature, air movement, humidity, and carbon dioxide, and new sensors are constantly entering the market. These sensors can be programmed to record data and to control multiple elements of the HVAC system.

Housekeeping

Indoor air quality complaints can arise from inadequate housekeeping that fails to remove dust and other dirt. On the other hand, cleaning materials themselves produce odours and emit a variety of chemicals.

As they work throughout your building, cleaning staff or contractors may be the first to recognize and respond to potential IAQ problems. Educate them about topics such as the following:

(a) *Cleaning schedules*: Consider how cleaning activities are scheduled. Managers may want to schedule the use of some cleaning agents that introduce strong odours or contaminants during unoccupied periods. However, make sure that fumes from cleaning products are eliminated before air handling systems switch to their "unoccupied" cycles.

(b) *Purchasing*: Become more familiar with the chemicals in cleaning and maintenance products and their potential toxicity. Select the safest available materials that can achieve your purpose. Review the information provided by product labels and Material Safety Data Sheets. Request information from suppliers about the chemical emissions of products being considered for purchase.

(c) *Materials handling and storage*: Review the use of cleaning materials to ensure proper use and storage.

(d) *Trash disposal*: Follow proper trash disposal procedures. If there is a restaurant in the building, require daily pick-up of perishable refuse. Ensure

that the containers are recovered, pest control is effective, and that the trash collection area is cleaned at least daily.

(e) *Shipping and receiving*: Shipping and receiving areas can create indoor air quality problems regardless of the types of materials being handled. Vehicle exhaust fumes can be minimized by prohibiting idling at the loading dock. This is particularly important if the loading dock is located upwind of outdoor air intake vents. You can also reduce drafts and pollutant entry by pressurizing interior spaces (e.g., corridors) and by keeping doors closed when they are not in use.

30.1.4 Integrated pest management

Integrated Pest Management (IPM) is a coordinated approach to pest control intended to prevent unacceptable levels of pests, while causing the least possible hazard to people, property, and the environment and using the most cost-effective means. IPM uses a combination of tactics, including sanitation, monitoring, habitat modification, and the judicious application of pesticides when absolutely necessary.

IPM methods include:

- improved sanitation (e.g., removing food from desks, cleaning);
- inspection and monitoring of pest population sites;
- managing waste (e.g., keeping refuse in tight containers, locating waste containers away from building if possible);
- maintaining structures (e.g., fixing leaking pipes promptly, sealing cracks);
- adding physical barriers to pest entry and movement (e.g., screens for chimneys, doors, and windows; air curtains);
- modifying habitats (e.g., removing clutter, relocating outside light fixtures away from doors);
- using traps (e.g., light traps, and glue boards);
- using pesticides judiciously.

An efficient IPM program will integrate pest management planning with preventive maintenance, housekeeping practices, landscaping, occupant education, and staff training.

Pest control

Pest control activities that depend upon the use of pesticides involve the storage, handling, and application of materials that can have serious health effects. Common construction, maintenance practices, and occupant activities provide pests with air, moisture, food, warmth, and shelter. Caulking or plastering cracks, crevices, or holes to prevent harbourage behind walls can

often be more effective than pesticide application at reducing pest populations to a practical minimum.

Integrated pest management (IPM) is a low-cost approach to pest control based upon knowledge of the biology and behaviour of pests. Adoption of an IPM program can significantly reduce the need for pesticides by eliminating conditions that provide attractive habitats for pests. If an outside contractor is used for pest control, it is advisable to review the terms of the contract and include IPM principles where possible. The following items deserve particular attention:

Pest control schedule: Schedule pesticide applications for unoccupied periods, if possible, so that the effected area can be flushed with ventilation air before occupants return. Pesticides should only be applied in target locations, with minimum treatment of exposed surfaces. They should be used in strict conformance with manufacturers' instructions and EPA labels. General periodic spraying may not be necessary. If occupants are to be present, they should be notified prior to the pesticide application. Particularly susceptible individuals could develop serious illness even though they are only minimally exposed.

Materials selection, handling, and storage: Select pesticides that are species-specific and attempt to minimize toxicity for humans and non-target species. Ask contractors or vendors to provide EPA labels and MSDSs. Make sure that pesticides are stored and handled properly consistent with their EPA labels.

Ventilation of areas where pesticides are applied: If only limited areas of the building are being treated, adjust the HVAC system so that it does no distribute contaminated air throughout the rest of the building. Consider using temporary exhaust systems to remove contaminants during the work. It may be necessary to modify HVAC system operation during and after pest control activities, e.g., running air handling units on 100% outdoor air for some period of time or running the system for several complete air exchanges before occupants re-enter the treated space.

30.1.5 Material safety data sheets

Under OSHA regulations, responsible parties are required to document information on potentially hazardous products. These Material Safety Data Sheets (MSDSs) may be of limited help in identifying some products that may pose IAQ concerns. However, professional judgment and collection of additional information may be necessary in order to make full use of the MSDS. Table 30.1 summarizes some of the issues to keep in mind when deciding whether information from MSDSs is applicable to emission sources and exposures of concern in a building.

TABLE 30.1

Applicability of material safety data sheets

Item	Possible uses	Comments
Substances covered	MSDSs may identify significant airborne contaminants	MSDSs may not be available on-site for many products
		some components are listed as proprietary and are not disclosed
		MSDSs do not always highlight products most likely to be airborne
		contaminant byproducts inadvertently formed during manufacture will not always be listed
Personal protection / first aid	may suggest precautions for conducting source inspection	usually relates only to high-level, worst-case exposures in general industry
Health effects	generally presents types of health effects that may be expected primarily at high level (e.g. industrial) exposures	symptoms listed may not occur at low-level concentrations found in indoor air
		MSDSs may not include more subtle IAQ aspects such as nuisance factors and sensitivity to mixtures
Physical data	odour description may help identify sources	reference material on how to use physical data information to predict IAQ impacts may be scarce
	volatility may suggest which products are likely to be airborne	
	contaminants to expect in event of a fire or decomposition may be listed	
	reactivity data may suggest potential problems with storage or use	
Control measures	identifies proper storage and packaging procedures	many office chemicals are kept in much smaller amounts than found in industrial setting
	identifies steps for cleanup of gross spills	spill cleanup may not eliminate airborne contamination
		does not specify routine emission controls

A reasonable effort should be made to collect available MSDSs during IAQ profile development. Care should be taken to consider information that is relevant to IAQ concerns. Other important indicators of how a particular product may affect IAQ are available from direct odour and dust observations, a review of work practices, and interviews with operators and occupants. The manufacturer is a good source of follow-up information on a given product.

30.1.6 Occupant relations

Managing occupant relations to prevent IAQ problems involves: allocating space and monitoring the use of building areas to isolate odour- and contaminant-producing activities and avoid re-entrainment; establishing a communication strategy that is responsive to complaints and provides tenants with information about their role in preventing indoor air quality problems; and modifying employee manuals or lease agreements as necessary to clarify the responsibilities of occupants and building management. A health and safety committee or joint tenant- management IAQ task force the represent all of the major interest groups in the building can be very helpful in disseminating information and fostering a cooperative approach to IAQ management.

30.1.7 Renovation, redecorating, and remodelling

Renovation, redecorating, and remodelling activities can create indoor air problems by producing dust, odours, microbiologicals and their spores, and emissions. It is difficult to prevent IAQ problems if some building areas are undergoing renovation while adjoining areas continue normal operations.

Close monitoring of renovation, redecorating, and remodelling projects is recommended. The following suggestions may be helpful:

Working with professional consultants: Communicate your concern about preventing indoor air quality problems to the engineer, architect, interior designer, or other professionals involved in the project.

Product selection: Specify products and processes that minimize odours and emissions, while maintaining adequate safety and efficacy. Review the general information provided by the product labels and MSDSs. Request information from suppliers about the chemical emissions of products being considered for purchase.

Work schedules: Schedule activities that produce dust, odours, or emissions for unoccupied periods if possible.

Isolation of work areas: Block off return register so that contaminants are not recirculated from the demolition/construction area into adjoining areas, and install temporary barriers to confine dust and noise. If possible, install temporary local exhaust to remove odours and contaminants, and check to confirm that the temporary ventilation system is operating as planned.

Installation of new furnishings: Ask suppliers to store new furnishings in a clean, dry, ventilated location so that volatile organic compounds will be emitted before installation. Minimize the use of adhesives during installation or specify low-emitting products. After new furnishings are installed, increase the ventilation rate to flush the area with outdoor air and dilute emissions.

30.1.8 Smoking

Although there are many potential sources of indoor air pollution, both research and field studies have shown that environmental tobacco smoke (ETS) is one of the most widespread and harmful indoor air pollutants. Environmental tobacco smoke is a combination of sidestream smoke from the burning end of the cigarette, pipe, or cigar and the exhaled mainstream smoke from the smoker. ETS contains over 4000 chemicals; 43 of these chemicals are known animal or human carcinogens. Many other chemicals in ETS are tumour promoters, tumour initiators, co-carcinogens or cancer precursors. For more information on ETS composition, see Chapter 4.

In 1986, "The Health Consequences of Involuntary Smoking: A Report of the Surgeon General on Environmental Tobacco Smoke concluded" that ETS was a cause of lung cancer in healthy non-smokers and that "the scientific case against involuntary smoking as a public health risk is more than sufficient to justify appropriate remedial action, and the goal of any remedial action must be to protect the non-smoker from environmental tobacco smoke." In the same year, the National Research Council of the National Academy of Sciences issued a report, "Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects", which also concluded that passive smoking increases the risk of lung cancer in adults.

In June 1991, NIOSH issued a Current Intelligence Bulletin (#54) on ETS in the workplace that dealt with lung cancer and other health effects. In its Bulletin, NIOSH stated that the weight of evidence is sufficient to conclude that ETS can cause lung cancer in non-smokers (i.e., those who inhale ETS). It recommended that the preferable method to protect non-smokers is the elimination of smoking indoors and that the alternative method is to require that smoking be permitted only in separately ventilated smoking areas. The NIOSH Bulletin emphasized that provision of such isolated areas should be viewed as an interim measure until ETS can be completely eliminated indoors.

In January, 1993, EPA declared ETS a Class A Carcinogen. Smoking areas must be separately ventilated, negatively pressurized in relation to surrounding interior spaces, and supplied with much more ventilation than non-smoking areas. The NIOSH Bulletin also recommends that the air from the smoking area should be exhausted directly outdoors and not recirculated within the

building or vented with the general exhaust for the building. ASHRAE Standard 62-1989 recommends that smoking areas be supplied with 60 cubic feet per minute (60 cfm) per occupant of outdoor air; the standard also recognized that using transfer air, which is pulled in from other parts of the building, to meet the standard is common practice.

Both EPA and NIOSH advise that building owners or facility managers considering the introduction of smoking restrictions should implements smoking cessation programs. In addition, employees and labour unions should be involved in the development of non-smoking policies in the workplace.

30.2 MITIGATING INDOOR AIR QUALITY PROBLEMS

Over the years many types of mitigation (correction) strategies have been implemented to solve indoor air quality problems. The purpose of this section is to provide an understanding of basic approaches to mitigation and the various solutions that can be effective in treating commonly encountered IAQ problems. It is not intended to provide detailed instructions for using each type of mitigation approach but rather to give guidance in selecting a mitigation strategy and in judging proposals from in-house staff or outside consultants. Further details on specific aspects will be dealt in Chapter 31.

Mitigation of indoor air quality problems may require the involvement of building management and staff representing such areas of responsibility as:

- facility operation and maintenance;
- housekeeping;
- shipping and receiving;
- purchasing;
- policy making;
- staff training.

Successful mitigation of IAQ problems also requires the cooperation of other building occupants, including the employees of building tenants. Occupants must be educated about the cause(s) of the IAQ problems and about actions that must be taken or avoided to prevent a recurrence of the problems.

30.2.1 Introduction

Earlier sections introduced the idea that indoor air quality problems result from interactions between contaminant source, building site, building structure, activities within the building, mechanical equipment, climate, and occupants. Efforts to control indoor air contaminants change the relationships between these factors. There are many ways that people can intervene in these

relationships to prevent or control indoor air contaminant problems. Control strategies can be categorized as:

- source control;
- ventilation;
- air cleaning;
- exposure control.

Successful mitigation often involves a combination of these strategies. Possible remedies for the other environmental stressors are discussed briefly below.

30.2.2 Source control

All efforts to prevent or correct IAQ problems should include an effort to identify and control pollutant sources. Source control is generally the most cost effective approach to mitigating IAQ problems in which point sources of contaminants can be identified. In the case of a strong source, source control may be the only solution that will work.

The following are categories and examples of source control:

Remove or reduce the source

- prohibit smoking indoors or limit smoking to areas from which air is exhausted, not recirculated
- relocate contaminant-producing equipment to an unoccupied, better ventilated, or exhaust-only ventilated space
- select products which produce fewer or less potent contaminants while maintaining adequate safety and efficacy
- modify other occupant activities

Seal or cover the source

- improve storage of materials that produce contaminants
- seal surfaces of building materials that emit VOCs such as formaldehyde

Modify the environment

- after cleaning and disinfecting an area that is contaminated by fungal or bacterial growth, control humidity to make conditions inhospitable for regrowth

Source removal or reduction can sometimes be accomplished by a one-time effort such as thorough cleaning of a spill. In other cases, it requires an ongoing process, such as establishing and enforcing a non-smoking policy.

Sealing or covering the source can be a solution in some cases; application of a barrier over formaldehyde-emitting building materials is an example. Sealing may also involve educating staff or building occupants about the contaminant-producing features of materials and supplies and inspecting storage areas to ensure that containers are properly covered.

In some cases, modification of the environment is necessary for effective mitigation. If the indoor air problem arises from microbiological contaminants, for example, disinfection of the affected area may not eliminate the problem. Regrowth of microbiologicals is like to occur unless humidity control or other steps, such as adding insulation to prevent surface condensation, are taken to make the environment inhospitable to microbiologicals.

30.2.3 Ventilation

Ventilation modification is often used to correct or prevent indoor air quality problems. This approach can be effective either where buildings are underventilated or where a specific contaminant source cannot be identified. Ventilation can be used to control indoor air contaminants by:

Diluting contaminants with outdoor air

- increase the total quantify of supply air (including outdoor air);
- increase the proportion of outdoor air to total air;
- improve air distribution.

Isolating or removing contaminants by controlling air pressure relationships

- install effective local exhaust at the location of the source;
- avoid recirculation of the air that contains contaminants;
- locate occupants near supply diffuser and sources near exhaust registers;
- use air-tightening techniques to maintain pressure differentials and eliminate pollutant pathways;
- make sure that doors are closed where necessary to separate zones.

Diluting contaminants by increasing the flow of outdoor air can be accomplished by increasing the total supply airflow in the complaint area (e.g., opening supply diffuser, adjusting dampers) or at the air handling unit, (e.g., cleaning the filter on the supply fan). An alternative is to increase the proportion of outdoor air (e.g., adjusting the outdoor air intake damper, installing minimum stops on variable air volume (VAV) boxes so that they satisfy the outdoor air requirements of ASHRAE 62-1989).

Studies have shown that increasing ventilation rates to meet ASHRAE Standard 62-1989 (e.g., from 5 to 15 or 20 cfm/person) does not necessarily significantly increase the total annual energy consumption. The increase appears to be less than 5% in typical commercial buildings. The cost of ventilation is generally overshadowed by other operating costs, such as lighting. Further, improved maintenance can produce energy savings to balance the costs that might otherwise result from increased ventilation.

The cost of modifying an existing HVAC system to condition additional outdoor air can vary widely depending upon the specific situation. In some buildings, HVAC equipment may not have sufficient capacity to allow success-

ful mitigation using this approach. Original equipment is often oversized so that it can be adjusted to handle the increased load, but in some cases additional capacity is required.

Most ventilation deficiencies appear to be linked to inadequate quantities of outdoor air. However, inadequate distribution of ventilation air can also produce IAQ problems. Diffuser should be properly selected, located, installed, and maintained so that supply air is evenly distributed and blends thoroughly with room air in the breathing zone. Short-circuiting occurs when clean supply air is drawn into the return air plenum before it has mixed with the dirtier room air and therefore fails to dilute contaminants. Mixing problems can be aggravated by temperature stratification. Stratification can occur, for example, in a space with high ceilings in which ceiling-mounted supply diffusers distribute heated air.

Note the side effects of increased ventilation:

- Mitigation by increasing the circulation of outdoor air requires good outdoor air quality.
- Increased supply air at the problem location might mean less supply air in other areas.
- Increased total air in the system and increased outdoor air will both tend to increase energy consumption and may require increased equipment capacity.
- Any approach which affects airflow in the building can change pressure differences between rooms (or zones) and between indoors and outdoors, and might lead to increased infiltration of unconditioned outdoor air.
- Increasing air in a VAV system may overcool an area to the extent that terminal reheat units are needed.

Ventilation equipment can be used to isolate or remove contaminants by controlling pressure relationships. If the contaminant source has been identified, this strategy can be more effective than dilution. Techniques for controlling air pressure relationships range from adjustment of dampers to installation of local exhaust.

Using local exhaust confines the spread of contaminants by capturing them near the source and exhausting them to the outdoors. It also dilutes the contaminant by drawing cleaner air from surrounding areas into the exhaust airstream. If there are return grilles in a room equipped with local exhaust, the local exhaust should exert enough suction to prevent recirculation of contaminants. Properly designed and installed local exhaust results in far lower contaminant levels in the building than could be accomplished by a general increase in dilution ventilation, with the added benefit of costing less.

Note that replacement air must be able to flow freely into the area from which the exhaust air is being drawn. It may be necessary to add door or wall

louvres in order to provide a path for the make-up air. Make sure that this action does not violate fire codes. Correct identification of the pollutant source and installation of the local exhaust is critically important. For example, an improperly designed local exhaust can draw other contaminants through the occupied space and make the problem worse. The physical layout of grilles and diffusers relative to room occupants and pollutant sources can be important. If supply diffusers are all at one end of a room and returns are all at the other end the people located near the supplies may be provided with relatively clean air while those located near the returns breathe air that has already picked up contaminants from all the sources in the room that are not served by local exhaust.

Elimination of pollutant pathways by air sealing (e.g., caulking cracks, closing holes) is an approach that can increase the effectiveness of other control techniques. It can be a difficult technique to implement because of hidden pathways, e.g., above drop ceilings, under raised flooring, against brick or block walls. However, it can have other benefits such as energy savings and more effective pest control by eliminating paths used by vermin.

30.2.4 Air cleaning

The third IAQ control strategy is to clean the air. Air cleaning is usually most effective when used in conjunction with either source control or ventilation; however, it may be the only approach when the source of pollution is outside of the building. Most air cleaning in large buildings is aimed primarily at preventing contaminant build-up in HVAC equipment and enhancing equipment efficiency.

Air cleaning equipment intended to provide better indoor air quality for occupants must be properly selected and designed for the particular pollutants of interest (for example, gaseous contaminants can be removed only by gas sorption). Once installed, the equipment requires regular maintenance in order to ensure good performance; otherwise it may become a major pollutant source in itself. This maintenance requirement should be borne in mind if an air cleaning system involving a large number of units is under consideration for a large building. If room units are used, the installation should be designed for proper air recirculation.

There are four technologies that remove contaminants from air:

- particulate filtration;
- electrostatic precipitation;
- negative ion generation;
- gas sorption.

The first three approaches are designed to remove particulates, while the fourth is designed to remove gases.

Particulate filtration removes suspended liquid or solid materials whose size, shape and mass allow them to remain airborne at the air velocity conditions present. Filters are available in a range of efficiencies, with higher efficiency indicating removal of a greater proportion of particles and of smaller particles. Moving to medium efficiency pleated filters is advisable to improve IAQ and increase protection for equipment. However, the higher the efficiency of the filter, the more it will increase the pressure drop within the air distribution system and reduce total airflow (unless other adjustments are made to compensate). It is important to select an appropriate filter for the specific application and to make sure that the HVAC system will continue to perform as designed. Filters are rated by different standards (e.g., arrestance and dust spot) which measure different aspects of performance (see also Chapters 28 and 29).

Electrostatic precipitation is another type of particulate control. It uses the attraction of charged particles to oppositely charged surfaces to collect airborne particulates. In this process, the particles are charged by ionizing the air with an electric field. The charged particles are then collected by a strong electric field generated between oppositely-charged electrodes. This provides relatively high efficiency filtration of small respirable particles at low air pressure losses.

Electrostatic precipitators may be installed in air distribution equipment or in specific usage areas. As with other filters, they must be serviced regularly. Note, however, that electrostatic precipitators produce some ozone. Because ozone is harmful at elevated levels, EPA has set standards for ozone concentrations in outdoor air, and NIOSH and OSHA have established guidelines and standards, respectively, for ozone in indoor air. The amount of ozone emitted from electrostatic precipitators varies from model to model.

Negative ion generators use static charges to remove particles from the indoor air. When the particles become charged, they are attracted to surface such as walls, floors, table tops, draperies, and occupants. Some designs include collectors to attract the charged particles back to the unit. Negative ion generators are not available for installation in ductwork, but are sold as portable or ceiling-mounted units. As with electrostatic precipitators, negative ion generators may produce ozone, either intentionally or as a by-product of use. They also have the disadvantage that in principle all room surfaces are used to collect particles and become soiled.

Gas sorption is used to control compounds that behave as gases rather than as particles (e.g., gaseous contaminants such as formaldehyde, sulphur dioxide, ozone, and oxides of nitrogen). Gas sorption involves one or more of the

following processes with the sorption material (e.g., activated carbon, chemically treated active clays): a chemical reaction between the pollutant and the sorbent; a binding of the pollutant and the sorbent; or diffusion of the contaminant from areas of higher concentration to areas of lower concentration. Gas sorption units are installed as part of the air distribution system. Each type of sorption material performs differently with different gases. Gas sorption is not effective from removing carbon monoxide. There are no standards for rating the performance of gaseous air cleaners, making the design and evaluation of such systems problematic. Operating expenses of these units can be quite high, and the units may not be effective if there is a strong source nearby.

30.2.5 Exposure control

Exposure control is an administrative approach to mitigation that uses behavioral methods, such as:

Scheduling contaminant-producing activities to avoid complaints:

- schedule contaminant-producing activities to occur during unoccupied periods;
- notify susceptible individuals about up-coming events (e.g., roofing, pesticide application) so that they can avoid contact with the contaminants.

Scheduling contaminant-producing activities for unoccupied periods whenever possible is simple common sense. It may be the best way to limit complaints about activities, such as roofing or demolition, which unavoidably produce odours or dust.

Relocating susceptible individuals:

- move susceptible individuals away from the area where they experience symptoms.

Controlling exposure by relocating susceptible individuals may be the only practical approach in a limited number of cases, but it is probably the least desirable option and should be used only when all other strategies are ineffective in resolving complaints, or when protecting very sensitive individuals.

30.2.6 Remedies for complaints not attributed to poor indoor air quality

Specific lighting deficiencies or localized sources of noise or vibration can sometimes be readily identified, and remedial action may be fairly straightforward (more or fewer lights on, adjustments for glare; relocating, replacing or acoustically insulating a noise or vibration source). Similarly, flagrant ergonomic stress or blatant psychosocial stress may be apparent even to an untrained observer.

In other cases, however, problems may be more subtle or solutions more complex. Since specialized knowledge, skills, and instrumentation are usually needed to evaluate lighting, noise, vibration, ergonomic stress, or psychosocial stress, such evaluations are generally best done by a qualified professional in that particular field.

Remedial actions for lighting, noise, and vibration problems might range from modifications of equipment or furnishings to renovation of the building. Ergonomic deficiencies may require furniture or equipment changes or different work practices. The solution to psychosocial problems may involve new management practices, job redesign, or resolution of underlying labour- management problems.

30.2.7 Judging proposed mitigation designs and their success

Mitigation efforts should be evaluated at the planning stage by considering the following criteria:

- permanence;
- operating principle;
- degree to which the strategy fits the job;
- ability to institutionalize the solution;
- durability;
- installation and operating costs;
- conformity which codes.

Permanence

Mitigation efforts that create permanent solutions to indoor air problems are clearly superior to those that provide temporary solutions (unless the problems are also temporary). Opening windows or running air handlers on full outdoor may be suitable mitigation strategies for a temporary problem such as outgassing of volatile compounds from new furnishings, but would not be good ways to deal with emissions from a print shop. A permanent solution to microbiological contamination involves not only cleaning and disinfection, but also modification of the environment to prevent regrowth.

Operating principle

The most economical and successful solution to IAQ problems are those in which the operating principle of the correction strategy makes sense and is suited to the problem. If a specific point source of contaminants has been identified, treatment at the source (e.g., by removal, sealing, or local exhaust) is almost always a more appropriate correction strategy than dilution of the contaminant by increased general ventilation. If the IAQ problem is caused by

the introduction of outdoor air that contains contaminants, increased general ventilation will only make the situation worse, unless the outdoor air is cleaned.

Degree to which the strategy fits the job

It is important to make sure that you understand the IAQ problem well enough to select a correction strategy whose size and scope fit the job. If odours from a special use area such as a kitchen are causing complaints in a nearby office, increasing the ventilation rate in the office may not be a successful approach. The mitigation strategy should address the entire area affected.

If mechanical equipment is needed to correct the IAQ problem, it must be powerful enough to accomplish the task. For example, a local exhaust system should be strong enough and close enough to the source so that none of the contaminant is drawn into nearby returns and recirculated.

Ability to institutionalize the solution

A mitigation strategy will be most successful when it is institutionalized as part of normal building operations. Solutions that do not require exotic equipment are more likely to be successful in the long run than approaches that involve unfamiliar concepts or delicately maintained systems. If maintenance or housekeeping procedures or supplies must change as part of the mitigation, it may be necessary to plan for additional staff training, new inspection checklists, or modified purchasing practices. Operating schedules for HVAC equipment may also require modification.

Durability

IAQ mitigation strategies that are durable and low-maintenance are more attractive to owners and building staff than approaches that require frequent adjustment or specialized maintenance skills. New items of equipment should be quiet, energy-efficient, and durable, so that the operators are encouraged to keep them running.

Installation and operating costs

The approach with the lowest initial cost may not be the least expensive over the long run. Other economic considerations include: energy costs of equipment operations; increased staff time for maintenance; differential cost of alternative materials and supplies; and higher hourly rates if odour-producing activities (e.g., cleaning) must be scheduled for unoccupied periods. Although these costs will almost certainly be less than the cost of letting the problem continue, they are more readily identifiable, so an appropriate presentation to management may be required.

Conformity with codes

Any modification to building components or mechanical system should be designed and installed in keeping with applicable fire, electrical, and other building codes.

Judging the success of a mitigation effort

Two kinds of criteria can be used to judge to success of an effort to correct an indoor air problem:

- reduced complaints;
- measurement of properties of the indoor air (often only of limit usefulness).

Reduction or elimination of complaints appears to be a clear indication of success, but that is not necessarily the case.

Occupants who see that their concerns are being heard may temporarily stop reporting discomfort or health symptoms, even if the actual cause of their complaints has not been addressed. Lingering complaints may also continue after successful mitigation if people have become upset over the handling of the problem. Ongoing (but reduced)complaints could also indicate that there were multiple IAQ problems and that one or more problems are still unresolved.

However, it can be difficult to use measurements of contaminant levels as a means of determining whether air quality has improved. Concentrations of indoor air pollutants typically vary greatly over time; further, the specific contaminant measured may not be causing the problem. If air samples are taken, ratings taken before and after mitigation should be interpreted cautiously. It is important to keep the "before" and "after" conditions as identical as possible, except for the operation of the control strategy. For example, the same HVAC operation, building occupancy and climatic conditions should apply during both measurement periods. "Worst-case" conditions identified during the investigation should be used.

Measurements of airflows, ventilation rates, and air distribution patterns are the more reliable methods of assessing the results of control efforts. Airflow measurements taken during the building investigation can identify areas with poor ventilation; later they can be used to evaluate attempts to improve the ventilation rate, distribution, or direction of flow. Studying air distribution patterns will show whether a mitigation strategy has successfully prevented a contaminant from being transported by airflow.

Persistent problems

Solving an indoor air quality problem is a cyclical process of data collection and hypothesis testing. Deeper and more detailed investigation is needed to

suggest new hypotheses after any unsuccessful or partially-successful control attempt.

Even the best-planned investigations and mitigation actions may not produce a resolution to the problem. You may have made a careful investigation, found one or more apparent causes for the problem, and implemented a control system. Nonetheless, your correction strategy may not have caused a noticeable reduction in the concentration of the contaminant or improvement in ventilation rates or efficiency. Worse, the complaints may persist even though you have been successful at improving ventilation and controlling all of the contaminants you could identify. When you have pursued source control options and have increased ventilation rates and efficiency to the limits of your expertise, you must decide how important it is to pursue the problem further.

If you have made several unsuccessful efforts to control a problem, then it may be advisable to seek outside assistance. The problem is probably fairly complex, and it may occur only intermittently or cross the borders that divide traditional fields of knowledge. Is it even possible that poor indoor air quality is not the actual cause of the complaints. Bringing in a new perspective at this point can be very effective. An interdisciplinary team (such as people with engineering and medical or health backgrounds) may be needed to solve particularly difficult problems.

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Appendix B discusses HVAC system components in relation to indoor air quality.

Component	OK	Needs Attention	Not Applicable	Comments
Outside Air Intake				
Location _____				
Open during occupied hours?				
Unobstructed?				
Standing water, bird droppings in vicinity?				
Odors from outdoors? (describe) _____				
Carryover of exhaust heat?				
Cooling tower within 25 feet?				
Exhaust outlet within 25 feet?				
Trash compactor within 25 feet?				
Near parking facility, busy road, loading dock?				
Bird Screen				
Unobstructed?				
General condition?				
Size of mesh? (½" minimum)				
Outside Air Dampers				
Operation acceptable?				
Seal when closed?				

Fig. 30.2: HVAC checklist — long form.

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Actuators operational?				
Outdoor Air (O.A.) Quantity ***(Check against applicable codes and ASHRAE 62-1989.)***				
Minimum % O.A. _____				
Measured % O.A. _____ *Note day, time, HVAC operating mode under "Comments"*				
Maximum % O.A. _____				
Is minimum O.A. a separate damper?				
For VAV systems: is O.A. increased as total system air-flow is reduced?				
Mixing Plenum				
Clean?				
Floor drain trapped?				
Airtightness				
▪ of outside air dampers				
▪ of return air dampers				
▪ of exhaust air dampers				
All damper motors connected?				
All damper motors operational?				
Air mixers or opposed blades?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Mixed air temperature control setting _____°F				
Freeze stat setting _____°F				
Is mixing plenum under negative pressure? *Note: If it is under positive pressure, outdoor air may not be entering.*				
Filters				
Type _____				
Complete coverage? (i.e., no bypassing)				
Correct pressure drop? *(Compare to manufacturer's recommendations.)*				
Contaminants visible?				
Odor noticeable?				
Spray Humidifiers or Air Washers				
Humidifier type				
All nozzles working?				
Complete coil coverage?				
Pans clean, no overflow?				
Drains trapped?				
Biocide treatment working? *Note: Is MSDS on file?*_____				
Spill contaminant system in place?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Face and Bypass Dampers				
Damper operation correct?				
Damper motors operational?				
Cooling Coil				
Inspection access?				
Clean?				
Supply water temp. ____°F				
Water carryover?				
Any indication of condensation problems?				
Condensate Drip Pans				
Accessible to inspect and clean?				
Clean, no residue?				
No standing water, no leaks?				
Noticeable odor?				
Visible growth (e.g., slime)?				
Drains and traps clear, working?				
Trapped to air gap?				
Water overflow?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Mist Eliminators				
Clean, straight, no carryover?				
Supply Fan Chambers				
Clean?				
No trash or storage?				
Floor drain traps are wet or sealed?				
No air leaks?				
Doors close tightly?				
Supply Fans				
Location _____				
Fan blades clean?				
Belt guards installed?				
Proper belt tension?				
Excess vibration?				
Corrosion problems?				
Controls operational, calibrated?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Control sequence conforms to design/specifications? (describe changes)				
No pneumatic leaks?				
Heating Coil				
Inspection access?				
Clean?				
Control sequence conforms to design/specifications? (describe changes)				
Supply water temp. ____°F				
Discharge thermostat? (air temp. setting ____°F)				
Reheat Coils				
Clean?				
Obstructed?				
Operational?				
Steam Humidifier				
Humidifier type _____				
Treated boiler water?				
Standing water?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Visible growth?				
Mineral deposits?				
Control setpoint ——— °F				
High limit setpoint ——— °F				
Duct liner within 12 feet? (If so, check for dirt, mold growth.)				
Supply Ductwork				
Clean?				
Sealed, no leaks, tight connections?				
Fire dampers open?				
Access doors closed?				
Lined ducts?				
Flex duct connected, no tears?				
Light troffer supply?				
Balanced within 3-5 years?				
Balanced after recent renovations?				
Short circuiting or other air distribution problems? Note location(s) _____ _____				
Pressurized Ceiling Supply Plenum				
No unintentional openings?				
All ceiling tiles in place?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Barrier paper correctly placed and in good condition?				
Proper layout for air distribution?				
Supply diffusers open?				
Supply diffusers balanced?				
Balancing capability?				
Noticeable flow of air?				
Short circuiting or other air distribution problems? *Note location(s) in "Comments"*				
Terminal Equipment (supply)				
Housing interiors clean and unobstructed?				
Controls working?				
Delivering rated volume?				
Balanced within 3-5 years?				
Filters in place?				
Condensate pans clean, drain freely?				

VAV Box

Minimum stops _____ %				
Minimum outside air ___ % *(from page 2 of this form)*				
Minimum airflow _____ cfm				
Minimum outside air _____ cfm				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Supply setpoint ____°F (summer) ____°F (winter)				
Thermostats				
Type _____				
Properly located?				
Working?				
Setpoints ____°F (summer) ____°F (winter)				
Space temperature ____°F				
Humidity Sensor				
Humidistat setpoints ____ % RH				
Dehumidistat setpoints ____ % RH				
Actual RH ____ %				
Room Partitions				
Gap allowing airflow at top?				
Gap allowing airflow at bottom?				
Supply and return each room?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Stairwells				
Doors close and latch?				
No openings allowing uncontrolled airflow?				
Clean, dry?				
No noticeable odors?				
Return Air Plenum				
Tiles in place?				
No unintentional openings?				
Return grilles?				
Balancing capability?				
Noticeable flow of air?				
Transfer grilles?				
Fire dampers open?				
Ducted Returns				
Balanced within 3-5 years?				
Unobstructed grilles?				
Unobstructed return air path?				
Return Fan Chambers				
Clean and no trash or storage?				
No standing water?				
Floor drain traps are wet or sealed?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
No air leaks?				
Doors close tightly, kept closed?				
Return Fans				
Location _____				
Fan blades clean?				
Belt guards installed?				
Proper belt tension?				
Excess vibration?				
Corrosion problems?				
Controls working, calibrated?				
Control sequence conforms to design/specifications? (describe changes)				
Exhaust Fans				
Central?				
Distributed (locations) _____ _____				
Operational?				
Controls operational?				
Toilet exhaust only?				
Gravity relief?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Total powered exhaust _____ cfm				
Make-up air sufficient?				
Toilet Exhausts				
Fans working occupied hours?				
Registers open, clear?				
Make-up air path adequate?				
Volume according to code?				
Floor drain traps wet or sealable?				
Bathrooms run slightly negative relative to building?				
Smoking Lounge Exhaust				
Room runs negative relative to building?				
Print Room Exhaust				
Room runs negative relative to building?				
Garage Ventilation				
Operates according to codes?				
Fans, controls, dampers all operate?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
Garage slightly negative relative to building?				
Doors to building close tightly?				
Vestibule entrance to building from garage?				
Mechanical Rooms				
General condition?				
Controls operational?				
Pneumatic controls:				
▪ compressor operational?				
▪ air dryer operational?				
Electric controls? Operational?				
EMS (Energy Management System) or DDC (Direct Digital Control):				
▪ operator on site?				
▪ controlled off-site?				
▪ are fans cycled "off" while building is occupied?				
▪ is chiller reset to shed load?				
Preventive Maintenance				
Spare parts inventoried?				
Spare air filters?				
Control drawing posted?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments
PM (Preventive Maintenance) schedule available?				
PM followed?				
Boilers				
Flues, breeching tight?				
Purge cycle working?				
Door gaskets tight?				
Fuel system tight, no leaks?				
Combustion air: at least 1 square inch free area per 2000 Btu input?				
Cooling Tower				
Sump clean?				
No leaks, no overflow?				
Eliminators working, no carryover?				
No slime or algae?				
Biocide treatment working?				
Dirt separator working?				
Chillers				
No refrigerant leaks?				
Purge cycle normal?				
Waste oil, refrigerant properly disposed of and spare refrigerant properly stored?				
Condensation problems?				

Fig. 30.2: HVAC checklist — long form (*continued*).

Building: _____ File Number: _____

Completed by: _____ Title: _____ Date Checked: _____

Component	OK	Needs Attention	Not Applicable	Comments

Fig. 30.2: HVAC checklist — long form (*continued*).

Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 31

Specific Aspects of IAQ and Climate Control

31.1 MOISTURE CONTROL

Moisture in buildings exists in both liquid and vapour phases. Liquid moisture stems from the use of water, stormwater, meltwater and groundwater leaks from the environment of the building, water or wastewater leaks from damaged plumbing or a leaking faucet inside the building, and condensation of water from humid air on cold visible or hidden surfaces. Vapour phase moisture stems from metabolism of people and pets, drying of clothes or any wetted surface, and from outdoor air. Normal living in houses brings in fairly large amounts of water per each day and occupant, and all this moisture needs to be rapidly drained or ventilated out of the building to prevent build-up. Moisture can evaporate, condense and re-evaporate a number of times on its way out of the building depending on air circulation, ventilation rate, and surface temperatures.

The movement of moisture in, around, and through the envelope of the house should be a prime consideration in the design and construction of the house. Air humidity should be sufficient to prevent drying and cracking of wood and irritation to the mucous membranes, but it must be controlled to prevent biological contamination (NATO/CCMS, 1993).

To prevent moisture damage, which may result in rapid deterioration of the affected building constructions, equipment, and finishings, and health damage to the building occupants, all sources of moisture accumulation must be controlled in the design, construction, maintenance and use of the building. In cold climates, to avoid moisture damage to structures, and health hazards caused by microorganisms, the general practice is to design the building for a slight underpressure of no more than 30 Pa (Finnish Building Regulation Code 1987).

Moisture accumulation in buildings can result for several different reasons (see also Fig. 31.1):

- too high relative humidity (RH) of the air;
- moisture remaining from the construction of the building (new buildings), or from fire-fighting;

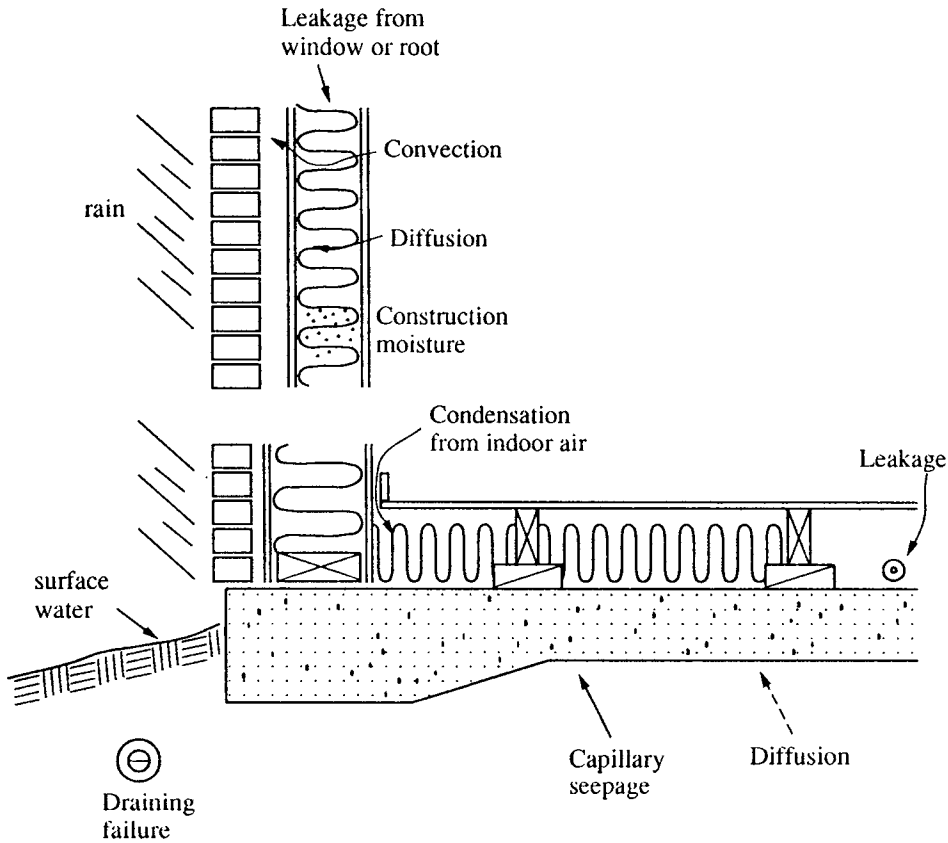


Fig. 31.1: Mechanisms of moisture accumulation in the wall and concrete slab floor structures of a building.

- moisture condensing on cold surfaces and into non-visible constructions of the building;
- water leaking from drinking, hot water, central heating, or wastewater pipes, joints, valves, etc.;
- storm- or meltwater leaking through the roof or walls;
- groundwater seeping or leaking through basement walls or concrete slab;
- water, used in kitchens, laundries and bathrooms, remaining on the surfaces due to poor ventilation, missing drains, or incorrectly sloped floors, or seeping into porous surface materials and underlying constructions.

Everyday human activities result in large amounts of water being evaporated in buildings, and the resulting humidity needs to be diluted by ventila-

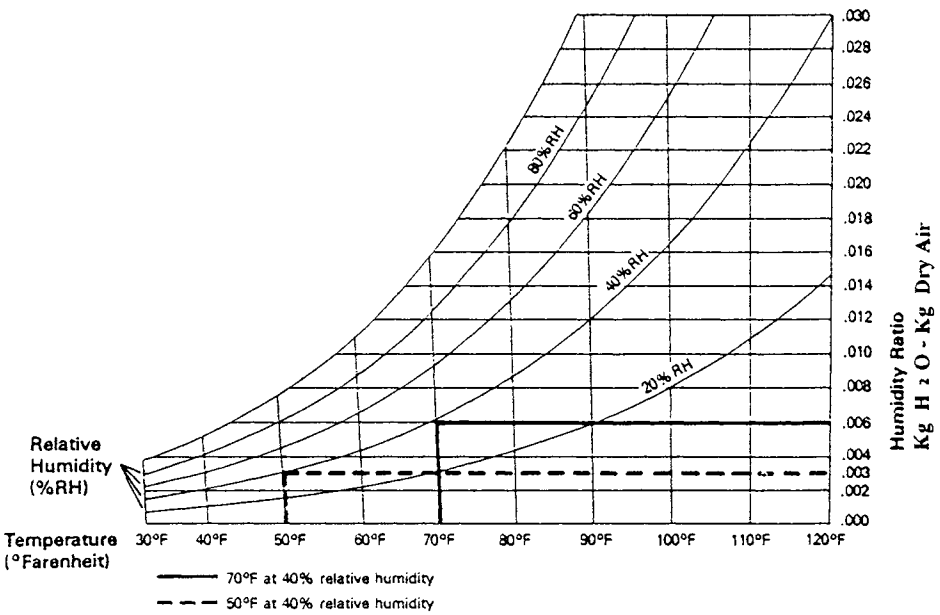


Fig. 31.2: Relationship of temperature, relative humidity, and moisture in the air.

tion to levels, where the dew point of water stays below the coldest surface temperature in the space. The emission rate of water vapour in buildings is primarily determined by the presence and activities of occupants and by the use of unflued heating equipment. The current, generally accepted values in the U.K. are a normal daily total of 5–10 kg/occupant, max 10–20 kg/occupant. Other studies about German and Canadian houses are in broad agreement with the values reported from the U.K. (Samuelsson, 1985). The water vapour produced by a room humidifier could be about 20 kg/24 h, unvented gas/kerosene appliances produce about 1.6/1.2 kg of water vapour per each m^3/kg of gas/kerosene burned. Washing and rinsing a 2.5 × 3.5 m room produces about 1 kg of water vapour, drying 3.5 kg of clothes produces about the same amount, and 4 occupants in the home produce about 1.3 kg of water vapour each day (Hedden, 1982).

The relationships between water content of air (kg H_2O/kg dry air), relative humidity of air (%), temperature of air (°C) and dew point are presented in the psychometric chart (Fig. 31.2).

As buildings age, repairs and changes are made, sealants and vapour retarders begin to leak, storms, temperature and R.H. variations occur, it becomes obvious that the moisture, which inevitably leaks or condenses on surfaces and into constructions, should evaporate as easily as possible. This requires open, naturally vented constructions and spaces instead of closed and sealed ones.

Residential buildings are occupied by people of all ages, interests and levels of education, and are also often left unoccupied for days to months due to travel or moving of the occupants or resale of the building. Consequently the design of such buildings should not rely on mechanical ventilation alone for removal of moisture from spaces and constructions.

31.1.1 Essentials for microbial growth

The ACGIH-Guidelines for the Assessment of Bioaerosols in the Indoor Environment (Burge et al., 1989) lists 8 potential microbial sources in the occupied space. The last was people themselves, all the others were different sources of moisture, including humidifiers.

Excessive levels of biological aerosols in buildings, including homes, are caused by persistent moisture and inadequate ventilation in spaces and building elements; proper design and construction are essential to prevent these conditions (WHO, 1990). The necessity of controlling moisture in the buildings stems from the fact that moisture is the only essential that can be denied from micro-organisms in buildings. The first bacteria or fungal spores to infect a surface or material and begin the growth will always be available, because their concentrations even in "clean" outdoor and indoor air range from tens to thousands of living organisms per m^3 of air, organic material that provides nutrient base for the micro-organisms, cannot be eliminated from buildings, and the temperature range of building structures and indoor air is suitable to the growth of almost all widely occurring micro-organisms (AIVC 1989; EPA, 1991).

"Where water occurs, there is a risk of growth of fungi and other micro-organisms". "The presence and growth of moulds[1] and other micro-organisms in the indoor environments should be avoided by controlling moisture" (Key conclusion adopted by consensus, Quebec, NATO/CCMS 1993, Bienfait et al., 1991).

High air humidity results in high moisture content of porous building materials, and moist surfaces, but also leaks, condensation, water seepage by capillary action, and water use in the kitchens and bathrooms can wet building materials and keep them moist regardless of relative humidity of the air, and it is the moisture of the growth medium, not the relative humidity of air, which supports microbial growth (Jarvis, 1990; Pasanen et al. 1991; 1992a).

In addition to controlling microbial growth by controlling moisture, biocides have also been used, especially in hospitals and other sensitive areas, including HVAC-components. However, the use of biocides in the cleaning and

1 The term mould is used in this report interchangeably with fungi, depending on the usage in the cited material.

maintenance of the HVAC systems or surfaces in the buildings presents risks, both directly and through the promotion of resistant micro-organisms (WHO, 1990). In general, exposure to biocides is at least as dangerous as exposure to most microbial aerosols and should be avoided (Burge et al., 1989). Biocides should therefore only be used when the control of micro-organisms is absolutely essential, and it cannot be achieved by controlling moisture or changing the process. If the use of biocides is unavoidable, they should be prevented from entering spaces that can be occupied (Burge et al., 1989; WHO 1990). Since some surface bound long lasting antimicrobials do not diffuse or partition into the surrounding environment, an effective level of the material remains on the treated surface, and promotion of resistant micro-organisms is less likely to occur (Kemper, 1991).

31.1.2 Effects of moisture for the building and occupants

"Leaks from installations can give rise to extensive indirect problems such as mould growth or chemical degradation of building materials, with deterioration in the quality of air as a consequence" (NKB, 1991). This statement is also true for any other source of persistent, unintended moisture in the building. The indoor air quality will be deteriorated by micro-organisms, mostly fungi, and malodorous gases, stemming from chemical and biological degradation of materials, and also by irritating VOCs produced by micro-organisms — possibly for defensive purposes. One can safely state that any building structures, which are not particularly designed to be in continuous contact with water, will rapidly deteriorate if they are not kept dry, or if they are not quickly dried whenever they might get wet.

Moisture damage in the building is not necessarily reflected in the total levels of micro-organisms in the indoor air, but it does affect composition of fungal flora (Pasanen et al., 1992b). Specifically *Stachybotrys atra* is uncommon in "healthy" buildings, but often found in samples collected from buildings with known water damages, and homes with endogenous mould problems (Jarvis, 1990).

The health effects of fungal spores and other micro-organisms are discussed in detail elsewhere in this book. It is sufficient to say here that they are known to affect the health of people by spores, toxins and VOCs, and they may cause infectious diseases, allergies, cancer, irritation in the respiratory system, eyes and skin, and acute toxicosis (Burge et al., 1989). Exposure to indoor moulds may be more dangerous than one might think. Studies in Holland, the USA, and Canada have correlated the respiratory illness with the presence of mould and moisture problems in dwellings. The Canadian study found that in the homes that reported mould and moisture problems, 19% of the occupants

suffered respiratory illness symptoms versus 11–13% in the dry homes (NATO/CCMS, 1992). The expert meeting organized by WHO in 1988 recommended that:

- To reduce allergic diseases in the community, total exposures to allergens should be minimized by controlling allergens and their sources in buildings.
- Biological irritants and infectious agents cause nonspecific aggravation of respiratory and skin diseases, and they should be minimized by controlling their levels and sources in buildings.

31.1.3 Control of relative humidity of indoor air

The humidity in the indoor air may directly or indirectly have an impact on the occupants. High air humidity, condensation or ingress of moisture stimulates the growth of moulds and other fungi etc., which may cause allergy and malodours. Increased humidity may also enhance the emission of chemicals like formaldehyde from materials. Normally few problems occur, when the relative humidity of air is between 30 and 70%, assuming that no condensation takes place (Bienfait et al., 1991). The WHO expert group that met in Rautavaara in 1988 stated "it is best that moisture levels be maintained in the 30–50% relative humidity range. At levels higher than 65% the incidence of upper respiratory illness might increase and adverse effects might occur in people suffering from asthma and allergies. Lower moisture levels (RH below 20%) may induce dryness or itching of the skin, and aggravate certain skin conditions." (WHO, 1990).

Humidity of indoor air is a problem in most areas of the world for at least one season of the year. In the warm and humid climates high humidity favours the growth of bacteria, fungi and house dust mites, which deteriorate building materials, and bring health risks for the people. In the cold northern winter climates the outdoor air RH may be reduced to below 10% when this air is warmed up to room temperatures. Very dry air causes respiratory, eye and skin problems to a large portion of the people, especially asthmatics and the elderly (and ex-smokers). It also causes drying and cracking of wood materials in the building structure and furniture, and irritation to mucous membranes. Striking a balance between these two extremes can be difficult but it is not impossible. Most moisture problems can be solved by minimizing unnecessary sources, minimizing temperature differences, increasing air circulation and ventilation, and altering the moisture transfer rate with the use of vapour retarders.

Because of the variety of mould types and the consequent wide range of conditions for growth there are difficulties in defining a precise limiting value.

However, experience in the U.K. indicates that the incidence of mould growth is very small if the relative humidity in the room is kept below a value of 70% (AIVC, 1989).

Vapour pressure-dominated mould and mildew can be reduced by one or more of the following strategies:

- source control (e.g., direct venting of moisture generating activities such as showers) to the exterior
- dilution of moisture-laden indoor air with outdoor air that is at a lower absolute humidity
- dehumidification

Note that dilution is only useful as a control strategy during heating periods, when cold outdoor air tends to contain less moisture (EPA, 1991).

Source control

The reduction of water vapour production can generally only be attained by a change in the behaviour of the occupant.

Direct removal of water vapour at the locations of main production due to household activities can be achieved by extract ventilators in kitchen and bathrooms. Extract fans controlled by humidity would improve condensation risk better than those under the control of tenants.

In rooms other than kitchen or bathrooms the moisture content of internal air may be reduced by dehumidifiers. These are available in a range of sizes and water extraction capability. Most operate using a closed refrigerant heat pump cycle. Room air is passed over the cold evaporator, usually driven by a small fan, causing water vapour to condense. In general, dehumidification was found to be successful in reducing condensation problems in houses with both high internal humidity and temperature. In houses with lower temperatures dehumidifiers were found to have little effect. Although welcomed by the majority of users and perceived to alleviate condensation, noise was found to be a major drawback and to inhibit use in bedrooms.

Ventilation rate

Assuming average external conditions for relative humidity and air temperature during the heating period, taking estimated water vapour production rates, typical fabric transmittance and average energy consumption, the effect of average ventilation rates on relative humidity can be determined.

It is clear that adequate ventilation is a necessary but not sufficient condition to maintain internal relative humidity below 70%, the level of heating and insulation being equally important. For these reasons it is not possible to define a universally applicable ventilation rate that will ensure control over relative humidity. As a general guide, however, an air change rate

lying in the range of 0.5–1.0 ach (air changes per hour) should provide a necessary, but not sufficient condition to maintain internal relative humidity in United Kingdom dwellings below 70% and hence to contain mould growth.

Detailed calculations appropriate to German conditions have resulted in a minimum whole house air change rate for average user behaviour in the range 0.5–0.8 ach. They note that this conclusion applies at each end of the heating seasons and that the rates may be reduced to about 2/3 in the colder months.

Adequate ventilation is only one factor in controlling the incidence of condensation and mould growth. However, given adequate levels of heating and thermal insulation minimum ventilation rates can be calculated, either on the basis of maintaining relative humidity below a set level or ensuring that dew-point temperature of the indoor air remains below that of the internal surface of any sensitive area of the building envelope. Calculations using either of both methods give comparable values for whole house ventilation rate, in the range 0.5–1.0 ach, for German and British conditions. Lower rates may apply to countries with different climatic conditions and living habits (AIVC, 1989).

Dry winter climates

In Scandinavia, where low humidity of indoor air is a problem, guidance has been developed for the use of humidifiers and other means of avoiding the problems of dry air. Symptoms caused by dry air increase when temperature reaches and exceeds 22°C. Also the sensation of dryness increases with increasing temperature (Jaakkola, 1986). Clearly the method of choice for reducing the problems of dry air, which also complies with the requirements for energy conservation and VOC emissions reduction, is avoiding temperatures higher than necessary in the heating season (The Finnish Building Regulation Code 1987).

In the light of present knowledge humidification of room air is seldom necessary unless a certain moisture content is required for production engineering reasons or, e.g., in certain hospital rooms. A lot of problems associated with dryness can be remedied by humidification when humidity is less than 20%.

At the Nordic seminar, The Healthy Building, the following statement was made regarding humidification:

> From the hygienic point of view, general humidification of air is not recommended. General humidification may have side effects such as proliferation of house dust mites, legionnaires's disease, and allergies. Symptoms of "dry air" shall be mainly counteracted by methods other than humidification. Selective humidification may be required for spe-

cial individuals/environments/processes. In such cases it is essential that a "safe" technology should be chosen. (NKB, 1991)

The safety of the humidification technology, mentioned above, refers to humidifiers, where micro-organisms cannot grow. Because the use of biocides was not recommended by the WHO expert group (WHO, 1990), safe humidification technology means high temperature steam humidifiers. Preference should be given to humidification through the injection of steam that does not contain corrosion inhibitors and is delivered at temperatures above 65°C (WHO, 1990). However, the safety requirements of humidification must also include the avoidance of any condensation in the ventilation system, and the control of air humidity in the rooms at levels where the dew-point of air always remains below the temperature of the coldest surface in the building that can come into contact with indoor air.

Whether the problem is low or high humidity, human senses do not measure it. Dry air can be sensed as cold, for evaporation, or dusty, for respiratory, eye and skin irritation. Humid air may be sensed as hot, for wet skin, etc. As a consequence of false sensations humidification is often used in places, where it does not solve indoor climate problems but actually creates new indoor air quality problems. Therefore home-owners should install a humidity meter to monitor and maintain healthy humidity levels (NATO/CCMS, 1993).

31.1.4 Condensation of moisture

Control of dew-point in an indoor space is clearly the most important single way in which the growth of micro-organisms can be controlled, and many of the control strategies are aimed at that aspect (WHO, 1990). In order to limit the incidence of mould growth effectively, it is necessary to control condensation, and therefore to maintain the dew-point temperature of the internal air below the temperature of the coldest internal surface of the building envelope. As part of this strategy attention has to be paid to avoiding 'cold bridges' due to defects in building's insulation (AIVC, 1989). The ventilation machinery, equipment rooms, plenums and ducts shall be thermally insulated so as to prevent any damage caused to structures or ventilation system by condensation (The Finnish Building Regulation Code 1987).

Relative humidities measured in Scandinavian climate inside the walls roofs and floors against the cold surfaces are most of the time between 70 and 95% (see Fig. 31.3), close to condensation and within the growth zone of many fungi (Samuelsson, 1985). In the winter, when the humidities are highest, fungal growth is mostly controlled by low surface temperatures, below 5°C, on these surfaces.

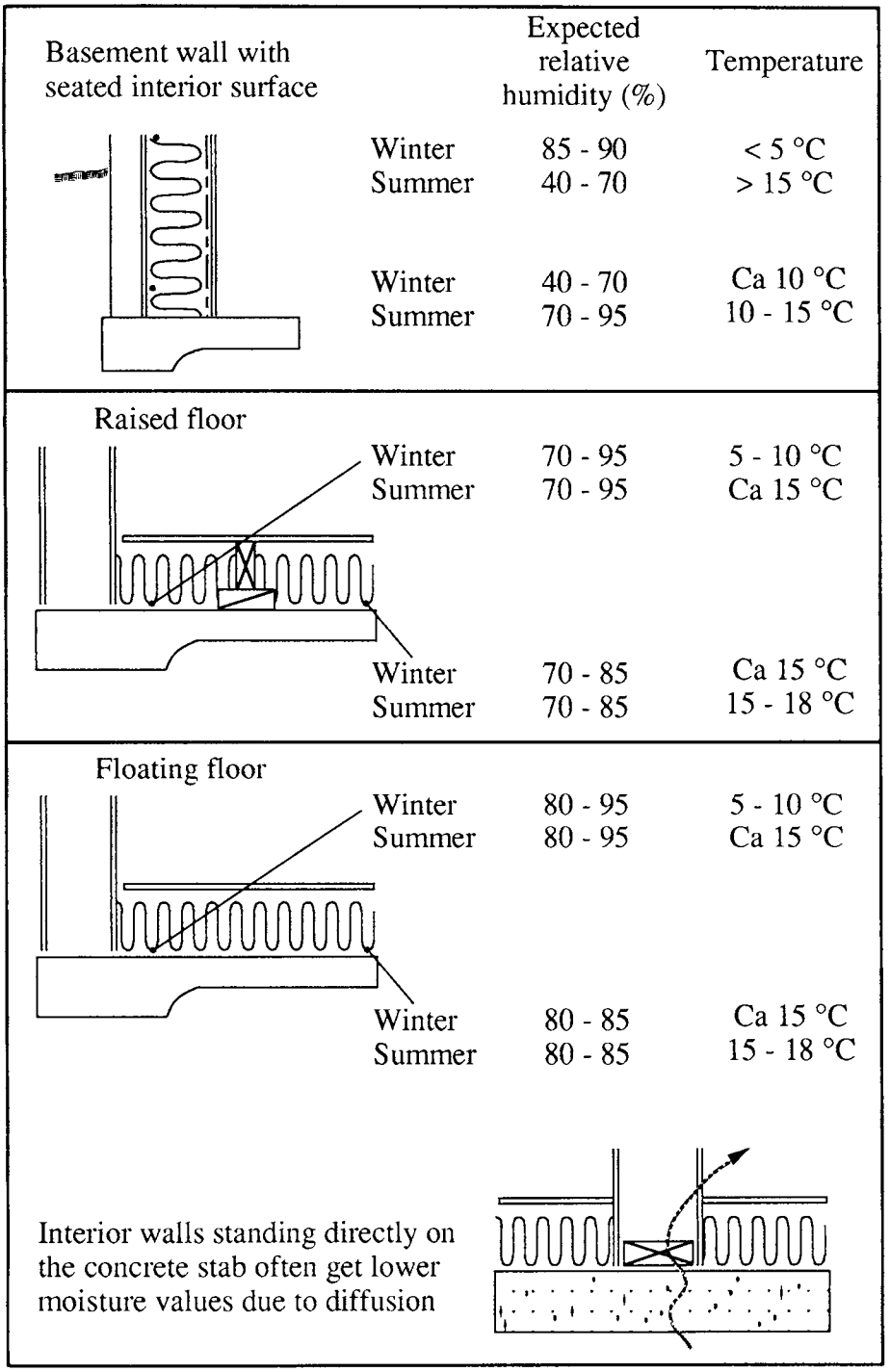


Fig. 31.3: Expected temperatures and relative humidities inside different wall, floor, and roof structures in Scandinavian climate conditions.

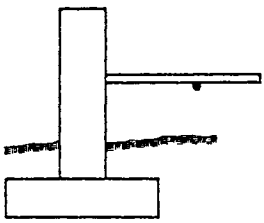
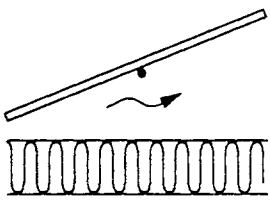
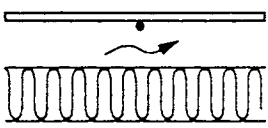
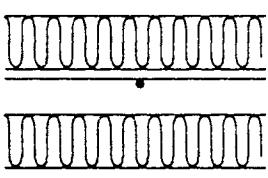
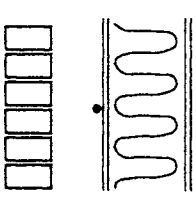
		Expected relative humidity (%)	Temperature
Crawl space 	Winter	70 - 85	< 5 °C
	Summer	80 - 95	> 10 °C
Stanted roof ventilated attic 	Winter	85 - 95	< 5 °C
	Summer	40 - 70	> 15 °C
Parallel roof 	Winter	85 - 95	< 5 °C
	Summer	40 - 70	> 15 °C
Double isolated parallel roof 	Winter	50 - 70	0 - 10 °C
	Summer	50 - 70	15 - 20 °C
External wall of bricks 	Winter	85 - 95	< 5 °C
	Summer	40 - 95	> 15 °C
	The high values relate to moist bricks		

Fig. 31.3: *continued.*

Either surface temperature or vapour pressure can be the dominant factor in causing a mould problem. A surface temperature-related mould problem may not respond very well to increased ventilation. Reduction of surface temperature-dominated mould and mildew is best accomplished by increasing the surface temperature through either or both of the following approaches:

- Increase the temperature of the air near room surfaces either by raising the thermostat setting or by improving air circulation.
- Decrease the heat loss from room surfaces either by adding insulation or by closing cracks in the exterior wall to prevent wind-washing (EPA, 1991).

Set back thermostats

Set back thermostats are commonly used to reduce energy consumption during the heating season. Mould and mildew growth can occur when building temperatures are lowered during unoccupied periods. Mould and mildew can often be controlled by increasing interior temperatures during heating periods. Unfortunately, this also increases energy consumption and reduces relative humidity in the breathing zone, which can create discomfort.

Air conditioned spaces

The problems of mould and mildew can be as extensive in climates which require cooling, as in climates which require heating. The same principles apply: either surfaces are too cold, moisture levels are too high, or both.

A common example of mould growth in climates, which require cooling, can be found in rooms where conditioned "cold" air blows against the interior surface of an exterior wall. This condition, which may be due to poor duct design, diffuser location, or diffuser performance, creates a cold spot at the interior finish surface. A mould problem can occur within the wall cavity as outdoor air comes in contact with the cavity side of the cooled interior surface. It is a particular problem in rooms decorated with low maintenance interior finishes (e.g., impermeable wall coverings such as vinyl wallpaper) which can trap moisture between the interior finish and the gypsum board. Mould growth can be rampant when these interior finishes are coupled with cold spots and exterior moisture.

Possible solutions for this problem include:

- preventing hot, humid exterior air from contacting the cold interior finish (i.e., controlling the vapour pressure at the surface);
- eliminating the cold spots (i.e. by elevating the temperature of the surface) by relocating ducts and diffusers;
- ensuring that vapour barriers, facing sealants, and insulation are properly specified, installed, and maintained;
- increasing the room temperature to avoid overcooling.

In this case, increasing temperature decreases energy consumption, though it could cause comfort problems.

Thermal bridges

Localized cooling of surfaces commonly occurs as a result of "thermal bridges", elements of the building structure that are highly conductive of heat (e.g., steel studs in exterior frame walls, uninsulated window lintels, and the edges of concrete floor slabs). Dust particles sometimes mark the locations of thermal bridges, because dust tends to adhere to cold spots.

The use of insulating sheathings significantly reduces the impact of thermal bridges in building envelopes.

Windows

In winter, windows are typically the coldest surfaces in a room. The interior surface of a window is often the first condensing surface in a room.

Condensation on window surfaces has historically been controlled by using storm windows or "insulated glass" (e.g., double-glazed windows or selective surface gas filled windows) to raise interior surface temperatures. The advent of higher performance glazing systems has led to a greater incidence of moisture problems in heating climate building enclosures, because the buildings can now be operated at higher interior vapour pressures (moisture levels) without visible surface condensation on windows. In older building enclosures with less advanced glazing systems, visible condensation on the windows often alerted occupants to the need for ventilation to flush out interior moisture.

Concealed condensation

The use of thermal insulation in wall cavities increases interior surface temperatures in heating climate, reducing the likelihood of interior surface mould, mildew and condensation. However, the use of thermal insulation also reduces the heat loss from the conditioned space into the wall cavities, decreasing the temperature in the wall cavities and therefore increasing the likelihood of concealed condensation. The first condensing surface in a wall cavity in a heating climate is typically the inner surface of the exterior sheathing, the "back side" of plywood or fibreboard. As the insulation value is increased in the wall cavities, so does the potential for hidden condensation.

Concealed condensation can be controlled by either or both of the following strategies:

- reducing the entry of moisture into the wall cavities (e.g., by controlling infiltration and/or exfiltration of moisture-laden air); and/or
- elevating the temperature of the first condensing surface. In heating climate locations, this change can be made by installing exterior insula-

tion (assuming that no significant wind-washing is occurring). In cooling climate locations, this change can be made by installing insulating sheathing to the interior of the wall framing and between the wall framing and the interior gypsum board (EPA, 1991).

During the winter, warm interior air may pass to the outside through cracks and openings in the building. If the warm, moist air comes into contact with cooler surfaces, the water vapour will condense onto the cooler surfaces. During the summer when the outside air is warmer and moister than inside air, the air flows into the house carrying moisture with it. Wind can also cause pressure differentials, and the downwind side of a house may show more signs of moisture because water vapour is being forced through this side.

Too much water vapour can cause window sweating when warm, moist air contacts cold surfaces. Warm air holds more water vapour than cold air, and the water in the warm air will condense on the cold surface as the air comes in contact with the window. Even if a storm sash is used, problems can result. If condensation appears on the inside window, the storm sash may be leaking cold air which cools the inner pane causing condensation; or there is simply too much moisture in the house. If the outside sash has condensation, the inside window is leaking warm, moist air which condenses on the cold exterior pane.

A problem which is not immediately obvious is condensation which occurs inside walls. Condensation can occur in exterior walls without vapour retarders or in those that have vapour barriers on the cold side of the wall or on both the cold and warm sides. If a home is uninsulated, water vapour is released from warm interior air as it passes through the building envelope, and if the temperature is cold enough, this moisture freezes. Over the course of the winter, the ice builds up and melts as the weather warms. Over time, serious structural damage can result. If a house has insulation (loose, batt, or foam), but no vapour retarder, the same process occurs. The insulation does not prevent the moisture from passing through the envelope, and water vapour will condense inside the wall and also pass through the wood and lift paint off the surfaces: lifting paint is, therefore, one sign of moisture problems.

Since condensation depends on temperature differences, minimizing these can solve some problems. Strategies include insulating metal window frames, insulating heating and cooling system ducting, improving heating patterns, and installing a vapour retarder.

Installation of vapour retarders

The effectiveness of the vapour retarder is measured in permeance (perms) of the material. One perm equals 1 g of water per ft^2 per hour per unit vapour pressure difference. The lower the perm rating of a material, the better it is at reducing the transfer of moisture. For example, 0.15 mm polyethylene (pro-

vides good control) has a perm rating of 0.06 perm compared to concrete, which has a rating of 3.2 perm.

The purpose of a vapour retarder is to keep warm, moist air from coming in contact with surfaces that are below the dew-point of the water vapour (the temperature at which condensation occurs). For this reason, a vapour retarder must be located on the warm side of the wall, floor, or ceiling, and it must be tight to prevent the transfer of water vapour.

Conventional retrofits

In the attic or ceiling, vapour retarders may be the only way to prevent moisture problems if sufficient ventilation cannot be provided. It is important to install the vapour retarder carefully. Batt insulation can be tightly stapled to the rafters; or in attics with loose fill insulation, polyethylene can be cut into strips and tightly fitted between the joists (Fig. 31.4). Regardless of the type of installation, there must be sufficient attic ventilation (eaves, ridge, or vent in Fig. 31.5).

In both warm and cold climates, crawl spaces carefully fitted with ground cover and floor vapour retarders can prevent moisture problems. Ground vapour retarders are installed over the soil and can be held in place with weights or bricks; a more secure method is to cover the retarder with a layer of sand. Floor vapour retarders can also be added to provide greater protection and insulation. The installation differs depending on the climate. In cold climates, the vapour retarder is installed on the warm side of the floor; in warm climates, the warm side is the bottom of the floor (Fig. 31.6).

The most cost-effective strategy for minimizing moisture problems in walls in retrofit installations is to seal air leakage points from the inside and outside of the wall. After ensuring that the leakage points have been sealed, a vapour retarding paint can be applied to the wall surfaces to provide some resistance to water vapour diffusion. In some instances it may be necessary to install a vapour retarder on interior walls.

Weeping windows should be weather-stripped and caulked inside first, and then weatherproofed outside. If an interior-side storm window is used, it must be sealed tightly around all edges. If an outer storm window is added, weep holes at the bottom are recommended. In very cold climates, triple glazing is recommended.

Vapour retarders in new construction

When moisture problems are encountered in new construction, problems are sometimes due to an improperly installed retarder. Figure 31.7 shows elements of a properly installed retarder. Any holes or other leakage points will allow moisture to be transferred. Seams must be carefully overlapped and

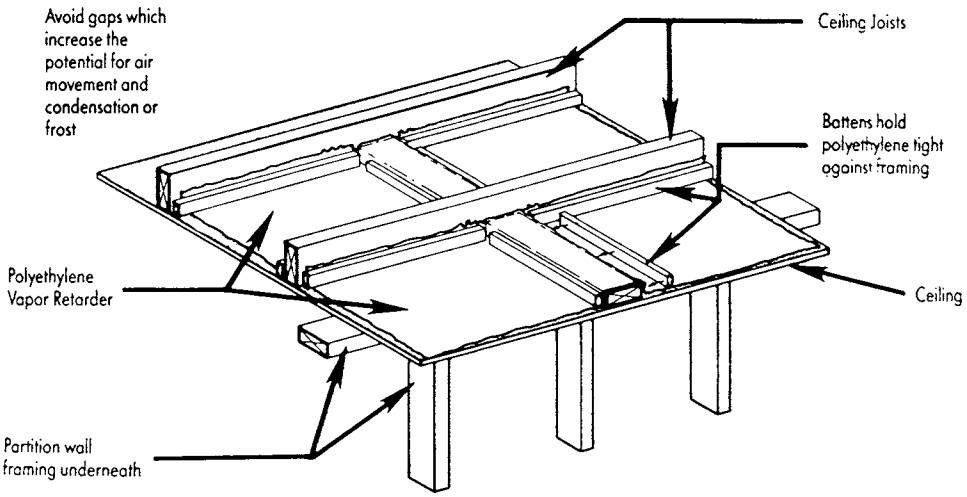


Fig. 31.4: Installation of a vapour retarder in the attic.

sealed, gaps around windows, doors, floor/ceiling/wall joints, and electrical and plumbing installations must be sealed. This can be done with polyurethane foam and other sealants and products.

If the basement is to be used for habitation, the enclosure should be finished so as to minimize the possibility of condensation problems on cold floor slabs, which could lead to mould growth (WHO, 1990). The obvious means of minimizing the possibility of condensation on basement walls and the concrete slab, is thermal insulation, and prevention of moisture entry to the cold surface by ventilation and use of sealed, vapour-proof surface materials. In some climates dehumidification devices may be required in basements for operation

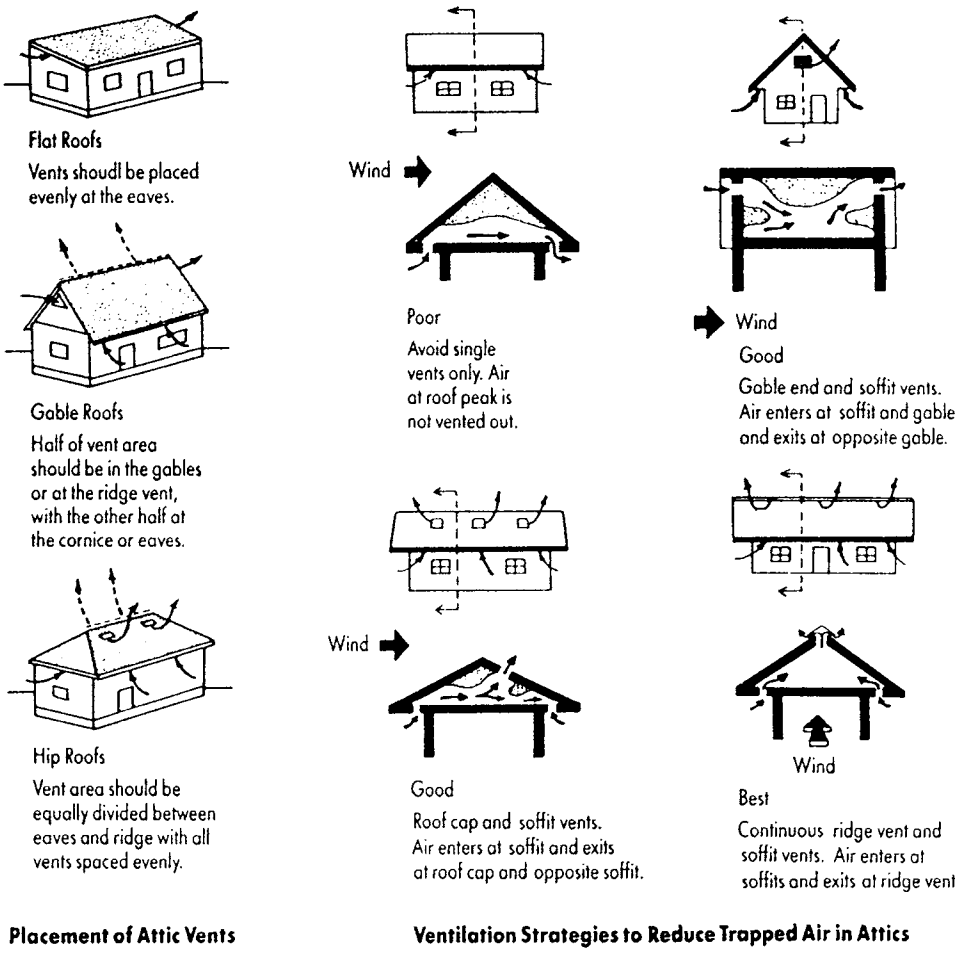


Fig. 31.5: Attic ventilation strategies.

in the summer and autumn, when condensation is likely to occur on basement floor slabs (WHO, 1990). In the basements temperature gradients between table height and basement floor slab are typically 2–6°C, enhancing condensation. This problem can be remedied by insulation and ventilated subfloor constructions (NATO/ CCMS, 1992).

31.1.5 Water leaks from the water systems, air conditioners humidifiers and dehumidifiers

This section deals with the avoidance of water leaks from systems which are known to contain water and which are located in known places in the building. The keys to control of water damages from such systems are:

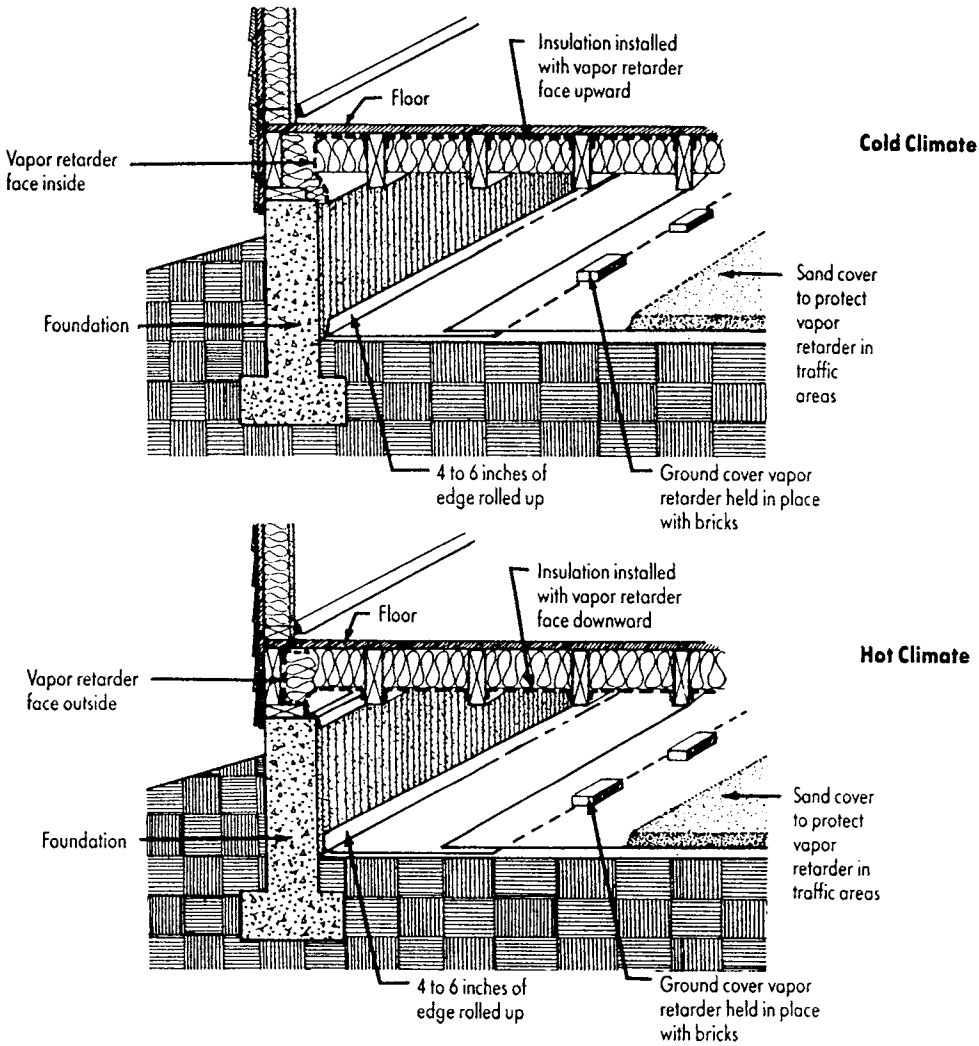


Fig. 31.6: Installation of a vapour retarder in the crawl space or floor.

- watertight, sloped and drained floors in wet spaces,
- drip pans and drains for any building components that may leak or condense water,
- visibility and easy inspection of all piping and draining,
- protection of all pipes and drains from breaks and cracks resulting from temperature changes and movements of the building components, and
- protection from freezing by locating all pipes and drains in warm spaces of the building.

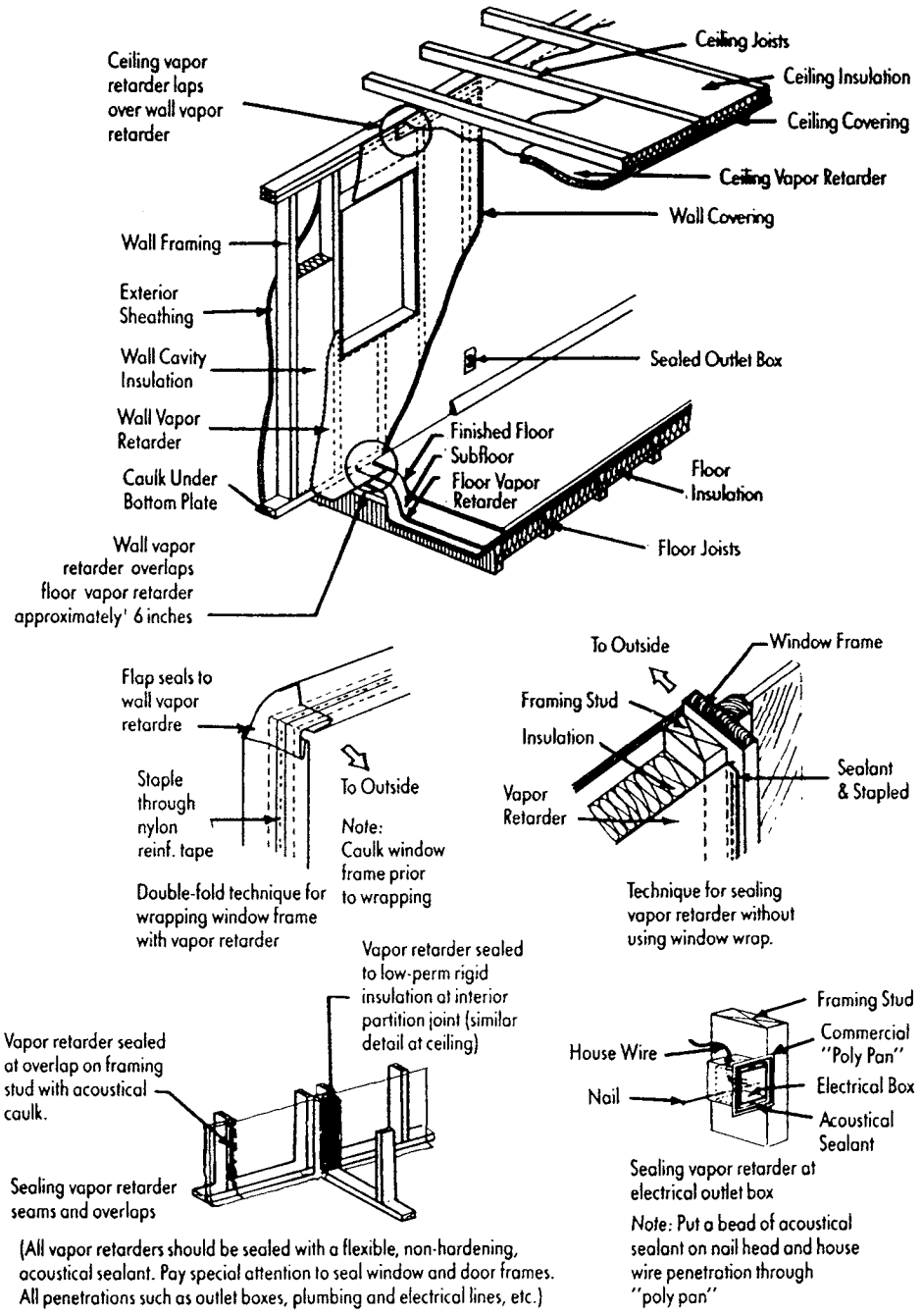


Fig. 31.7: Installation of a continuous vapour retarder.

Recirculated water sprays and stagnant waters in humidifiers and the drip pans of chillers and dehumidifiers can be significant sources of biological aerosols. While drip pans can be designed and installed so that they are drained by gravity, non-steam humidifiers must be continuously maintained or they will become sources of biological aerosols (WHO, 1990). A humidification system that relies on the spraying or otherwise dispersing of water kept and discharged at temperatures below 65°C can easily become a major multiplier and disseminator of microbial contamination in a building (WHO, 1990). Micro-organisms are always abundant in portable cool mist and ultrasonic humidifiers unless they are cleaned and disinfected (with chlorine bleach or hydrogen peroxide) daily (Burge et al., 1989). The public has to be made aware that the use of cool mist or ultrasonic room humidifiers could be a major source of microbial agents, a source that could be reduced or eliminated if steam humidifiers were substituted (WHO, 1990).

Dehumidification and cooling of indoor air in cooling coils leads to condensation of water in such devices. This condensation must be able to drain and the overall system must be kept clean and free from microbial growth. This requires easy access to all the components on which water can condense and to the systems used for collection and drainage (WHO, 1990). If an air supply unit, located inside the building is connected to a piping system conducting a liquid, the building structures shall be protected from the effects of any liquid leaks by means of e.g. a floor drain and a watertight floor (The Finnish Building Regulation Code, 1987).

All plumbing should be installed so that it is protected from freezing and all plumbing lines should be installed so that they can easily be inspected for leaks and repaired promptly when leaks occur (WHO, 1990). Buildings should be annually inspected for water leaks and water damage, whether from rainwater, from plumbing leaks or from water entering the basement through the soil. In case of water damage the damaged area should be thoroughly dried, and any damaged material should be removed and replaced (WHO, 1990).

31.1.6 Storm- and meltwater leaks through the roofs and walls

The best way of dealing with storm- and meltwater leaks through the roofs and walls is, naturally, to prevent them. The structure should be designed so that rainwater and melting snow cannot gain entry into the structure, even when the building and its materials age over the years (WHO, 1990). Also in ideal constructions any meaningful amounts of storm- or meltwater leaking into the building and its construction should be easily detected and the condition fixed.

On flat roofs even a small leak will give rise to extensive consequential damage. For instance problems are often encountered due to icing and crack-

ing in climates with low temperatures and snow. The cause of these problems may be bad planning, defective construction or poor maintenance. Roofs shall be so designed that water is drained. Account should be taken for local snow and ice conditions (NKB, 1991).

In practice these requirements can be best met with sloped roofs, simple roof shapes, and roof structures and surfaces, which, without compromising waterproofing, have enough flexibility to allow for the small dimensional changes which may result from snow loads, predictable movements of the building structure and aging of the building materials. Moisture resistant or waterproofed exterior wall and window materials and constructions reduce the possibilities of stormwater leaks, and wide roof overhangs may be used to give additional protection.

Ventilation components like the air intake and exhaust openings and the air handling unit are typically located on the roof or in the attic, and are subject to both rainwater and snow entry. Intake and exhaust openings and their connections to the ventilation system shall be arranged, protected and dimensioned to prevent any entering rainwater or snow from causing damage to the building structures or the ventilation system operation. Ventilation compartments or ducts subject to accumulation of rainwater or snow must be provided with drainage (The Finnish Building Regulation Code 1987).

31.1.7 Groundwater leaks through the concrete slab and basement walls

At most building sites the soil, adjacent to the concrete slab or basement walls and floor, will be wet some or all of the time. To prevent groundwater from seeping or leaking into the building materials, constructions and into the building, where it would cause material, structural and eventually health damage, foundation structures of the type slab laid directly on the ground require particular care in design and construction, for instance with regard to the placing of thermal insulation and waterproofing, and the quality of the anticapillary layer (NKB, 1991). Capillary action refers to the movement of liquid water from the source through a porous material such as soil. Capillary action can be an important route of water vapour entry through basement and crawl spaces. The moisture transfer rate can be reduced by using waterproofing on the exterior or interior of basement walls and installing exterior drainage systems. The basement structure should be surrounded by a ground surface that slopes away from it, so as to minimize the possibility of rain run-off entering through cracks (WHO, 1990). One way of removing moisture from the basement walls and floor, and also to reduce radon entry into the building, is a basement design where the walls are covered by polyethylene

sealed, insulated stud walls, the slab is covered by a subfloor, and the connected cavity space is depressurized and ventilated by a blower exhausting outdoors (NATO/CCMS, 1992).

31.1.8 Removing water and moisture from wet spaces; kitchens, bathrooms and laundries

In the spaces where water is routinely handled, and where considerable amounts of water are handled in cooking, bathing, washing and drying of clothes and dishes, and in the drying of intermittently wet surfaces, all this water must be either drained or evaporated and ventilated. Wherever these two are alternatives, draining should be the preferred choice. The bigger the drained portion of the handled water is, the smaller the portion that needs to be evaporated and ventilated by considerable energy costs, and the smaller the change that evaporated water will condense elsewhere in the building. Effective draining requires smooth, non-porous and sloped surfaces, and floor and sink drains that will not develop leaks. The requirements of smoothness and non-porous, "glassy" surfaces needs to be emphasized here, because water that remains on/in the surface due to its roughness or porosity will only disappear through evaporation, and typical room surfaces need to be wetted only a few times per day, and they need to remain moist only for tens of minutes each time to support the growth of invisibly small, yet productive fungal microcolonies (Pasanen et al., 1992c).

All spaces in which water is introduced and spilled should have waterproof floors,and should be well drained. This is of greatest importance in bathrooms, saunas and kitchens, but should also apply to basements (WHO, 1990). Floor drains should be trapped and the water in the traps should be continuously flushed so that the trap does not become polluted (WHO, 1990). Also in rooms where there is a great risk of water leaks there should be either floor drains or the floor covering should be arranged so that any leaks can be seen (NKB, 1991). This refers clearly to rooms where the air handling units are located. Water leaks into those rooms can come from rain or snow, condensation on the cooling coils or exhaust air heat recovery units, or humidifiers. The Finnish building code requires floor drains and watertight floors, but "This does not apply for air circulating units, nor for air supply units arranged in the immediate vicinity of a building entrance if the flow rate of outdoor air does not exceed 0.9 m^3/s. Any accumulation of water in the room must, however, be prevented, e.g., by means of sloping the floor" (The Finnish Building Regulation Code 1987).

Most of the discussion about the minimum ventilation rates has been mainly based upon uniform steady state conditions. In practice many of the

processes which generate water vapour within a dwelling, such as cooking, bathing or clothes washing, occur intermittently and in specific locations. This allows the possibility of locally and/or temporarily increased ventilation, possibly using mechanical exhaust systems to prevent the water vapour mixing with the remainder of the dwelling (AIVC, 1989).

Controlling the source of moisture problem is usually a cost-effective solution. Strategies include ducting clothes dryer exhaust outdoors, improving drainage, and using kitchen and bathroom exhaust fans (Bienfait et al., 1991). Air circulation and ventilation strategies include venting moisture out of enclosed paces; using ceiling fans to improve circulation; installing wall, roof, and crawl space vents; installing heat recovery ventilators; and installing whole house fans and roof ventilators (EPA-RM).

Kitchen and bathroom exhaust fans controlled by humidity tend to reduce condensation risk better than those under control of the occupants (AIVC, 1989). Ducts containing humid air should be as short as possible, should be in warm areas of a building, and should not run horizontally so that any condensation will collect and drain (WHO, 1990).

31.1.9 Dealing with moisture from the construction period and materials

Many building materials, which are not designed to be in contact with water, like wood, mineral fibre insulations, gypsum or woodchip boards, are often left uncovered at the building site, and wetted by rain. They may or may not get wet before they are used. Also whole constructions, already in their places, like insulated floors, walls, and ceilings are sometimes thoroughly wetted before they are protected by waterproof roofs and outer walls. Construction moisture may accumulate into the constructions of new as well as renovated buildings. The amount of accumulated moisture depends on the weather during specific construction phases, on the type of building and its constructions, and on the concern and professional skill of the builders. Normally most of the moisture is removed by heating and ventilation before the internal surfaces of the building are prepared. Once wet concrete, for example, is locked behind watertight floor or wall surfaces, in kitchens, bathrooms or elsewhere, drying will be extremely slow, and this moisture may lead to both construction damages and indoor air quality problems before it has dried away. Depending on the kind of floor surface selected, the highest accepted relative moisture levels[1] for concrete range from 60 to 90% (Saarela, 1992).

1 Relative moisture level of the material is defined so that it is 0% for a completely water free material, and 100% for material which is thoroughly saturated with water.

The Nordic Committee on Building Regulations (NKB) requires that "During the construction period there should be in existence a quality assurance programme which ensures that unnecessary moisture is not incorporated in the structure. Many problems can be avoided by careful handling of building and building services materials (e.g. by ensuring that materials sensitive to moisture are always covered in the event of rain) and by continuous tests and checks of the moisture content of materials prior to their incorporation into the building" (NKB 1991).

The building weathers, building materials and constructions, and building practices vary across the world, and accumulated common sense and good professionalism should be applied to prevent construction moisture problems. Dealing with construction moisture may pose little problems or cost in many countries, and be a considerable time and cost factor elsewhere. This should be taken as a fact of life comparable to, e.g., different insulating, heating and air conditioning costs. The few common rules could be as follows:

- Prevent materials that are not meant to resist moisture from getting wet.
- Plan the building process so that water cannot enter into constructions that should stay dry for the life of the building.
- Allow for sufficient drying of concrete, plaster, levelling material, etc. and measure their moisture content before covering with water- and vapour-tight surfaces.
- Design all constructions so that moisture, whether from construction, condensation or minor leaks, will rapidly evaporate and be ventilated away by natural convection.

31.1.10 Chemical damage in materials under high humidity (Saarela, 1992)

Moisture has many different effects on materials. Moisture may dissolve chemical components from the building materials and move them towards dry zones. Moisture may enable, enhance or change chemical reactions in building materials and their chemical constituents. Moisture also enables growth of fungi and other micro-organisms in the building materials, and formation of chemical compounds that they produce (this effect has already been discussed earlier in this chapter, and will not be covered here).

The harmful effects of moisture are usually preceded by high moisture levels for extended periods of time and temperatures ranging from about 5 to 60°C, which covers practically all temperatures encountered in the buildings. Table 31.1 contains examples of the damages of high and persistent moisture

TABLE 31.1

Examples of materials that are susceptible to the effects of moisture or alkaline moisture, and estimates of the lowest damaging material moisture levels and the emissions produced.

Material	RH (%)	Damage: emission
casein-based levelling materials	75–85	hydrolysis:ammonia, amines, organic sulphur compounds
PVC carpets and PVC wallpapers for wet spaces	> 95	discolouring disintegration reactions: 2-ethyl-1-hexanol
water-based glues	> 85–95	saponification: hydrolysis products
ureaformaldehyde-based resins	> 60–70	disintegration: formaldehyde
paints, injection resins, plastic filters	varying	prevention of drying or hardening: monomer emissions

levels on building materials. The given moisture levels are the relative moisture levels of the materials.

The effect of moisture on materials is most often based on chemical (and physical) disintegration by hydrolysis reactions, and consequent release of the reaction products. The damages of moisture become most pronounced in those plastics, where the polymerization reaction is reversible, i.e. the direction of the reaction may also go back from polymer to monomer. One of the most instable of such materials is urea-formaldehyde (UF) resin, best known as a common binding material in chipboards, but also in acid hardened lacquers. For some time UF resins were also used to improve thermal insulation of old buildings, by filling wall cavities with sprayable UF-foam, where the acid, used to harden the foam, often caused excessive reactions and led, in addition to high formaldehyde emissions also to complete disintegration and consequent evaporation of the foam.

Moisture affects similarly the UF resin used to glue woodchips into chipboard, and the UF based acid hardened twin component lacquers. Due to the more solid structure of the chipboards, the hydrolysis reaction in them is slower than in UF-foam. The UF resin used in lacquers is often copolymerized with other monomers such as melaminformaldehyde, and copolymer resins are more resistant against hydrolytic disintegration than homopolymer resins.

The man made mineral fibre insulation materials may be exposed to moisture already before use in the building site, or later from leaking or condensing water. The glass and mineral fibres are chemically quite inert materials, but the resins used to bind them together may disintegrate when their moisture level is high and temperature sufficient. Consequently they may emit formaldehyde and higher aliphatic and aromatic aldehydes such as

benzaldehyde, and ketones. Laboratory experiments have shown that at the temperature of 50°C moist mineral fibre insulating materials emit, as an average, about one hundred times more aliphatic and aromatic aldehydes and aliphatic ketones than dry materials, and in that study they also produced strong odours. Again, phenolformaldehyde-melaminformaldehyde based co-polymer resins are more resistant to hydrolysis reactions than UF based resins.

The water vapour emitted from concrete is alkaline and causes alkalihydrolytic reactions in many polymer materials.

Many materials, e.g. binding materials in linoleum carpets, have been produced by polycondensation reactions from natural oil polymers. Under the influence of alkaline moisture these materials may begin to disintegrate releasing fatty acids, higher alcohols and ketones. Also many phthalate softeners used in PVC carpets and some other polymer materials may disintegrate in moist, alkaline conditions. One of the disintegration products is 2-ethyl-1-hexanol, which causes the strong sweet smell typical for damaged PVC.

Casein and other nitrogen containing organic compounds have been used in building materials for decades. These compounds are found in glues, base paints, waterproofing films/materials, levelling materials, and natural organic fibres, such as jute. In moist alkaline conditions such compounds disintegrate and release ammonia and possibly other organic compounds like amines, reduced sulphur compounds and alcohols.

Most of the above mentioned reaction products are highly odorous and irritating. Many of them also discolour both natural and man made building materials, like oak panels and PVC carpets.

A summary of the disintegration reactions of proteins is shown in Figure 31.8. Figure 31.9 illustrates the ammonium emitted from seven different levelling materials and concrete exposed to increasing levels of moisture.

In addition to the hydrolytic disintegration reactions that moisture causes in many polymers, moisture also disturbs the hardening reactions of many resins. Consequently the relations of the compounds taking part in the hardening reactions change, and this in turn results in incomplete hardening and emission of unreacted monomers from the resin. The hardening reactions of polyester resins are particularly sensitive to such disturbance, and even slightly incomplete hardening results in considerable styrene emissions.

In closing this section it is necessary to mention that all the above reactions, both chemical and physical are faster and result at higher emissions at higher temperatures. However, new reaction types, such as pyrolysis require temperatures in excess of 100°C, which can be found at most only in highly localized spots in normal buildings.

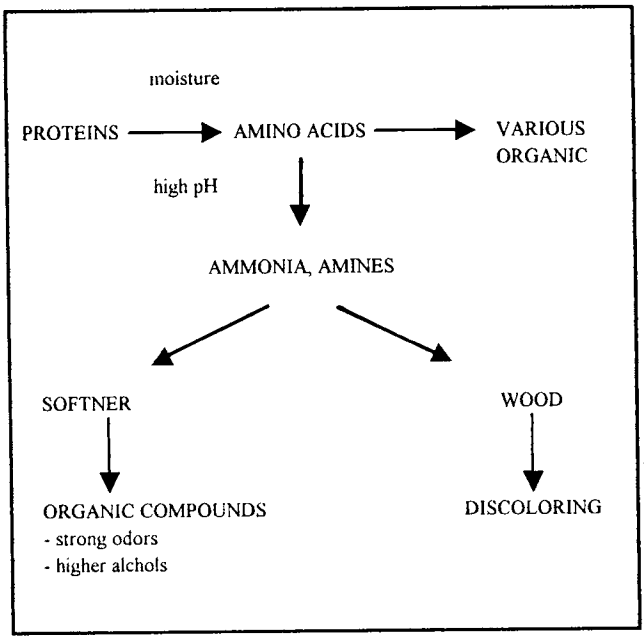


Fig. 31.8: Summary of the disintegration reactions of proteins and their effects on other materials.

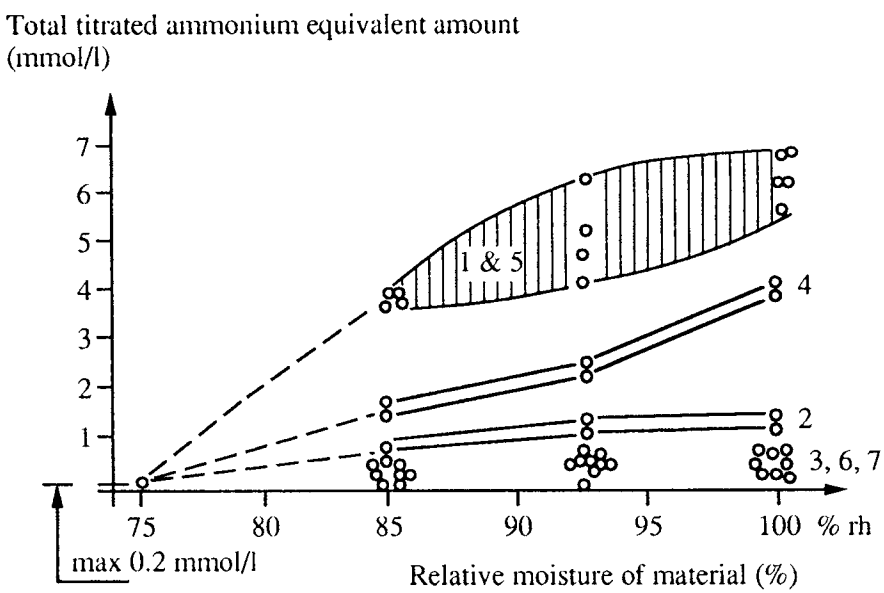


Fig. 31.9: Ammonium emitted from levelling materials and concrete.

31.2 SOIL GAS CONTROL

The principal soil gas component of health significance is radon but in special situations, e.g. areas of waste disposal, a broad range of potentially toxic chemicals may be present and a case-by-case evaluation of a possible health risk may be necessary. Landfill gas is produced by the action of micro-organisms on organic waste material deposited in landfill sites. The gas principally consists of methane and carbon dioxide but other gases such as hydrogen sulphide may be present. Over a hundred trace volatile organic compounds have also been detected in landfill gas including: aliphatic and aromatic hydrocarbons, alcohols, esters, ethers and organosulphur compounds. However, these compounds make up less than 1% of the total gas mixture and in general represent more of an odour nuisance rather that a toxic hazard. The major hazard that landfill gas represents is that of explosions: methane is flammable when mixed with air in the concentration range 5–15%. There have been numerous incidents where an explosive mixture of landfill gas has been ignited in a building damaging the structure and injuring the occupants. There is also a risk of asphyxiation from landfill gas. Buildings constructed on or near sites where there are high levels of these hazardous gases need to be sealed to prevent gas ingress and a means to disperse the gases provided, for example a ventilated subfloor void.

Geology and soil structure are important determinants of the potential contamination indoors by contaminants in soil gas. The physical characteristics of the soil of most relevance to the movement of soil gas are porosity, gas permeability and moisture content. Aspects of the geology which influence soil gas movement include the presence of faults, voids, groundwater behaviour, geothermal gradients etc. The relationships between these soil and geological characteristics and the mechanisms of soil gas movement are quite complex. In broad terms soil gas movement may be divided into the categories of diffusion and transport. The transport or migration of soil gas contaminants indoors can occur when the interior of buildings become depressurised due to the action of the wind, by the operation of exhaust fans or by an improperly balanced mechanical ventilation system (Nazaroff et al., 1987). Whether or not soil gas being transported as a result of depressurisation becomes a significant contribution to indoor air pollution depends on the availability of suitable entry routes. These are usually cracks and other openings in the part of the building envelope in contact with the soil that can act as entry routes.

The case of soil gas radon is now used here as an example of a soil gas constituent which can and does enter indoor air spaces.

31.3 REMEDIAL AND PREVENTIVE MEASURES TO REDUCE RADON

31.3.1 Introduction

The main sources of indoor radon are: soil, building materials and, in some cases, water from deep wells (see also Section 3.4). Experimental work carried out at international level has shown that radon from soil represents generally the most important source of indoor radon (e.g. Tanner, 1978; OECD, 1979; Asikainen and Kahlos, 1980; Bruno, 1983; Damkjaer and Korsbech, 1985; Kristiansson and Malmqvist, 1984; Nazaroff et al., 1987b; Nero 1989).

Radon occurs in high concentrations in soil gas with large variations due to the characteristics of local geology. Typical radon concentrations in soil gas range from 10000 to 50000 Bq/m^3. In areas of elevated indoor radon levels soil gas concentrations in excess of 500000 Bq/m^3 may occur. In such situations dilution of the incoming soil gas by indoor air will generally be insufficient to maintain indoor radon concentrations below most reference or action levels. In Sweden a categorisation of soils has been used as part of a system of classification of risk to occupants of houses from radon. In this system a low risk radon ground is a soil with a radon soil gas concentration less than 10000 Bq/m^3, while high radon ground would have a concentration greater than 50000 Bq/m^3 (Åkerblom et al., 1984; Snihs, 1992). Normal radon ground would lie between these two categories.

The actions to reduce indoor radon concentration are mainly oriented to limit the ingress of radon from soil. This goal can be reached by removal of the source, diverting the radon before entering the building and/or using barriers between the soil and the living space.

The techniques available at present can be grouped in two types; passive methods (which do not require further intervention after installation) and active methods (requiring extraction fans, air cleaning devices, etc.).

31.3.2 General approach to control radon indoors

The problem of remedial action has been faced since the 70s in Canada (DSMA 1979; Leung, 1979; McLaren, 1979; Scott, 1979), Nordic countries (Ericson, 1980; Ericson et al., 1984) and in the following years in the United States, mainly by the EPA, and later on in the UK (EPA, 1987c, 1988, 1989c, 1991, 1992b; Cliff, 1980; Green at al., 1992). In these countries experience has been gained, although restricted to single family dwellings and some schools (e.g. EPA, 1987a). As far as our present knowledge is concerned, a complete reference picture of all the different methodologies available to reduce indoor

radon and their effectiveness and durability is still not not available (CEC/DOE/EPA, 1993).

The methods available to reduce or prevent the ingress of indoor radon can be summarised as follows:

- reduction of radon entry from soil through *depressurisation* of subfloor spaces;
- increasing the building *ventilation* rate with a consequent increase of radon removal;
- increasing the resistance of the building to radon entry by *sealing* the floor (or the walls, in the case of building materials with high radon exhalation rate);
- removing the radon source: applicable only to water supply and not discussed in the following.
-

31.3.3 Depressurisation

Normally the pressure in a building is less than in the soil gas and this causes soil gas to enter the building. If, however, the pressure differential between the soil and the building is reduced the radon entry is decreased. In order to obtain soil depressurisation, a zone of subjacent soil is maintained at a lower pressure difference than the building, by means of a small fan or by a vented stack.

Different methods are available to obtain such soil depressurisation (DOE, 1990b; NCRP, 1989; EPA 1991). If the building has a concrete floor, a fan can be used to suck air from beneath the floor and vent it to the atmosphere. Usually a radon sump is built which consists in a hole in the ground with a suitable fan connected to it sucking from the hole and generating a negative pressure in the hole.

The more permeable the material below the slab, the more efficient is the sump. The pipe which connects the sump to the outdoor air can go under the floor to an external wall or to an internal wall and then up to the roof. According to the Building Research Establishment of the U.K., an effective exhaust fan for a single family house can have a power rating around 60 W and a pipe 110 mm diameter. This should achieve good results and has been found to remove in some cases 90% of radon (Wolliscroft, 1992).

In some circumstances, mainly when a new building is going to be constructed, a plastic membrane could be placed over the soil within the foundation, which will act as a barrier to radon (EPA, 1991).

An effect similar to the radon entry prevention obtained by the subfloor space depressurisation can also be achieved through the overpressurisation of a dwelling by a fan mounted in the attic of a dwelling.

31.3.4 Ventilation

Increasing the ventilation rate to reduce the radon concentration is one of the easiest methods available. Moreover, a knowledge of the radon source is not required. There are, however, practical difficulties in determining how much the ventilation rate should be increased and how it can be supplied in order to be acceptable by the occupants.

Mechanical ventilation with fans which blow fresh air into the house may have other effects other than to increase air flow. The air pressure inside the house can be increased to exclude ingress of radon. The efficiency of the system is strictly connected to the tightness of the building (DOE, 1990; Holub et al., 1985; Nazaroff et al., 1981; NCRP, 1989; Wolliscroft, 1992).

Exhaust fans can be used to increase the ventilation rate in the subfloor space; in this way, significant reduction can be achieved in the living space (Renken et al., 1992).

To compensate for the heating cost during the winter season due to increased ventilation a balanced mechanical ventilation system with heat recovery can be used. Quantitative data on the effectiveness of such systems are rather poor (Renken et al., 1992).

Another possible use of ventilation is the "natural" ventilation of basements: in a basement (or cellar) with open windows radon concentrations become lower not only because of dilution, but also because it reduces basement depressurisation (Cavallo et al., 1992).

In conclusion, it should be cautioned that while ventilation techniques may be effective, in some cases changes in building ventilation patterns may exacerbate the radon situation by increasing the underpressure in the building.

31.3.5 Sealing

In the foundations of a dwelling, usually many openings exist through which soil gas can penetrate. These openings include junctions between walls and floor, gaps between floor, slab sections, openings left for service entries, etc.

The remedies which can be applied are to fill the gaps and openings by using sealing materials like epoxy resin. A rigid sealant is not convenient since the cracks are in constant motion due to climate changes, shrink-swell cycle, etc. As an alternative a plastic cover can be applied on the floor having care to seal accurately the junctions between wall-floor, etc. (EPA, 1991). This solution can be easily applied in the case of new dwellings built in high radon prone areas as suggested by the U.K. Building Research Establishment (Wellincroft, 1992). The achievable reduction factor is at maximum

around 50%; the durability of this remedy is not well established. A drawback of the system could also be linked to possible emission of VOC from the sealing materials.

31.3.6 Air cleaning devices

As far as the use of cleaning devices are concerned most of experience gained in removing radon and its decay products derives from the mining industry. Although the principles on which the devices to remove radon daughters are based — mechanical and electrostatic filters — are independent of their use whether in mines or in homes, nevertheless the volumes of air involved and environments are completely different, so that this technique has been considered not applicable in houses (EPA, 1987c). Some years ago, this had been considered as a developing field and laboratory tests were carried out. The data available to date do not suggest that these techniques are very effective, but it could be useful perhaps in cases where the building materials are the principal radon source (McLaughlin, 1989).

31.3.7 Conclusions

The preventive and remedial measures described above have been experienced in single family houses of some countries. It is very difficult to give general rules applicable to every situation. Each house seems to represent its own unique problem. It has often been said that it is almost impossible to have two houses which behave in the same way with respect to radon.

A large amount of work remains to be done in order to establish general criteria for old and for new dwellings, to collect data on effectiveness and durability of remedies, to explore the best predictors to identify radon prone areas, etc. The approaches to be followed are obviously strongly dependent on building styles, constructions regulatory regime and on the use of buildings.

It has to be stressed that until now emphasis has been given to housing rather than to commercial buildings since the greatest need arises in housing stock where people usually spend most of their time. Studies are being undertaken in recent years to model the ingress of radon in large commercial buildings, offices, schools, etc., to single out their peculiarities with respect to single-family houses and to ascertain the kinds of remedies or preventive measures which are likely to reduce indoor radon (e.g. EPA, 1991b, 1992b).

31.4 COMBUSTION PRODUCT CONTROL

31.4.1 Introduction

Whenever unvented furnaces take place indoors or venting systems attached to stoves or hot water heaters malfunction, a wide range of combustion products can be discharged directly into the indoor atmosphere. When the externally vented heating system is properly designed, maintained and operated, combustion products that could directly affect indoor air quality do not enter the indoor environment. However, excessive negative pressures in the interior spaces caused by the operation of various exhaust appliances or faulty venting systems can result in levels of indoor air contaminants in excess of the recommended values (WHO, 1987).

The levels of indoor contaminants produced by combustion devices depend on the emission rate and the removal rate. For vented appliances, the rate of removal depends on the effectiveness of the venting system. For vented appliances, the rate of removal depends on the effectiveness of the venting system. For unvented appliances it depends on the house air exchange rate.

The major pollutants associated with indoor combustion are carbon monoxide, nitrogen dioxide, organic compounds and particulates. These usually occur in low concentrations compared with carbon dioxide and water vapour which are the major combustion products. Adverse health effects from exposure to indoor combustion pollutants depend upon concentrations and exposure time and range from annoyance and irritation to more severe health risks. Limiting concentrations of combustion products are given by WHO-guidelines for indoor air.

31.4.2 Control measures

There are several possible control measures which will be discussed in the following text.

Source Removal: The obvious approach, but not necessarily the most economical, is substitution of combustion appliances by electrical appliances for heating and cooking.

Source Modification: Combustion appliances can be modified to improve their efficiencies and thereby reduce fuel consumption and/or reduce the emission rate of combustion products. The NO_X emission rate can be reduced by lowering the flame temperature but at the expense of increasing the CO emission rate.

Air Treatment: There are currently no commercially available air-cleaning devices for removing gaseous combustion products such as CO, CO_2 and NO_2

from indoor air for residential application; however, airborne particulates can be removed by filtration and electrostatic precipitation.

Ventilation Rates: The use of ventilation, whether by air infiltration or by natural or mechanical processes, as a means of air contaminant control depends much on whether the combustion appliances are unvented or vented. In the former case the ventilation air is used to dilute the combustion products, whereas in the latter it is provided for proper combustion and operation of the venting systems.

Unvented Combustion Appliances: Test results with an unvented gas cooking stove indicate about a 20% reduction in CO and NO_2 levels when the whole-house air change rate was increased from 0.10 to 0.90 ach. Comparison with range hood experiments, however, show 60–80% reductions in the levels of CO, CO_2 and NO_2 with exhaust rates of 42–113 l/s, indicating that exhaust at source is much more effective than increasing the whole-house ventilation rate.

Tests with a radiant type kerosene space heather or a convective-type in a small bedroom show that WHO guidelines for indoor air for combustion products can be exceeded with unvented heaters in spaces of small volume such as bedrooms, house trailers and cabins, even with heaters that are well adjusted. Opening a window or door can reduce the pollutant levels by increasing the ventilation rate, but can result in longer burn time and, hence, exposure time.

Vented Combustion Appliances: Central furnaces require air for combustion and dilution. The combustion air is brought into the appliance at the burner, where it is mixed with the fuel, and the combustion products pass through the heat exchanger before venting. The dilution air is brought in downstream of the furnace and is used primarily to control the amount of chimney draft. The dilution air flow accounts for a much larger air requirement and heat loss than does the air actually required for combustion, see Table 31.2. This table summarizes the air requirements for various residential combustion appliances. The air requirements for naturally aspirated gas and oil furnaces are about 0.5 and 0.4 ach, respectively, based on an internal house volume of 500 m^3. Thus the amount of air required by heating appliances corresponds to the generally accepted amount of air supplied to the occupants of 0.5 ach. Conventional fireplaces have the highest air demand of 1.4 ach and are extremely inefficient.

Advanced furnace design such as the high-efficiency oil furnace and the induced-draft or condensing gas furnace do not need dilution air. Hence, their air requirements which range from 0.06 to 0.09 ach, are considerably lower than those of naturally aspirated furnaces.

If the house pressure is decreased to the extent that the chimney draft of about 25 Pa is overcome, e.g. by use of extract ventilation systems, a flow

TABLE 31.2

Air demands for residential combustion appliances

Appliance	Air requirement			
	Combustion	Dilution	Total	
	l/s	l/s	l/s	ach*
Conventional Oil	18	54	72	0.52
Retention Head Oil	12	54	66	0.48
High-Efficiency Oil	10	–	10	0.07
Conventional Gas	14	40	54	0.39
Induced Draft Gas	12	–	12	0.09
Condensing Gas	8	–	8	0.06
Fireplace	188	–	188	1.40
Airtight Wood Stove	5	–	5	0.03

* Based on internal volume of 500 m^3.

reversal in a furnace chimney can occur to cause combustion products to be released inside the house. The operation of a fireplace, because of its high air requirement, has the greatest potential to cause flow reversal in the furnace chimney. Operation of a furnace and/or exhaust appliance can result in a flow reversal in the fireplace chimney when the fireplace is burning at low fire.

For vented combustion appliances, furnaces of advanced design requiring no dilution air such as condensing gas furnaces and airtight wood stoves should be used as the air requirements are considerably lower and their efficiencies are substantially higher as compared to those of conventional heating appliances. Also, in airtight houses, the operation of these furnaces, as compared to conventional furnaces, has little detrimental effect on the efficiencies of an air-to-air heat exchanger for heat recovery from the exhaust air. Fireplaces should have a separate supply of outside air and glass doors to isolate the fireplace from the house ventilation system; this increases their efficiencies and minimizes the potential for back drafting of the furnace chimney.

Relatively airtight houses should be checked for adequacy of combustion air and chimney back drafting. If necessary, corrective measures should be taken. These include providing adequate outside air supply and/or reducing the amount of air exhaust by various household exhaust and supplementary heating appliances.

31.5 AIR CLEANING

31.5.1 Evaluation of air cleaners

The evaluation of air cleaners requires information on the efficiency, airflow resistance, dust-holding capacity, effect of dust retention on efficiency and on resistance, and the maintenance which is required to maintain the air cleaner at top efficiency. There is no single test which adequately characterizes all filters or air cleaners. Testing is complex and there are many factors that affect the performance of air cleaners in actual use.

Ideally, testing procedures should approximate the conditions and contaminant concentrations which can be expected to exist during actual use of the devices. The use of standardized tests do allow the performance of different air cleaners and filters to be compared.

Test methods for evaluating air cleaner performance are contained in ASHRAE Standard 52-76 (ASHRAE, 1976) and Military Standard 282 (US: DOD, 1956), which is also referenced by ASHRAE.

These tests are used to evaluate in-duct systems (filters and electronic air cleaners). Recently, the American National Standards Institute (ANSI) and the Association of Home Appliance Manufacturers (AHAM) have developed a standard for evaluating portable air cleaners.

A useful publication, "Residential Air-Cleaning Devices: A Summary of Available Information", which summarizes the available information on residential air cleaners, is available from the Public Information Center of Environmental Protection Agency.

ASHRAE Test methods

The ASHRAE Standard 52-76 test method specifies the evaluation of in-duct air cleaners based on collection efficiency (for total mass and by particle size), pressure drop across the filter, and dust-holding capacity.

Fractional Efficiency or Penetration Test

This test is used for high efficiency filters (efficiencies greater than 98%) which are used in clean rooms and nuclear applications. The Thermal DOP (di-octyl phthalate or bis-[2-ethylhexyl] phthalate) test is conducted by feeding a cloud of uniform particles (0.3 micron DOP) into the filter and determining the percentage of particles removed by the filter (US: DOD, 1956). The concentration of particles upstream and downstream of the filter is measured using a light-scattering photometer or condensation nuclei counter. Results are usually expressed in percent penetration (equal to 100 minus the percent efficiency) rather than efficiency because HEPA filters are almost 100% efficient.

Weight Arrestance Test

The weight arrestance test measures the mass collection efficiency of a filter based on a standard synthetic dust of various particle sizes. The synthetic dust specified by ASHRAE (1976) is composed of 72% standardized dust fine, 23% Molocco black, and 5% cotton liners. The synthetic dust cloud has a particle size range that is larger than typical atmospheric dusts. This test is appropriate for evaluating low efficiency filters which remove larger particulates. These filters are used in residential furnaces, air-conditioning systems, or as upstream filters for other air cleaning devices. The test is not appropriate for evaluating the removal of respirable particulates.

Dust Spot Efficiency Test

The dust spot efficiency test (ASHRAE, 1976) measures how a filter reduces the soiling of residential interiors. The test is conducted by passing untreated atmospheric air through filter paper targets and measuring the difference in light transmittance before and after air is passed through the filter being tested. The spot efficiencies are taken at intervals allowing the efficiency to be evaluated as a function of filter loading. This test is most appropriate for determining the efficiency of high to medium efficiency filters and electronic air cleaners.

Dust-Holding Capacity Test

Dust-holding capacity is the amount of a particular type of dust that an air cleaner can hold before its efficiency drops significantly as a result of the resistance imposed by the collected dust. Airflow resistance, or simply resistance, is the static pressure drop across the filter at a given airflow rate, and the term pressure drop is used interchangeably with resistance.

Because of filter variability, ASHRAE's test (ASHRAE, 1976) determines that the dust holding capacity has been reached based on the following criteria: (1) when the maximum pressure drop specified by the manufacturer or when two consecutive measures of arrestance are less than 85%, or (2) when one value is equal to or less than 75% of the maximum arrestance.

Efficiency Ratings of Filters

Control strategies for indoor air contaminants have recognized the need for efficiency versus size data for sub-micron particles. Manufacturers have attempted to respond to this need by categorizing the removal efficiencies of filters based on particle size. However, these data are difficult to interpret because there is no standard method for determining the efficiency of air cleaners as a function of particle size.

Figure 31.10 summarizes the performance of viscous impingement and dry media filters (ASHRAE, 1988). Note that the percent arrestance, percent

atmospheric dust spot efficiency, and percent DOP efficiency do not correlate with one another. For example, a filter with a 92% arrestance may be 20% efficient based on the dust spot test. This lack of correlation is explained by the types of particles that each test measures.

Group I: Panel-type filters of spun glass, open cell foams, expanded metal and screens, synthetics, textile denier woven and non woven, or animal hair.

Group II: Pleated panel-type filters of fine denier non-woven synthetic and synthetic-natural fibre blends, or all natural fibre.

Group III: Extended surface supported and unsupported filters of fine glass fibres, fine electret synthetic fibres, or wet-laid paper of cellulose-glass or all-glass fibres.

Group IV: Extended-area pleated HEPA-type filters of wet-laid ultra-fine glass fibre paper. Biological grade air filters are generally 95% DOP efficiency; HEPA filters are 99.97 and 99.99%; and ULPA filters are 99.999%.

Notes: (1) Group numbers have no significance other than their use in this figure. (2) Correlation between the test methods shown are approximations for general guidance only.

Potential applications of these filters can be summarized in terms of efficiency ratings from the ASHRAE tests. Based on the atmospheric dust spot test to evaluate efficiency, the following applications and limitations apply (ASHRAE, 1979).

Efficiency rating 10%: applications include window air conditioners; protection of heat exchanger from lint accumulations; relatively inefficient on smoke, settling dust, and pollen.

Efficiency rating of 10–20%: applications include window air conditioners, domestic warm air heating; effective on lint; somewhat effective on common ragweed pollen; relatively ineffective on smoke and staining particulates.

Efficiency rating of 20–40%: applications include air conditioners, domestic heating, central system; at 20% efficiency, fairly effective on ragweed pollen; relatively ineffective on smoke and staining particles; effective as a prefilter for final cleanup filters for clean room; same applications at 40%, but with greater degree of effectiveness; somewhat effective in removing smoke and staining particulates.

Efficiency rating of 40–60%: applications include building recirculated and fresh-air systems; domestic heating and air-conditioning; use as prefilters to high efficiency types; effective on finer airborne dust on pollen; reduce smudge and stain materially; slightly effective on fume and smoke; ineffective on tobacco smoke at 40%; slightly effective on tobacco smoke at 60%.

Efficiency rating of 60–80%: 60% includes same uses as for 40%, but with better effectiveness; 80% used in hospitals and other controlled areas; effective on all pollens, majority of particles causing smudge and stain, fume, coal and

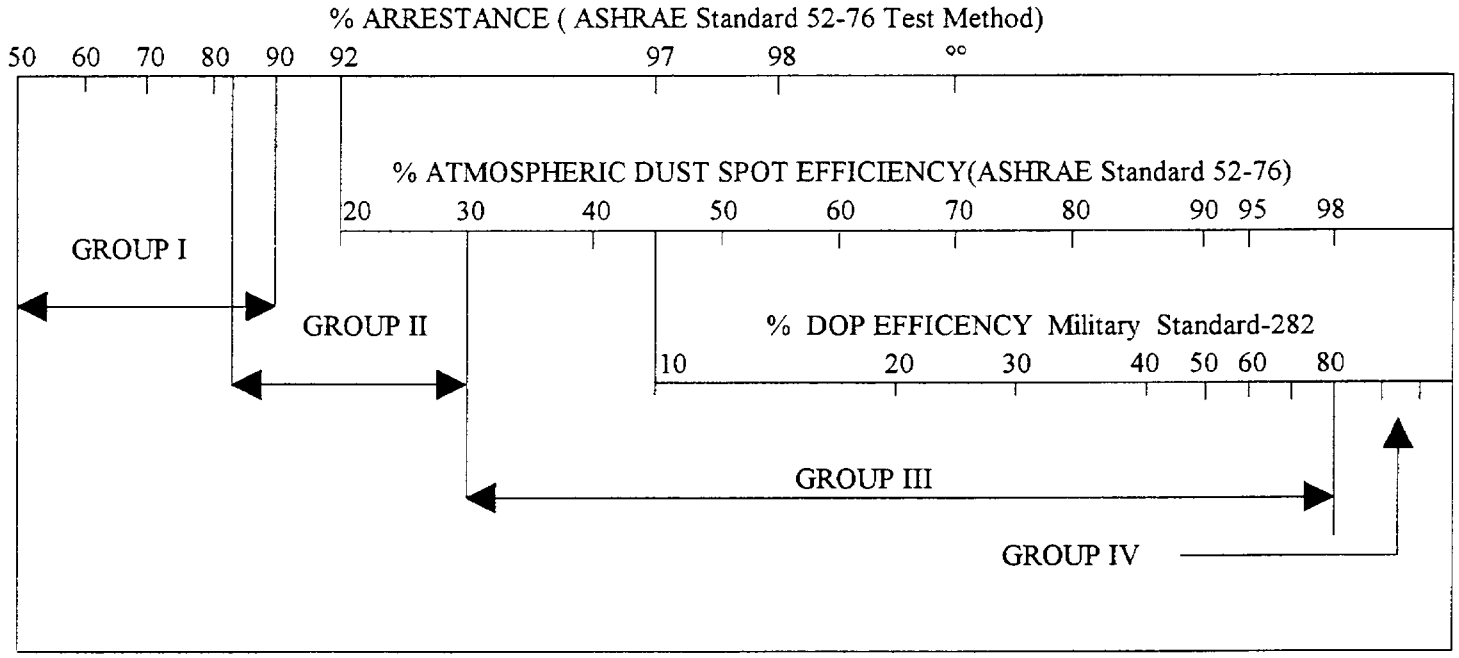


Fig. 31.10: Comparative performance of viscous impingement and dry media filter.

oil smoke; partially effective on tobacco smoke; some types reasonably effective on bacteria, but filters (especially in large buildings) can become a medium for growth.

Efficiency rating of 80–95%: applications include hospital surgeries, pharmaceutical preparation areas, and other controlled areas; very effective on particles causing smudge and stain, coal and oil smoke and fume; highly effective on bacteria but filters (especially in large buildings) may become a medium for growth; quite effective on tobacco smoke.

Efficiency rating of 95%: applications include hospital surgeries, intensive care wards, clean rooms, pharmaceutical packaging; excellent protection against bacteria, radioactive dusts, toxic dusts, all smokes and fumes; filters above 98% efficiency are generally rated using the DOP test method (Military Standard 282).

ANSI/AHAM Test Methods

The ANSI/AHAM AC-1-1988 standard rates the "clean air delivery rate" (CADR) of portable cleaners (AHAM, 1988). The CADR is a measure of how much air a unit is delivering. The delivery of "fresh" air is given in cfm. The fresh air is not 100% fresh because some contaminants may not be removed from the airstream. A cleaner with a CADR rating of 100 can reduce the concentration of a given contaminant equivalent to reductions achieved by adding 100 cfm of fresh air.

Air cleaners with CADR certification based on the ANSI/AHAM standard are evaluated in terms of the removal of dust (10–350 CADRs), tobacco smoke (10–300 CADRs), and pollen (25–400 CADRs).

Table 31.3 shows a comparison of the removal of smoke, dust, and pollen for portable units as a function of CADR and room size, as estimated by AHAM. These figures are representative of air cleaning results based on tests in an airtight room, and they should only be used as guides. At higher CADRs, the importance of fallout from gravity becomes less important.

Table 31.4 compares the amount of time required to achieve 90% removal of airborne particles based on CADRs. As a general rule the higher the CADR, the less time needed to remove the same amount of contaminants from a room of identical size because all things being equal, the more air a cleaners processes, the faster it can remove contaminants.

31.5.2 Particles and air cleaning devices

Source characteristics

The discussion about particles complements chapter 4 of this book which deals with tobacco smoke. Tobacco smoke is the dominant indoor source of

TABLE 31.3

Estimated percentage of particle removal for portable air cleaners by CADR and room size

Room size	Percentage of particles removed						
	CADR	Smoke (20 min)		Dust (20 min)		Pollen (20 min)	
		AC[a]	T[b]	AC[a]	T[b]	ACa	T[b]
5×6	10	49	68	49	70	–	–
	40	89	97	88	98	57	93
	80	95	100	95	100	75	99
9×12	40	53	71	52	72	24	78
	80	76	89	75	89	40	86
	150	89	98	89	98	58	94
12×18	80	53	71	52	72	24	78
	150	74	87	73	88	38	85
	300	89	97	–	–	–	–
	350	–	–	91	99	–	–
	450	–	–	–	–	69	97
18×24	150	51	70	50	71	23	78
	300	73	87	–	–	–	–
	350	–	–	77	91	–	–
	450	–	–	–	–	50	91
20×30	300	63	79	–	–	–	–
	350	–	–	67	84	–	–
	450	–	–	–	–	40	86

[a] Removal by the air cleaning device.
[b] Removal by air cleaning device plus natural settling.
Note: Estimates ignore the effect of incoming air. For smoke and, to a lesser extent, dust, the more drafty the room, the smaller the CADR required. For pollen, which enters from outdoors, a higher CADR is needed in a drafty room.
Source: U.S. EPA (1990). Adapted from Association of Home Appliance Manufacturers (AHAM, 1990). AHAM Consumer guide for Room Air Cleaners. AHAM, 20 North Wacker Drive, Chicago, IL 60606. Used with permission of AHAM.

respirable particles and failure to recognize this fact would represent an artificial separation. Other major indoor particle sources are the outdoor ambient air, combustion sources (dominated by smoking), people and animals, and consumer aerosol products (see also Chapter 3.1).

TABLE 31.4

Time (minutes) to achieve 90% removal of airborne particles*

	Dust (min)	Smoke (min)	Pollen (min)
No air cleaner operating	128	144	22
CADRs-air cleaner operating			
25	49	51	17
40	36	37	15
80	21	21	12
150	12	12	8
300	6	6	5

*Includes removal by fallout from natural forces.
Source: Association of Home Appliance Manufacturers (AHAM), 1990. AHAM Consumer Guide for Room Air Cleaners. AHAM, 20 North Wacker Drive, Chicago, IL 60606, USA. Used with permission.

Examination of indoor and outdoor concentrations of elemental composition of particles showed that particles containing potassium to be from indoor sources, while particles containing sulphur, lead, bromine, and iron had predominantly outdoor sources. Particles are also carriers of organic species in the indoor environment. Characterization of these organic compounds aids both source identification and estimation of personal exposures and possible health effects.

Effects of pollutant

Since the main way of particle absorption is by respiration, only the particles of 0.25–10 μm (aerodynamic diameter) can be considered 'lung-damaging' because bigger particles generally settle in the upper respiratory tract and do not reach the lung. For a complete discussion of health effects of particulate see Chapters 3.1, and 5.2.

Air cleaning devices

The discussion in this section concentrates on particulate matter, hence only air cleaning devices using filter systems are considered.

A typical air cleaning device functions as follows: air is drawn in near the base, through side openings, and the passes through a system of filters before being exhausted at the top. The two parameters critical to the evaluation of such systems are:

(a) The efficiency with which the filter system eliminates the airborne contaminants.

(b) The capacity of the unit, i.e. the volume flow rate through the filter system.

As suspended particles, capable of reaching the lower airways of the lung, form the most important fraction of the total particulate matter in the indoor air, the filter system should be efficient in removing this fraction. Most filters, however, become less efficient as particle size decreases. Average particle sizes in the tobacco smoke e.g. are in the order of 0.5 μm and are retained with an efficiency of only about 20%. A higher degree of retention might be achieved with electrofilters or high efficiency absorption filters (HEPA); but devices equipped with such filters are usually much more expensive.

It is also important to note that the use of air cleaners to control particle concentrations from tobacco smoking is a well-defined procedure but will not eliminate the odours associated with tobacco smoke. Gaseous contaminants may be eliminated from the air by activated carbon filters or with other adsorbents. However it is important to note that not all gaseous contaminants are adsorbed by such filters.

Even the most efficient filter systems fail to improve air quality if the amount of air drawn through the filters is too small: for a 50% reduction in air contaminants (e.g. particulate matter) the room air should be passed through the filters 3–5 times per hour.

31.6 VENTILATION

31.6.1 Introduction

Occupied buildings are designed to provide safe, comfortable environments for people to live and work. Within this statement, the terms safe and comfortable must be applied to two types of occupants; the general population and specific individuals. Thus, what may be considered safe and comfortable (acceptable) to most people may not be acceptable to specific individuals.

In an attempt to define acceptable environments, regulations, standards or guidelines for specific environmental factors (light levels, ventilation and some air pollutants) have been developed by various agencies. The underlying focus of these agencies has been to minimize the negative health effects of exposure while recognizing that there are economic limits to most situations. If the standard is generated by a public health agency, effects on occupant productivity and perceived comfort may be of less concern than documented health risks. In addition to the variation in individual responses and perceptions, the economic relationships among owners/employers/occupants must be considered.

Many standards have established maximum permissible levels based on health risk plus recommendations for enhanced environmental conditions which will increase the number of people who consider the environment acceptable.

It is generally recognized that a supply of ventilation air (outdoor air or indoor air that has been treated to maintain acceptable indoor air quality) is a basic requirement for occupied buildings. Some design standards require specific amounts of ventilation to be supplied to buildings, others require certain indoor pollutant concentrations to be controlled to specified levels.

The purpose of this section is to outline the role of ventilation in controlling indoor pollutant concentrations in buildings and discuss general strategies for providing adequate ventilation.

31.6.2 Role of ventilation in pollutant control

Ventilation is only one component of the heating, ventilating and air conditioning (HVAC) system of a building. Most HVAC systems supply air to a space through grilles or diffusers. The supply air is a mixture of outdoor air plus room air (return air) that is recirculated. Since outdoor air is being added to the system, some provision must be made to exhaust air from the building. This exhaust may occur through leaks in the building envelope or may be vented outdoors with fans. Heat may be extracted from the warm exhaust air stream and reused in the HVAC system. Within the HVAC system, the various air flows may be heated, cooled, cleaned or conditioned in various ways.

To understand the function (and limitation) of ventilation as a means of controlling indoor pollutant concentrations, the relationship between the major factors that influence indoor pollutant levels must be developed.

In the simplified case of a building with one open area and with well mixed air:

$$C_i = C_o + \frac{N}{V} \tag{31.1}$$

where C_i = indoor pollutant concentration ($\mu g/m^3$); C_o = outdoor pollutant concentration ($\mu g/m^3$); N = indoor pollutant generation rate ($\mu g/h$); and V = ventilation rate (m^3/h).

Although this is a simplified model of a real environment, it shows that indoor pollutant concentrations are related to three major variables: the outdoor pollutant concentration, the indoor pollutant generation rate and the ventilation rate. When a pollutant is present in the outdoor air, the effectiveness of the ventilation is reduced since it will carry some of the pollutant

indoors. Variations with time of any of the factors will result in a change in the indoor concentration.

This model also indicates potential control methods for indoor pollutants. Halving the strength of the indoor pollutant source will have the same effect as doubling the ventilation rate. It is very important to identify the origin of pollutant emissions to ensure that an appropriate control method is selected.

Equation 31.1 assumes well mixed air. It does not therefore, highlight the fact that in order for the ventilation to be effective in diluting the indoor pollutants, it must be distributed to the areas where pollutants are being generated. For a more complete discussion on Indoor pollution modelling see also Chapter 23.

31.6.3 Methods of providing ventilation

There are two basic methods used for supplying ventilation to buildings:

(1) *Natural ventilation* is supplied by air leakage into and out of the building enclosure, either through unintentionally created openings in the building (infiltration) or through intentional openings such as windows, doors and louvres. During the winter, cold drafts may exist at places where the outdoor air enters the building while outgoing air can deposit moisture in the building envelope. The flow of air is governed by the size and location of the openings, occupant activities and wind and temperature forces. As a result, the air flow rate is uncontrolled and highly variable. At times, the rate may be excessive (wasting heating or cooling energy) or insufficient, resulting in elevated indoor pollutant concentrations. It is also difficult to ensure that the ventilation is distributed to proper areas of the building.

(2) *Mechanical ventilation* is supplied by a mechanical system, usually including fans and ductwork system for distributing the air throughout a building. The system may also include equipment for controlling humidity, heating or cooling and air cleaning. This type of system can provide a controlled flow of conditioned air to all areas of a building. Control systems (thermostats, humidistats, timers) are normally installed on mechanical systems to regulate their operation and adjust the flows to control indoor temperatures or conserve energy when possible. Problems can occur when the control system does not respond to the indoor pollutant sources. This situation can result from improper design, maintenance deficiencies or changes in building operation or occupancy that were not anticipated in the original design. One example of this type of problem is variable air volume (VAV) systems in large building. The amount of air supplied to a space is regulated by the heating or cooling requirement (thermostat) and unless an acceptable minimum flow is provided, indoor pollutant problems can occur even though thermal conditions are

acceptable. A parallel problem can occur in houses with fresh air intake ducts connected to the furnace return air ductwork. Ventilation is only actively supplied to and distributed within the building when the furnace fan is operating.

Non-residential buildings are usually ventilated by mechanical systems. Tightly sealed residences cannot rely on natural ventilation to supply sufficient quantities of ventilation to all areas. Problems have occurred in defining criteria for deciding whether a building needs a mechanical ventilation system or whether the natural ventilation will be sufficient.

Unfortunately, for many houses, some combination of the two methods is necessary. As the building envelope is made tighter (careful new house construction or retrofitting of an existing house) a smaller fraction of the total ventilation requirement can be reliably supplied by natural ventilation. In the case of some houses, air tightening measures were used to reduce infiltration without consideration of the effect on indoor air pollutant concentrations.

Since the total ventilation requirement is controlled by the indoor pollutant sources, as natural ventilation quantities are reduced, mechanical ventilation quantities must be increased unless pollutant sources are reduced accordingly.

31.6.4 Pollutant sources

Pollutant sources must be clearly identified as originating from outdoors or indoors, since strategies for controlling the resulting indoor pollutant levels will be quite different.

Outdoor pollutants can include widespread, constantly elevated levels of pollutants (e.g. industrial emissions) or specific intermittent sources such as delivery vehicle exhaust fumes entering ventilation air intakes. Dust, pollen and other allergens are also outdoor pollutants.

Indoor pollutant sources can be broadly categorized as occupant related and non-occupant related sources.

(1) *Occupant related sources*: Indoor pollutants related to occupancy encompass a wide spectrum ranging from emissions from the human body (CO_2 water vapour, bacteria and viruses or chemicals from other metabolic processes) to emissions from human activities (smoking, perfumes, cleaning chemicals, photocopier emissions, clothing). Typically, these sources vary widely on an hourly and daily basis. Ventilation systems must be capable of diluting all of these highly variable "uncontrolled" sources to ensure acceptable levels.

(2) *Non-occupant related sources*: This category includes emissions from building materials and interior furnishings (formaldehyde, volatile organic compounds, asbestos and other particulates), indoor combustion sources (CO_2,

NO_X, CO) and a wide variety of other specific products. These sources can vary over a short (hourly or daily) or long (yearly) term.

In some cases, data on the strength of pollutant sources is available but more often the system designer must estimate the sources based on experience alone. New buildings may have an initially high rate of off-gassing of chemicals from the building materials which will decrease with time.

31.6.5 Ventilation system design

The ventilation system designer must select a system which will provide adequate ventilation for a potentially endless, undefined mixture of pollutants. The system must be cost effective which often means energy efficient, comfortable and flexible enough to accommodate future changes.

Ventilation codes and recommendations have been developed based on some typical pollutant sources to give designers guidance for selecting reasonable ventilation rates (Table 31.5). Individual circumstances must be reviewed to ensure that other special pollutant sources do not exist. In special situations, the pollutant mass balance model (Eq. 31.1) must be used.

Ventilation for diluting internally generated pollutants should be used as supplement to good engineering practice, rather than a cure for all indoor air quality problems. Often, removing a pollutant source or isolating it from the main occupied space is more appropriate than allowing it to escape into the air and controlling the concentration by dilution.

In general, the key elements to proper ventilation system design include:

Continuous supply

Since indoor pollutant sources include continuous and variable sources, if maximum indoor pollutant concentrations are to be controlled without "over ventilating", the ventilation system must supply a basic minimum flow of air continuously as well as being able to increase the air flow to compensate for increased pollutant emissions. Universal sensors for detecting all forms of "unacceptable" indoor air quality do not exist. The system designer must, therefore, arbitrarily select a control criteria. Manual switching, programmable timers and temperature, humidity or CO_2 sensors are some methods used to try and reduce mechanical ventilation when possible.

Infiltration cannot provide reliable ventilation for pollutant control. Natural ventilation is normally controlled by individuals who open and close windows based on their perception of comfort. These changes can have major effects on the ventilation and pollutant concentrations in other areas of the building.

TABLE 31.5

Selected ventilation recommendations

Application		Occupancy (people/1000 ft^2)	Cfm/person	Cfm/ft^2
Food and beverage service	Dining rooms	70	20	
	Cafeteria, fast food	100	20	
	Bars, cocktail lounges	100	30	
	Kitchen (cooking)	20	15	
Offices	Office space	7	20	
	Reception areas	60	15	
	Conference rooms	50	20	
Public spaces	Smoking lounge	70	60	
	Elevators			1.00
Retail stores, sales floors, showroom floors	Basement and street	30		0.30
	Upper floors	20		0.20
	Malls and arcades	20		0.20
	Smoking lounge	70	60	
Sports and amusement	Spectator areas	150	15	
	Game rooms	70	25	
	Playing floors	30	20	
	Ballrooms and discos	100	25	
Theatres	Lobbies	150	20	
	Auditorium	150	15	
Education	Classroom	50	15	
	Music rooms	50	15	
	Libraries	20	15	
	Auditoria	150	15	
Hotels, motels, resorts, dormitories	Bedrooms			30 cfm/room
	Living rooms			30 cfm/room
	Lobbies	30	15	
	Conference rooms	50	20	
	Assembly rooms	120	15	

Control strategies should be developed from a through understanding of the construction and operation of the building. Occupant behaviour floor plans, time dependent characteristics of the potential pollutant sources and use schedules must be developed and evaluated.

Distribution and flow

Proper air distribution is required to ensure that stale or "dead" areas in the building do not exist. The selection and placement of supply air diffusers and return air grills can have a major impact on the effectiveness of the ventilation to control concentrations. Changes in interior floor plan and architectural layout can also affect air distribution.

Since the ventilation air is supplied in a relatively "clean" state and increases in pollutant concentration as it moves through the building to the return air system, the location of supply and return air grilles relative to the occupants can minimize the exposure to the more contaminated portions of the room air.

Provision for maintenance and adjustment

All mechanical equipment is subject to wear and neglect. Air cleaning and humidifying equipment, drive belts, fans and dampers will require cleaning and adjustment to ensure their proper performance. New mechanical systems must be thoroughly checked for proper operation and the owner/operator must be instructed as to the purpose and maintenance needs of the system components.

Relative humidity control

During cold winter conditions, interior relative humidity levels can become excessive. Condensation can occur on windows, areas that are poorly insulated and areas that become cold due to inadequate air circulation. Reducing the set point temperature of heating thermostats (e.g. night setback) can aggravate the condensation problem since surface temperatures (especially exterior walls and windows) will drop and fan operation (air circulation) may be interrupted. Condensation can cause problems with appearance (staining), increased heat loss (wet insulation), building contaminants such as moulds and fungi.

Interaction with other activities

HVAC systems can affect or be affected by other processes within a building. Pressure differences within a building caused by wind or temperature forces (stack effect) or by mechanical equipment can affect the strength or distribution of indoor pollutant sources.

Chimney back-drafting (entry of combustion by-products) can occur when the negative pressure in a building interferes with the proper operation of a chimney. The rate of radon gas entry from the soil into a building foundation may also be increased when the below grade portion of a building is under a negative pressure.

The effect of any modification to a building should be carefully evaluated to assess its potential impact on the indoor environment.

31.6.6 Ventilation effectiveness

Ventilation requirements are generally derived using the assumption that both the ventilating air and the pollutants are uniformly mixed throughout the ventilated space. Such conditions may not occur in practice and it is necessary to take the effectiveness of any means of ventilation into account. This involves the following considerations:

1. The average rate with which "old" polluted air is replaced by "new" clean air in a space, characterized by the air exchange efficiency
2. The degree to which pollutants are removed and prevented from spreading to specific areas, such as the occupied zone. This is called pollutant removal effectiveness.

Air-exchange efficiency

The first task is described with the age of the room air, with the age equal to zero when it enters the space. The age of the air can be measured using tracer gases. The age itself is seldom used directly to describe the ventilation, but it is used to define air-exchange efficiency. This can be defined as the ratio of the shortest possible age to the mean age of the air in the actual space:

$$fg_a = \frac{n'^2}{<>} 100\%$$

where fg_a = the air-exchange efficiency; <> = the mean age of the air in the space with volume V; n'^2 = the shortest possible age of the air in the space; $_n$ = the nominal time constant of the ventilation, $n = V/q_o$; V = the effective volume of the room; and q_o = the total outdoor air flow into the space.

The average age depends on the air distribution system and can vary considerably even with the same air flow rate. A short average age of the air is desirable. The shortest possible age of the air, n'^2, is obtained with piston flow, in which a large piston is assumed to push the old air from the room. The highest value of air-exchange efficiency, 100%, is obtained with theoretical piston flow. The air-exchange efficiency of complete mixing is 50%; with

TABLE 31.6

Estimating outdoor air quantities

Using thermal mass balance

$$\text{Outdoor air (percent)} = \frac{T_{\text{return air}} - T_{\text{mixed air}}}{T_{\text{return air}} - T_{\text{outdoor air}}} \times 100$$

where: T = temperature (degrees Fahrenheit)

Using carbon dioxide measurements

$$\text{Outdoor air (percent)} = \frac{C_S - C_R}{C_O - C_R} \times 100$$

where: C_S = ppm CO_2 in the supply air (if measured in a room), or
C_S = ppm of CO_2 in the mixed air (if measured at an air handler
C_R = ppm of CO_2 in the return air
C_O = ppm of CO_2 in the outdoor air
(All these concentrations must be measured, not assumed.)

Converting percent to cfm

$$\text{Outdoor air (cfm)} = \frac{\text{Outdoor air (percent)}}{100} \times \text{total airfloor (cfm)}$$

where: cfm = cubic feet per minute

The number used for total airflow may be the air quantity supplied to a room or zone, the capacity of an air handler, or the total airflow of the HVAC system. Note: The actual amount of airflow in an air handler is often different from the quantity in design documents.

displacement flow pattern it is between 50 and 100%; and with short circuiting ventilation, it is under 50%.

Ventilation effectiveness calculation

Outdoor air for controlling contaminant concentration can be used for dilution or for sweeping the contaminants from their source. The values in Table 31.6 define the outdoor air needed in the occupied zone for well-mixed conditions (ventilation effectiveness approaches 100%). The ventilation effectiveness is defined by the fraction of the outdoor air delivered to the space that reaches the occupied zone.

Ventilation effectiveness may be increased by creating a plug flow situation. If the flow pattern is such that the ventilation air flows past the contaminant source and sweeps the contaminant towards an exhaust, the contaminant concentration in the exhaust can be greater than that for the well-mixed condition. Ventilation effectiveness can then be greater than that

which would be realized with perfect mixing. Local exhaust systems operate in this way. With perfect mixing between the ventilation air and the air in a space, ventilation effectiveness is 100% (E_v = 1.0). It is, however, not uncommon to find some of the ventilation air bypassing the occupants (moving from supply to exhaust without fully mixing in the occupied zone) and achieving E_v values as low as 0.5 or less. Such flow conditions should be avoided. The ability of the ventilation air to mix in the occupied zone can be improved through recirculation or active mixing of the air in the space.

A model for ventilation effectiveness

A model for ventilation effectiveness can be derived by considering a typical HVAC air-handling system as shown schematically in Figure 31.11. It is possible that a fraction, S, of the supply air may bypass directly to the return inlet without mixing at the occupied level, i.e. below the dotted line in Figure 31.11.

The total outdoor air in the supply air is V_{os}.

The fraction of the supply air that stratifies and bypasses directly to the return is designated by S. This model applies to the forced ventilation system and excludes the effects of passive infiltration. The amount of outdoor air supplied to the space is:

$$V_{os} = V_0 + R \times S \times V_{os} \tag{E1}$$

The amount of unvitiated (unused) air that is exhausted is:

$$V_{oe} = (1 - R)\, S\, V_{os} \tag{E2}$$

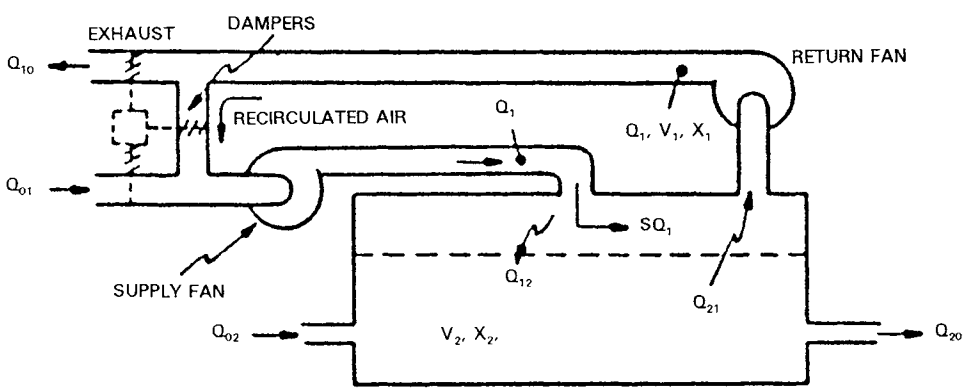


Fig. 31.11: Typical air distribution system.

The ventilation efficiency then can be defined as:

$$E_v = [V_o - V_{oe}] / V_o \tag{E3}$$

Combining Eqs. E-1, E-2 and E-3

$$E_v = [1 - S] / [1 - RS] \tag{E4}$$

Equation E-4 defines the effectiveness with which the outdoor air is circulated to the occupied space in terms of a stratification or mixing factor S and the recirculation factor R. If there is no exhaust flow, $R = 1$ and the effectiveness = 100%. If, however, there is both stratified flow and recirculation outdoor air can pass through the system without ever being used to dilute contaminants at the occupied level. This ventilation loss also represents an energy loss.

31.6.7 Calculating ventilation rates

Assuming homogeneous mixtures, the steady state concentration C_s of a gaseous contaminant at time in an indoor space of volume v being generated at a rate N can be calculated from mass balance as

$$C_s(t) = C_o + [N / V_o] \, [1 - \exp(- V_o t / v)] \tag{31.2}$$

where C_o = contaminant concentration in the outdoor air; N = contaminant generation rate in the space; V_o = ventilation rate (indoor/outdoor air exchange rate).

At steady state, the concentration is

$$C_s(t) = C_o + N / V_o \tag{31.3}$$

If the contaminant source in the space is removed, the concentration at a time t_a thereafter is

$$C_s(t) = C_i \exp(- V_o t_a / v)] \tag{31.4}$$

where C_i = contaminant concentration in the space at the time of source removal.

Equations 31.2–31.4 assume that the contaminant is uniformly distributed throughout the space, that it does not react chemically within the space, and

that it is not adsorbed into interior surfaces. Temperature and humidity factors are not included. Most significantly, these equations represent "steady state" relationships among the variables. In fact, indoor environments are dynamic and steady state conditions are not usually obtained indoors. Nonetheless, the equations describe the most important relationships and are useful in estimating values to within a first-order approximation.

An artificial tracer gas, such as SF_6, can be injected at a steady rate and its spatial concentration measured. The ventilation rate is then calculated from Eq. 31.2. Alternatively, a tracer gas can be injected into the space and its decay rate measured. Equation 31.4 is then used to calculate ventilation rate.

The use of a special tracer gas requires the availability of equipment to inject, capture, and quantify it. As a result, carbon dioxide is often used as the tracer. It is generated by people and so provides the ventilation rate on a per person basis. However, some precautions should be observed in its use. Concentrations must be measured outdoors as well as indoors. Non-human sources (e.g. combustion spillage, vehicular exhaust) must not be present.

In spaces with variable occupancy and a constant ventilation rate, carbon dioxide concentration rises from background levels with the arrival of the occupants. Sometimes CO_2 does not reach its equilibrium value before the occupants begin to leave for lunch. If the occupants return before the concentration has fallen significantly CO_2 will rise to a second peak before the occupants begin to leave again. In estimating the ventilation rate from CO_2 concentration, Eq. 31.3 applies only if the equilibrium value is measured. Failing that, one must use Eq. 31.2 or 32.4 along with the appropriate time measurement.

Carbon dioxide production rate can vary significantly depending upon activity in the space. A number of typical activity level carbon dioxide production rates are provided with the ASHRAE Ventilation Standard document. Production rates under other situations, for example, activities creating mental stress such as students writing examinations, may have to be calibrated specifically.

Some examples of ventilation calculations using different activity levels and Eq. 31.2 and 31.3 are provided in Table 31.7.

31.6.8 Minimum physiological requirements for respiration air

Oxygen is necessary for metabolism of food to sustain life. Carbon and hydrogen in foods are oxidized to CO_2 and H_2O, which are eliminated by the body as waste products. Foods can be classified as carbohydrates, fats, and proteins, and the ratio of carbon to hydrogen in each is somewhat different.

TABLE 31.8
Required outdoor air or space contaminant concentration with recirculation and filtration

Class	Required Recirculation Rate				Required Outdoor Air	Space Contaminant Concentration	Required Recirculation Rate
	Filter Location	Flow	Temperature	Outdoor Air			
I	None	VAV	Constant	100%	$V_o = \frac{N}{E_v F_r (C_s - C_o)}$	$C_s = C_o + \frac{N}{E_v F_r V_o}$	Not applicable
II	A	Constant	Variable	Constant	$V_o = \frac{N - E_v RV_r E_f C_s}{E_v (C_s - C_o)}$	$C_s = \frac{N + E_v V_o C_o}{E_v (V_o + RV_r E_f)}$	$RV_r = \frac{N + E_v V_o (C_o - C_s)}{E_v E_f C_s}$
III	A	VAV	Constant	Constant	$V_o = \frac{N - E_v F_r RV_r E_f C_s}{E_v (C_s - C_o)}$	$C_s = \frac{N + E_v V_o C_o}{E_v (V_o + F_r RV_r E_f)}$	$RV_r = \frac{N + E_v V_o (C_o - C_s)}{E_v F_r E_f C_s}$
IV	A	VAV	Constant	Proportional	$V_o = \frac{N - E_v F_r RV_r E_f C_s}{E_v F_r (C_s - C_o)}$	$C_s = \frac{N + E_v F_r V_o C_o}{F_r E_v (V_o + RV_r E_f)}$	$RV_r = \frac{N + E_v F_r V_o (C_o - C_s)}{E_v F_r E_f C_s}$
V	B	Constant	Variable	Constant	$V_o = \frac{N - E_v RV_r E_f C_s}{E_v [C_s - (1 - E_f) C_o]}$	$C_s = \frac{N + E_v V_o (1 - E_f) C_o}{E_v (V_o + RV_r E_f)}$	$RV_r = \frac{N + E_v V_o [(1 - E_f) C_o - C_s]}{E_v E_f C_s}$
VI	B	VAV	Constant	Constant	$V_o = \frac{N - E_v F_r RV_r E_f C_s}{E_v [C_s - (1 - E_f) C_o]}$	$C_s = \frac{N + E_v V_o (1 - E_f) C_o}{E_v (V_o + F_r RV_r E_f)}$	$RV_r = \frac{N + E_v V_o [(1 - E_f) C_o - C_s]}{E_v F_r E_f C_s}$
VII	B	VAV	Constant	Proportional	$V_o = \frac{N - E_v F_r RV_r E_f C_s}{E_v F_r [C_s - (1 - E_f)(C_o)]}$	$C_s = \frac{N + E_v F_r V_o (1 - E_f) C_o}{E_v F_r (V_o + RV_r E_f)}$	$RV_r = \frac{N + E_v F_r V_o [(1 - E_f) C_o - C_s]}{E_v F_r E_f C_s}$

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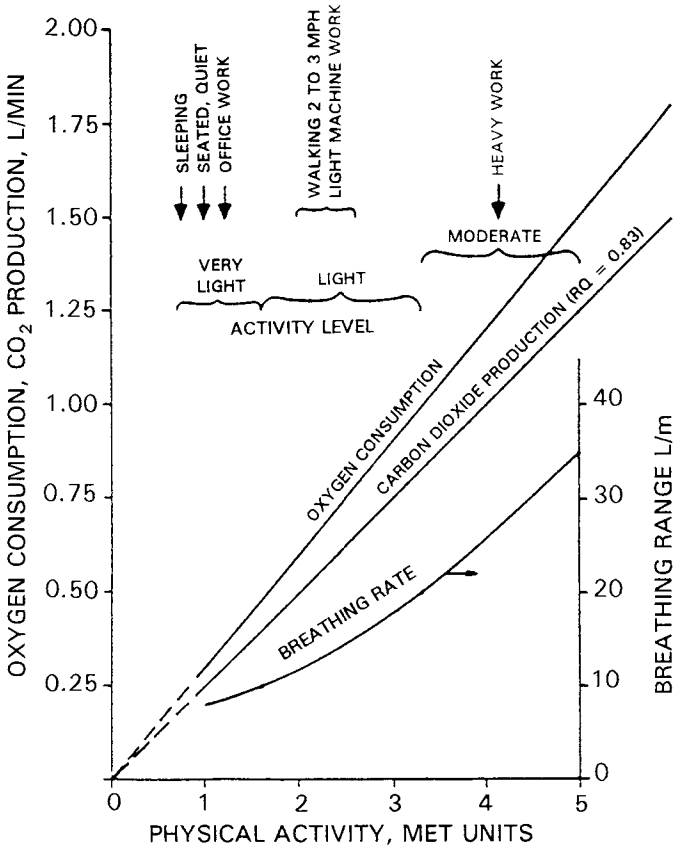

Fig. 31.12. Metabolic data.

The respiratory quotient (RQ) is the volumetric ratio of carbon dioxide produced to oxygen consumed. It varies from 0.71 for a diet of 100% fat to 0.8 for a diet of 100% protein and 1.00 for a diet of 100% carbohydrates (see Ref D-1). A value of RQ = 0.83 applies to a normal diet mix of fat, carbohydrate, and protein.

The rate at which oxygen is consumed and carbon dioxide is generated depends on physical activity. These relationship are shown in Figure 31.12. The breathing rate is also shown. A simple mass balance equation gives the outdoor air flow rate needed to maintain the steady-state CO_2 concentration below a given limit.

Thus, for an activity level of 1.2 met units (1.0 met = 18.4 Btu/h·ft^2) the CO_2 generation rate is 0.30 l/min. If the maximum space concentration is to be held to 0.1% and the outdoor concentration is 0.03% the outdoor air flow needed can be calculated.

Figure 31.5 shows the outdoor air flow rate required as a function of physical activity and steady-state room concentration. If the activity level is greater than 1.2 met, the required ventilation must be increased to maintain the same carbon dioxide level.

Also the decrease in oxygen content of the room air can be found from Figure 31.12 when oxygen concentration is substituted for carbon dioxide concentration.

The term N now has a negative value with respect to its use in equation notion since oxygen is consumed rather than generated.

The oxygen consumption rate is 0.36 l/min when the activity level is 1.2 met. For ventilation at a rate of 15 cfm (429 l/m) and an activity level of 1.2 met units, the room oxygen level will be reduced from an outdoor concentration to 20.9%. The oxygen content of the room is reduced from 21 to 20.9%, a change of only 0.5%. The carbon dioxide is raised from the background of 0.03 to 0.1%. a change of 230%. Thus dilution of carbon dioxide is clearly more significant than replacing oxygen.

31.7 SELECTION OF BUILDING MATERIALS, FURNISHING AND MAINTENANCE MATERIALS

31.7.1 Introduction

Many materials contain and emit chemical substances which can cause a wide spectrum of adverse effects. A scientific risk assessment of a building material should be based on chemical and biochemical, animal and human physiological, toxicological, and epidemiological data as well as on adequate exposure data. If a full risk assessment cannot be made at least the following requirements on materials should be met:

- information on composition and emission rates;
- emission analysis by chemical and sensory chamber testing;
- theoretical evaluation of the toxicity of the emissions; and
- a relative comparison as to alternative products.

There is sufficient knowledge to classify all building materials or their emissions into groups with respect to carcinogenesis, and most components in building materials as to chemical irritation.

For healthy buildings it is essential to choose building materials with a minimum pollutants emission to the indoor air. In addition, the materials should be used in an adequate and safe way with regard to environmental factors such as humidity, temperature, etc. The following solutions are considered to be risky:

- materials with a large surface area, such as wall-to-wall carpets in public premises; if materials of this kind are essential in view of, for example, noise protection, the cleaning level must be raised;
- materials that are not accompanied by a "statement of contents" in respect of health importance or have not been "well-tried and proven for a long time";
- materials containing substances that may be suspected of being adverse to health or comfort in the concentrations concerned;
- use of protective agents for control of biological degradation; the building shall be constructed so that such agents are superfluous.

Many different types of materials are used in the construction, furnishing, maintenance, and operation of a building. In addition, typical buildings contain materials and products used by the occupants for various purposes. In broad terms, these items can be classified as building materials, furnishings, maintenance materials, and other contents. At any given time, emissions from materials and products in any of these four categories can dominate the impact on indoor air quality in a building.

Responsibility for the selection of these materials may rest with the designer, owner, or occupants. Information on IAQ impacts of materials therefore needs to be developed for a wide range of people. This section will summarize the various criteria that can be used in material selection, and the types of material and product testing that can be especially useful for selection.

31.7.2 Selection criteria

Any building materials, furnishings, maintenance materials, or other contents of a building are selected with various physical, aesthetic, economic, and environmental criteria in mind. Examples of such criteria are listed in Table 31.8. Hundreds (sometimes thousands) of material selections need to be made for a typical building, and several of these criteria often conflict.

Historically, environmental criteria have not played a major role in materials selection. In recent years, however, the balance has shifted somewhat to give environmental considerations greater weight. Evidence has to give to the life-cycle concept that includes total environmental impacts during production, use, and disposal of the materials.

A "life-cycle" concept is also important to apply to materials during the entire period they are used in buildings. To date, most discussions of material selection for IAQ protection have dealt with building materials and furnishings, and have focused only on the period when they are new. This is mainly because of many well-publicized complaints of irritation by occupants of new and newly renovated buildings. Emissions of organic vapours can be high in

TABLE 31.8

Selection criteria for indoor materials

Attribute	Example
Physical	strength
	durability
	heat transmission
	light transmission
	maintainability
	effectivness (e.g. as a cleaning agent)
Aesthetic	colour
	texture
	odour
	noise
Economic	initial cost
	maintenance cost
	operating cost (e.g. energy)
Environmental	emission to air
	other releases
	support of microbial growths
	life-cycle impacts

such situations, but frequently decrease markedly over a period of days, weeks, or months.

Occupant exposures to emissions from periodic building maintenance, other work, and personal activities can also be high. These periodic, short-term exposures, in addition to initial emissions from new materials, are thought by some to be the triggering causes of many "sick" buildings that are investigated-without success-several weeks or months after complaints are first expressed.

Degradation of materials leading to particle emissions and biocontamination of materials that have become soiled and wet can also be potentially severe sources of IAQ problems. Therefore, maintainability, durability, and susceptibility to microbial growth are important (and often overlooked) factors in selection of materials for good IAQ.

31.7.3 Desirable characteristics of materials

From an indoor environmental point of view, there are several characteristics that could be considered in describing an "ideal" material. These

TABLE 31.9
Desirable characteristics of indoor materials

Factor	Desirable characteristic
Emission properties	low emission rates
	low toxicity of emissions
"Sink" properties	Non-sorbent
	if sorbent, not re-emitting
	if not re-emitting, non-nutrient
Microbial properties	as needed for the application
Aesthetic properties	as desired for the application
Cost	reasonable

characteristics are listed in Table 31.9, along with comments on physical, aesthetic, and economic factors.

Not many materials will have all of these characteristics, of course. Compromises will usually have to be made, and many that at first seem undesirable may turn out to be quite acceptable. For example, a material with a high initial emission rate but rapid decay may be quite acceptable if used in a manner that does not lead to high exposures, either during use or from re-emissions from interior surfaces that have adsorbed the emissions. Note, however, that products with high contents of volatile organic compounds are coming under scrutiny in many urban areas where there are high levels of photochemical oxidants, or "smog", in the outdoor air.

31.7.4 Indoor air quality criteria

Some criteria exist for acceptable concentrations of various indoor air pollutants. Most are in the form of guidelines instead of official standards. None have been accepted by national or international agencies as necessarily sufficient to ensure acceptable IAQ. For those substances that are most likely to be of concern as emissions from materials, the criteria are usually close to following maximum values:

Odours	Levels that are acceptable to 80–90% of visitors as they enter a space
Particles	50 $\mu g/m^3$ (respirable particles)
Organic Vapours (total)	300 $\mu g/m^3$ (long-term exposures); 3000 $\mu g/m^3$ (short-term exposures)

Organic Vapours (individual)	less than 100 $\mu g/m^3$ (long-term exposures); much less than that for especially noxious compounds

Similar criteria for fibres and for microbial contaminants like fungi, viruses, and bacteria have not been widely proposed. The guideline criteria for organic vapours are generally based on limited studies of human exposures and analyses of data from field investigations of buildings; they do not, therefore, necessarily translate into a particular (acceptable) level of risk.

While IAQ criteria provide a basis and starting point, they are not by themselves useful for selecting acceptable materials. Criteria for emissions are needed.

For indoor air quality protection, the key to selecting materials is knowing the maximum emission rates that will lead to acceptable indoor concentrations-or more accurately, acceptable exposures to occupants -in the particular building of concern. These maximum emission rates will be very situation-specific. The major situation-specific factors that affect occupant exposures to pollutants emitted from indoor materials and products include:

- the number and types of materials and products present;
- the amounts present, especially relative to the volume of the indoor space affected;
- air exchange (ventilation) rates between the building and the outdoor air;
- indoor recirculation rates and air flow patterns;
- the proximity of the material to occupants (which can greatly affect how well ventilation will reduce exposures).

Since these factors are highly variable from building to building, and often from area to area within a building, maximum emission rates are best determined by IAQ modelling. This can range from back-of-the-envelope calculations to sophisticated computer modelling, but for most situations a relatively simple personal-computer-based model will be sufficient.

Starting from the best available IAQ criteria, modelling can estimate the maximum advisable emission rate. The emission rate then usually needs to be divided by the amount of material or product to be used; that will yield an emission factor (emission rate per unit). The most common units will be square meters, kilograms, or the number of items used.

This maximum advisable emission factor can then become the IAQ basis for selection of indoor materials and products. Since emissions from many materials change greatly with time, the time dependency of the emission factor should be considered. For most situations, emissions at the time when building occupants are first exposed are the most relevant.

One approach to material selection is to obtain the lowest-emitting product available. We also have to consider the products in relation to their own performances over the time. While this approach is relatively simple in that it avoids the need to do any modelling, it may sometimes lead to selection of lower-emitting materials than are really necessary. It should also be kept in mind that lower-emitting is not necessarily lower-impact; not all emissions are toxicologically equal. However, given our current state of knowledge of the health and comfort effects of mixtures of pollutants — which is what most materials emit — the most practical approach at present may be to select materials with the lowest overall emission factors that also meet the non-environmental selection criteria listed in Table 31.8.

31.7.5 Emission testing

If emission factors are to be used in material selection, data from emissions testing are needed. Proper testing involves the use of environmental chambers with carefully controlled air flows and environmental conditions. Many researchers have been using such chambers in recent years. Testing procedures, while not really standardized yet, are sufficiently well understood that a few commercial testing laboratories now do emission testing for product manufacturers, designers, or building owners. Several manufacturers are also set up to test their own products.

Most IAQ-related testing of materials and products measures the physical and chemical substances emitted. As mentioned above, the predicted exposures to these emissions can be difficult to evaluate toxicologically. That is because IAQ criteria are generally not available for mixtures, and human responses are therefore very difficult to predict.

Until methodological improvements in evaluating chemical and physical mixtures are made, health and comfort assessments will have to rely on evaluations of the individual toxic pollutants that are emitted, and on relative comparisons of the total emission rates of classes of pollutants. The latter approach is now often used for total organic vapours (often called total volatile organic compounds, or "TVOCs").

Using human responses directly to evaluate emissions is done less often, although olfactory response is the basis of the "olf" concept for assessment of perceived IAQ. Of course, many other health endpoints (e.g., eye irritation, airway irritation) are also of concern. For some of those end points, testing methods that use biological response systems (humans, animals, or in vitro assays) may be more accurate predictors than chemical-analysis-based methods. Over the next several years, the feasibility of using biological-response-based tests will be evaluated.

Either type of testing — chemical/physical or biological-response-based — could be used as the basis for labelling indoor materials and products. Labels could show numerical values for emission factors or intensities of biological responses under standard testing conditions. This would be conceptually similar to energy efficiency labelling of household appliances or gasoline mileage ratings of automobiles.

Selection of a material could then be based on (1) direct comparison of information on the labels, or (2) modelling to predict occupant exposures and comparison to appropriate IAQ criteria. The first approach has the advantage of simplicity, but may lead to selections of materials that are lower-emitting (and potentially more costly or less desirable from other standpoints) than necessary for the application. The second approach has the advantage of being specific to the application, but is often not feasible because appropriate IAQ criteria do not exist. In summary, labelling that is based on emission testing holds promise for making more informed selections of indoor materials and products, but much work lies ahead to develop satisfactory testing protocols and labelling schemes.

In summary:

- Many IAQ problems are caused by unnecessarily strong sources of emissions of chemical, physical, and microbial contaminants.
- Methods for evaluating emissions rates and the compositions of those emissions are reasonably well developed.
- Methods for predicting emission dispersion and inhalation exposures are reasonably well advanced but further development is needed.
- Methods for directly evaluating the health effects of emissions are needed.
- Bioresponse methods have potential for improving our prediction of health effects.

31.8 EVALUATING BUILDING MATERIALS

The designer, builder, product manufacturer, and installer all share the responsibility for providing safe building materials. The manufacturer is in the best position to assure a product's safety by testing and quality control, by providing installation and maintenance specifications, and by developing safer products. However, the design professional must take the lead in compiling product information, choosing the best products, and encouraging safe product development.

The building materials evaluation process has four phases: identifying target products; screening target products; emissions testing; analysis and recommendations.

31.8.1 Identifying target products

Most building projects, whether they be simple residences or complex skyscrapers, use literally hundreds of separate products. To make materials evaluation manageable, one should identify "target" products based on: the potential for occupant exposure to their emissions and the danger presented by such exposure.

Occupant exposure depends on a material's emission (off-gassing) characteristics, the quantity of the material used in the building, the proximity of the material to occupants, and other factors. The danger of exposure depends on the toxic, irritating, or sensitizing effects of the emitted chemicals and the susceptibility of the exposed population. A preliminary review of the building products and furnishings will result in a "short list" of target products that should be reviewed in more detail.

To identify target products one should first consider the overall building design, the anticipated use of space, and possible material and product selections, second review the intended use of major materials, and, third, consider all questionable products and materials for screening. In general, the list of target materials will include adhesives, paints, caulks, sealants, and insulations as well as floor coverings, wall coverings, ceiling system, HVAC duct materials, and most furnishings.

31.8.2 Screening target products

Target products should be screened by determining their quantity and distribution in the building, their chemical composition and the toxic, sensitizing, or irritation potential of their major chemical constituents, and their potential for emissions (e.g., wet versus dry materials).

The screening process helps to identify the products most likely to emit significant quantities of irritating or toxic substances. These usually include the carpet system (carpet, pad or backing, and adhesive), office furnishings (work surfaces, shelving, and interior partitions), and ceiling tiles. Storage systems, adhesives, caulking compounds, paints, sealants, and wood finishes are also materials of concern.

For the quantitative assessment one should start with determining the extent of product use and use per unit of floor area or building interior volume.

Materials such as floor coverings and ceiling tiles are significant due to the large extent of their use as a rough estimate, each have virtually 100% coverage (one unit of material per unit of floor area). If the ventilation system uses the concealed space above a suspended ceiling as a return air plenum,

then both the upper and the lower surfaces of the ceiling tiles are exposed to the circulating indoor air. Thus, ceiling tiles approach 200% of floor area coverage.

Work surfaces (desktops) usually have between 15 and 35% coverage. In modern work station component systems, this desktop material is often used for shelving in work station closets which can add an additional 10 to 20% to the coverage ratio. This material is usually exposed on both upper and lower sides and is especially significant due to the large amount of contact or close proximity between the office workers and the product. The work surface is often a plastic laminate covering a composite board core. If the laminate does not completely seal the unit, the core is exposed to the air stream and emissions are larger.

The coverage of work station interior partitions generally approaches or exceeds 200% of the floor area in open office areas and can exceed 300%. Again, two sides of the product are exposed to the indoor air, and the product is also in close proximity to office workers.

The plane geometric surface area can be significantly less than the virtual surface area of a material with respect to emissions and sink effects. So-called "fleecy" materials such as carpets, drapes, upholstery, and most insulations have very high surface areas due to their texture. Carpet pile height can greatly affect virtual surface area. Therefore, surface area evaluations should also include consideration of the fleeciness of materials.

31.8.3 Chemical content analysis

Theoretically, each material used in a building, product, and furnishing is covered by material safety data description. In some countries such descriptions are standard procedure in occupational settings. One should always require all potential vendors to provide a material safety data description for all products assembled by them and the names of suppliers of each product not assembled by them. Such descriptions, when they exist, sometimes are incomplete or inaccurate and should be relied upon with caution. Chemicals present in very low concentrations may not be reported. Contaminants unknown to the preparer may also be omitted.

The potential for chemical emission should be assessed by reviewing the vapour pressure and molecular weight data for chemicals of concern as identified. Emission factors can vary significantly — up to a factor of 1000 — for different brands of similar products. Therefore, it is important to obtain as much information as possible about the types and quantities of constituents in a given product. While such a "paper" evaluation is not definitive, it is useful in selecting acceptable products based on comparative emission rates. It will

also help identify some of the specific compounds to be measured if laboratory testing is performed.

Identify toxicity or irritation potential of constituent compounds from standard reference sources in the next task. It is important to point out that the toxicity and irritation potential of the components of a product, if known, still do not inform properly about the product itself. The LD_{50} (lethal dose for 50% of test animals) cannot be extrapolated to predict low-dose effects.

U.S. NIOSH's Registry of Toxic Effects of Chemical Substances (RTECS) provides an annotated listing of toxicity and irritation properties for tens of thousands of chemical substances. RTECS is available in hard copy or on-line through Toxnet (U.S. National Library of Medicine). RTECS contains a comprehensive list of alternative trade and generic names by which products may be known or marketed, chemical formulas, and cross-references to the Chemical Abstracts Service number for each chemical. Other useful sources include both printed and electronic databases such as Chemline, Toxline, NIOSHTIC, and The Merck Manual.

Some materials will require laboratory testing. For example, a combination of high volatility and moderate toxicity would dictate further consideration of the substance and the product. A substance of very low volatility and moderate toxicity should be examined in terms of the quantity of the product and the quantity of the substance present in that product.

31.8.4 Emission testing

Test methods include bulk testing and air sampling in an environmental chamber or from "headspace". Headspace testing involves placing a sample in a closed container for a specified period of time, then sampling the air in the "head space" above the sample in the container. Chamber tests are conducted in a very small chamber (usually less than 0.1 m^3) or in a medium-size chamber capable of accommodating full-size samples. Room-size chambers can also be used, but they are expensive and require larger quantities of materials.

Small chamber testing has provided the following information about typical characteristics of VOC emissions from building materials and furnishings.

Emission rates for "wet" materials are initially high, but decrease rather sharply during the first few hours after application. Tests of a variety of sealant products have found large variations in weight loss, and calculated complete drying times. A critical factor in the rate of decrease is the ventilation rate. Maximum available ventilation should be used during and immediately after the application of these materials.

Floors which are waxed on a Friday afternoon will still be emitting significant quantities of several compounds on Monday morning and beyond. This is

true even with ventilation rates greater than normally encountered during weekends in unoccupied (no mechanical ventilation) schools and office buildings.

Product characteristics vary considerably. A product may have very high emissions but dry rather quickly. Another may have low total emissions and dry slowly. Every other combination is also found. Slow-drying compounds or products are the worst from an IAQ perspective unless their emissions are either non-toxic, non-irritating, or negligible. Fast-drying products like the styrene butadiene rubber compound emit significant fractions (1/3–3/5) of their total weight, but they do so in a matter of 3–10 days, mostly in the first 2 or 3 days.

The size of the bead is also variable. Bead size affects the emission rate. Emission processes are a function of evaporation from the surface and diffusion through the material to the surface. Of course, a flat section will have more surface area and less interior volume than a round section. Drying or evaporation will be quickest from the surface and slowest from the centre. The further vapours must travel to reach the surface, the slower the drying time.

31.8.5 Recommendations

1. Information on available test methods for classification of materials/compounds needs to be exchanged between material scientists, producers of building and furnishings materials, constructors, and users. Means for facilitating this exchange should be developed.
2. The goal should be that ventilation is required just for the removal of the physical, chemical and biological emissions from the occupants and their activities and for keeping the indoor humidity sufficiently low. Ventilation for other purposes should become redundant. Emission control of materials, installations, other equipment and consumer products would be the major means of achievement.

There are several research and development areas that need attention to improve our ability to make informed selections of indoor materials to ensure acceptable IAQ. They include:

- improve chemical emissions testing methods, especially to make them simpler and lower-cost;
- improve and validate exposure models, to increase their use and improve confidence in their results;
- develop biological-response-based testing methods; to help improve predictions of acceptability of emissions for a variety of health and comfort endpoints;
- clarify the health and comfort endpoints of concern for protection of IAQ, and clarify the relevant periods of exposure for each of them;

- develop a labelling scheme for all types of users, to enable informed selections of high-quality materials and products that help ensure good IAQ.

31.9 NOISE CONTROL

Mechanical ventilation and air treatment systems produce noise. It is essential to keep this noise at adequately low sound pressure levels. The effective control of noise sources need to be combined with an appropriate acoustical indoor space design.

Any ventilation system should be evaluated with regard to the following aspects and the adequate countermeasures be taken to reduce the noise:

(a) equipment and aerodynamic noise, like fans, compressors etc.;
(b) airborne noise from the outdoor through the ventilation system or equipment, like air inlet and outlet devices etc.,
(c) noise from other spaces which may be transmitted by the ventilation system or equipment, like ductwork etc.

Noise generated and transmitted by the ventilation system is usually broad band noise and often contains a large proportion of low frequency sounds. Sometimes tonal components occur. The presence of signal, information or transient peaks in the noise requires a more stringent sound control than white noise.

Noises tend to interfere with *auditory communication* in which speech is a most important signal. Speech interference level starts from below 50 dB for octave bands centred to the main speech frequencies of 0.5, 1 and 2 kHz, when communication distance grows beyond a few meters.

With respect to interference with speech perception, a majority of the population belong to the sensitive groups. Most sensitive are the elderly and persons with impaired hearing. Even slight hearing impairments in the high-frequency range may cause problems with speech perception in a noisy environment. From 40 years of age and up, people demonstrate impaired interpretation ability of difficult, spoken messages with low linguistic redundancy compared to the ages between 20–30 years.

Speech communication is affected also by the reverberation characteristics of the room. Already reverberation times beyond 1 s can produce loss in speech discrimination. Even in a quiet environment a reverberation time below 0.6 s is desirable for an adequate speech intelligibility for sensitive groups. A long reverberation time combined with background noise makes speech perception still more difficult and staining.

In cases where the correct speech signal reception is of paramount importance, for example, in classrooms or conference rooms, or where listeners with

impaired hearing are involved, for example, in homes for the elderly, lower background levels of noise are desirable. To ensure satisfactory speech communication of the signal-to-noise ratio should always exceed zero dB.

For sensitive groups or when listening to complicated messages (at school or listening to foreign languages) the signal-to-noise ratio should be at least 10 dB. This means that in class rooms, one should strive for as low a background level as possible. Ideally this would mean that with a background level of 35 dB, the message should be ≥ 45 dB.

Sleep disturbance due to continuous as well as intermittent noise has been demonstrated by electrophysiological and behavioral methods. The more powerful the background noise is, the more disturbing is its effect on sleep. Measurable effects start from about 30 dB_{Leq}. Guidelines for noise to avoid sleep disturbance should be expressed in terms of equivalent noise level as well as maximum levels, and number of noise events. It should be noted that the low frequency noise, for example, from ventilation systems can disturb rest and sleep even at low sound levels. Where noise is continuous, the equivalent noise level should not exceed 30 dBA if negative effects on sleep are to be avoided. In the presence of a large proportion of low frequency noise a still lower guideline value is recommended, ca. 23 dBA_{Leq}.

If the noise is not continuous, the peak level is best correlated to sleep disturbances. Effects have been observed at individual exposures of 45 dBA or even less. It is especially important to limit the noise events exceeding 45 dBA where the background level is low. Primarily, efforts should be made to reduce the level of noise peaks and the number of noise events before reducing the equivalent level.

Noise annoyance may be defined as a feeling of displeasure evoked by a noise. The annoyance-inducing capacity of a noise depends upon many of its physical characteristics including its intensity, spectral characteristics, and variations of these with time, in more common terms on whether the noise carries a tone or signal, information or transient peak. However, annoyance reactions are sensitive to many non-acoustic factors of a social and, psychological, nature and there are considerable differences in individual reactions to the same noise.

Annoyance from noise in rooms varies with the activity the person is involved and it is most pronounced in situations pending on adequate verbal communication like conversation, listening to radio or TV. Annoyance responses starts to be experienced around 50 dBA_{Leq}.

Performance of tasks involving motor or monotonous activities is not always degraded by noise. At the other extreme, *mental activities* involving vigilance, information gathering, and analytical processes appear to be particularly sensitive to noise.

A noise measure based only on energy summation expressed as the conventional equivalent measure, Leq, is not enough for the characterization of most noise environments. It is equally important to measure and display the maximum values of the noise fluctuations, preferably combined with a measure of the number of noise events. If the noise includes a large proportion of low frequency or tonal components, still lower values than the recommended guideline values below will be needed. The penalty factor for low frequency noise may be as much as –7dB and for tonal components –5dB.

For *dwellings* the critical effects are sleep disturbance, annoyance and speech interference. Specifically, for bed rooms the critical effect is sleep disturbance. Recommended guideline values for bedrooms are 30 dBA_{Leq} together with 45 dBA_{Lmax}. The maximum level should be measured with the instrument set at "impulse". For living rooms in dwellings, annoyance as well as speech interference are the critical effects. The guideline value is 30 dBA_{Leq} combined with a maximum level of 50 dBA_{max}.

For *schools*, the critical effects are speech interference, disturbance of information extraction, message communication, and annoyance. In class rooms, the maximum noise level should not exceed 25 dBA_{max} during teaching sessions. The sound pressure level should be measured with the instrument set at "fast". The reverberation time in the class room should be considerably less than 0.6 s, for hearing impaired children preferably around 0.3 s.

The noise level demands on rooms in an *office building* are similar to those for schools since the work tasks are similar but less united attention among persons is required. By using modern technology, the guideline value should be 40 dBA_{Leq}, referring to 8 h or the actual time period the equipment is being used combined with a maximum value of 50 dBA_{max}.

31.10 LIGHTING

The health, biological, psychological and behavioral effects of light; the energy implications of lighting design; and, the economics of different lighting schemes all have a bearing on the design and operation of a good indoor climate.

Light "pollution" is the result of many variables interacting. These variables include light and other environmental factors, their connections or interactions with each other and with human beings.

Light is not only a physical agent involved with vision, it is also important psychologically. Vision is not simply a physical or chemico-physical process, but also a psychological one. In addition to its effects on the psychological component of vision, light also affects non-visual aspects of human psychology.

The concentration of fluorescent lamp output in the yellow-green portion of spectrum has biological and health effects that are not adequately understood.

The biological and health effects of light seemingly are modified by other environmental factors including other pollutants and design characteristic. However, research demonstrating the relationship between various health and biological effects of light and the influences of other environmental factors on these effects is sparse.

There is a strong relationship between lighting design and energy consumption patterns in buildings. Since the early seventies there has been increased application of modification lighting and building envelope design to conserve energy in buildings. These efforts have included the following measures which affect lighting quality, thermal conditions and indoor air quality:

1. Reduction of unnecessary or wasteful lighting;
2. Reduction of heat loss (or gain) through the building envelope by reducing window area and thereby reducing natural or daylight illumination of indoor environments;
3. Increased use of daylight to reduce energy use indoors;
4. More efficient use of light sources and more efficient lighting sources;
5. Increased use of south-facing glass for direct solar heating; and
6. Sealed building with no operable windows for ventilation.

A substantial literature on energy conservation is now available to those who design buildings. However, the health and biological effects of environmental lighting are not well understood. The development and introduction of new light sources and applications is proceeding rapidly. Energy costs of lighting have accelerated interest in new sources, and several are becoming commercialized.

There are intensified efforts to reduce energy use in buildings and to improve the quality of light. Prominent among these efforts are renewed interest in daylight sources of illumination; use of task lighting as opposed to ambient lighting; and increased use of fluorescent as well as the newer mercury and sodium vapour electric light sources. Designers and researchers have been concerned with illumination in relationship to visual task performance; glare, contrast, and reflection that affect visibility; energy efficiency of various sources of illumination; and manual and automatic lighting controls, to name a few. It should be noted that some of the discomfort perceived in many problem buildings today include symptoms that partly may be caused by visual difficulty and eye irritation.

In the past decade there has been growing interest in possible health and psychological effects of light. In particular, researchers have examined the effects of the differing spectral distributions of various light sources. A great deal of this work has focused on comparisons of various fluorescent sources,

and the results indicate that are differences in the biological, psychological, and health effects of light sources with varying spectral distributions.

There is currently a growing interest in "full-spectrum lighting" or lighting that approximates the colour (or spectral qualities) of sunlight. Several new products have appeared on the market during the past few years to respond to this growing interest.

As lighting levels are increased to meet new recommended standards, substantial quantities of excess heat may be generated by the fluorescent lights. While they produce less waste heat per watt of electrical input, the much higher levels of illumination, the manner of installation, and the sealed-exterior, larger-volume interiors result in the production and retention of large quantities of waste heat. New ceiling systems are designed to use the waste heat of lighting where heating is required. Where air conditioning is required, this waste heat is problematic.

Thus the waste heat from lighting may required considerable energy use for cooling and ventilation. Efforts to conserve energy in offices and other buildings included renewed use of operable windows to reduce cooling and ventilation energy requirements.

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PART VIII
Guidelines for Indoor Air Quality and Selected International Programmes

This part of the book presents a collection of the available guidelines and standards for indoor air quality adopted at national or international level.

Documents issued by international programmes are also included to provide information on ongoing activities and the policies and strategies recommended at national and international level. After a general discussion about possibilities and limitations of indoor air regulation, the Air Quality Guidelines of the World Health Organization are presented with ample extracts of the original WHO publication. The guidelines proposed at international level for radon, biological particles and volatile organic compounds are separately illustrated in one chapter. The recommendations of the NATO/CCMS Pilot Study on Indoor Air Quality are presented together with a summary of the activities carried out within the framework of this programme. The Nordic Committee on Building Regulations guidelines for indoor climate and air quality are also reported *in extenso*. A summary is then provided of selected indoor air quality programmes, namely the U.S. EPA guidelines and activities, some ASHRAE standards and guidelines, the residential air quality guidelines of Canada, the activities of the European Collaborative Action "Indoor Air Quality and its Impact on Man", the guidelines for IAQ of Norway, and the indoor air quality programme of the WHO Regional Office for Europe. A final chapter deals with the economic implications of indoor air quality and its regulation and control.

This part has been prepared by M. Maroni and R. Axelrad, with contributions from B. Seifert, M. Suess, M. Younes, F. Bochicchio, J. McLaughlin, B. Flannigan, H. Knöppel and F. Levy.

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Chapter 32

Approaches to Regulating Indoor Air[1]

It is the aim of this chapter to discuss the need for developing regulations for indoor air and the possibilities of providing acceptable indoor air quality. A brief consideration concerning the cost of protective measures is intended to complement this discussion.

32.1 THE NEED FOR REGULATIONS

Asking for a reduction in air pollution is a must for anybody concerned with the quality of our environment. Many countries now have regulations which limit emissions of air pollutants into outdoor air and contain ambient air quality standards. Such standards may include short-term and long-term values. Although one would like to have standards based on pure scientific considerations to protect human beings, animals, plants, and materials under all circumstances, actual standards are generally the result of a compromise between scientific knowledge and political will.

Like outdoor air, the air in working environments has been subject to regulations. Limit values have been set by the various bodies responsible for the protection of the working population. Generally, these values are defined as both averages over one shift of several hours and short-term exposure limits.

A first look at the indoor environment reveals that this compartment of potential exposure to air pollutants has equally been subject to certain regulations for many years. In many countries, building codes contain prescriptions which have an influence on the design of indoor spaces. In addition, research related to comfort has led to describing conditions of acceptable indoor climate (Pettenkofer, 1858; Fanger, 1970; McIntyre, 1980). National and international bodies have issued respective prescriptions (ASHRAE, 1981; International Organization for Standardization, 1984). However, the chemical and biological composition of the air of private spaces, schools, offices, transportation systems, etc., has only become a matter of concern since the mid-seventies and

1 This text was presented, with some modifications, by Dr. Bernd Seifert at the 5th International Conference on Indoor Air Quality and Climate, held in Toronto, in 1990.

has generally not been the subject of regulations.

In contrast to the outdoor environment and the work place, the indoor environment is not normally the responsibility of a single administration and no special laws exist in any country dealing with indoor air quality. Although some countries may have general legislation aimed at guaranteeing the individual's right for a healthy environment — and, consequently, also a healthy indoor environment — such general legislation is normally only the background for specific laws which are then assigned to different areas. Examples of such areas are pesticide control, hazardous chemicals, consumer product safety, household appliances safety, energy conservation, etc.

In the absence of clearly defined responsibilities it is likely that regulations are not easily established. The result is that private litigation becomes an important means of protection against damage such as that caused by environmental impact (Ricci et al., 1989). But, not only is such private litigation costly, it also puts the interpretation of scientific findings into the hands of non-scientists, namely judges. Even if a judge calls on experts for assistance, a court can never assemble and evaluate the full body of scientific information, especially if questions are to be answered for which there is no easy agreement among scientists.

Therefore, to exclude any arbitrary character of a court decision which may result from the judgement of a specific expert, i.e. to create a system of legal guarantee, is one of the most important rationales for establishing regulations, especially in the environmental field. With this and the assignment of clear responsibilities in mind the German Federal Minister for the Environment, Nature Conservation and Nuclear Safety has recently made a plea for environmental codes (Töpfer, 1989). It is hoped that such codes would discourage sectorial approaches which are now the general rule in environmental policy. As O'Riordan has pointed out, "anticipatory environmental policy should seek to establish the right mix of regulations and incentives to coordinate planning, fiscal and economical instruments with regulatory measures" (O'Riordan, 1989).

32.2 THE POSSIBILITIES OF ACHIEVING ACCEPTABLE INDOOR AIR QUALITY[1]

Creating an acceptable indoor atmosphere is a process of optimization, the ultimate goal of which should always be human welfare. Although cost, available technology and other parameters will all have to be considered,

1 ASHRAE (1989) gives the following definition of "acceptable indoor air quality": "air in which there are no known contaminants at harmful concentrations as determined by cognizant authorities and with which a substantial majority (80% or more) of the people exposed do not express dissatisfaction".

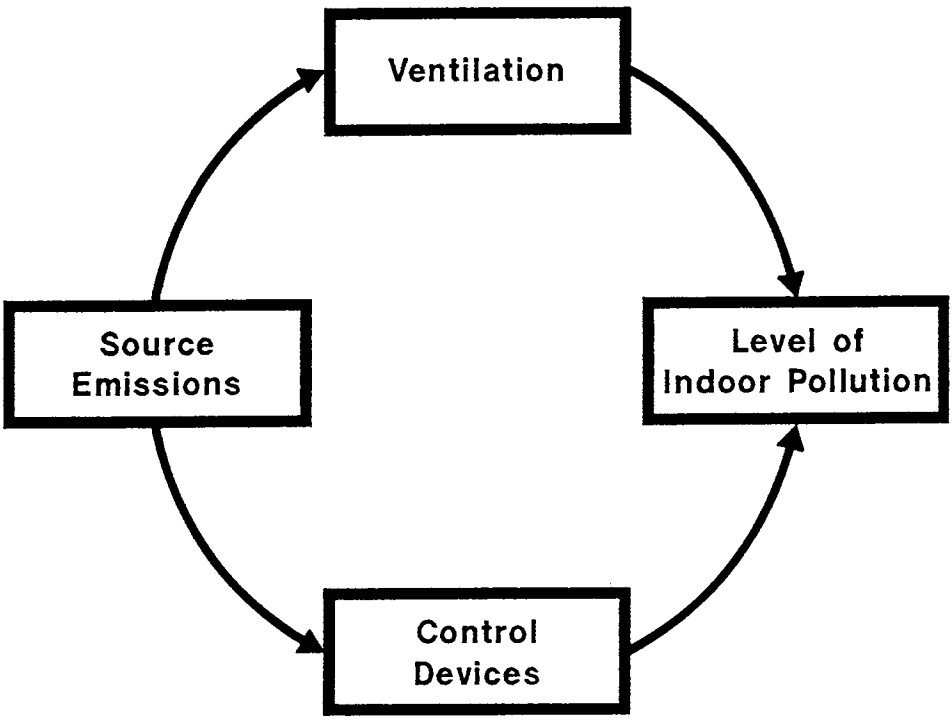

Fig. 32.1: The major elements of the indoor air cycle.

health and comfort are by far the most important aspects to be taken into account in this process. Unfortunately, the susceptibilities to illness and the notion of discomfort vary widely in a given population. As an example: while it cannot be excluded that a non-smoking individual may develop lung cancer from living in a smoky atmosphere, a smoker may attain the age of ninety without any health problem. To date, it is good practice to base regulatory decisions on the needs of the sensitive parts of the population, whenever the nature of the effect and the population groups are defined.

Figure 32.1 depicts four major elements of the indoor air cycle:

- sources are the emitters of pollutants;
- ventilation is one way of removing pollutants;
- control devices are another way of removing pollutants;
- the final level of indoor air pollutants is the result of the interaction of the first three elements.

Clearly, Figure 32.1 is a simplified picture as it does not include elements which, under certain circumstances, may play an important role such as the presence of sinks or the stability of pollutants over time.

In addition to the dilution of polluted indoor air by ventilation, there are

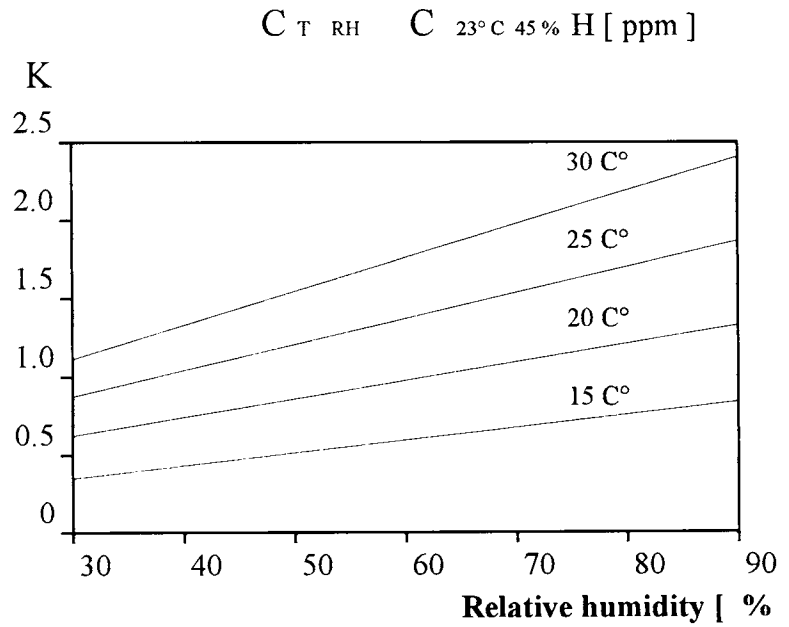

Fig. 32.2: Nomogram linking indoor air formaldehyde concentration, temperature and relative humidity (from Melhorn, 1986). C = concentration; K = correction factor.

other possibilities of providing acceptable indoor air quality, namely by product control. These possibilities range from a complete ban of certain chemicals or products to voluntary agreements on production or usage.

32.2.1 Ventilation standards

In the absence of ventilation and/or cleaning devices, there is a direct link between the emitted amount of a pollutant and its final concentration, which means that this concentration may be unacceptably high if the source strength is high. According to Figure 32.1, to lower the final concentration one can either diminish the source strength (even down to zero level by removing the source completely) or make use of one or both of the other two elements: ventilation, be it natural or forced, is an appropriate tool of reducing concentrations of indoor pollutants as can be the installation of an air cleaning device in a room.

At the 4th International Conference on Indoor Air Quality and Climate, Moschandreas (1987) gave an overview of the possibilities of indoor air pollution control in which he also mentioned dilution of the polluted atmosphere with fresh air as being one of these possibilities. In fact, ventilation has been —

and will continue to be — one major option of reducing pollutant concentrations indoors. However, mechanical ventilation implies the consumption of energy. This simple equation has created the idea of specifying minimum ventilation rates which should provide acceptable indoor air quality (ASHRAE, 1989; International Energy Agency, 1987).

The ASHRAE Standard 62-1989 (ASHRAE, 1989) describes two procedures: a ventilation rate procedure including outdoor air requirements for ventilation and "deemed to provide acceptable indoor air quality, *ipso facto*", and an indoor air quality procedure based on air quality criteria given in an annex which is not part of the standard but "included for information purposes only". As valuable as these efforts are, they leave unanswered two major questions: "Is acceptable good enough?", and "Is ventilation the best way to achieve good indoor air quality?".

32.2.2 Banning chemical substances or products

The most categorical decision that can be taken with regard to an anthropogenic pollutant is to ban it. Generally, one would expect that a complete ban needs a clear definition and/or proof of the detrimental effect of this pollutant. However, in the few cases where substances have been banned from the indoor environment, a definite proof was lacking. Rather, the decision was based on a good deal of evidence and might even have been triggered by public pressure.

Two examples may illustrate the situation: pentachlorophenol (PCP) and tobacco smoke. In the first case, a large field survey carried out in the Federal Republic of Germany in the late seventies (Krause et al., 1980) led to the conclusion that the relationship between five cases of severe illness among the study population and an exposure to PCP could neither be proved nor disproved. Although science has still no final "proof" of the adverse health effect of PCP at current exposure levels, there was enough evidence for such effects to decide to ban the production, the circulation and the use of PCP (Anonymous, 1989).

In a similar way, the evidence of the unhealthy properties of environmental tobacco smoke (ETS) and the results of the major part of the known epidemiological studies on passive smoking have led to ban smoking in many places, although no individual case of lung cancer has until now been and will most probably ever be, traced back to the influence of ETS exposure. There can be no doubt that prevention is the driving force in the development of regulations creating smoke-free atmospheres.

The difficulty that generally comes along with the ban of a substance or product is the lack of sufficient knowledge of the properties of the substitution product. Hence, most governments are very careful in using this regulatory tool.

32.2.3 Standards for indoor air quality

As has been pointed out previously, many countries have set standards for outdoor (ambient) air pollutants. However, there are no worldwide standards for indoor air pollutants.

Although there can be no doubt that standards contribute to establishing legal guaranties, two major criticisms can be levelled against the setting of standards for chemicals (and microorganisms) in indoor air:

- the existence of a standard favours the impression of having a limit below which there is no reason for concern;
- the enforcement of a standard is virtually impossible due to the large number of closed spaces.

A further difficulty results from the large variety of conditions encountered inside buildings. A room may fulfil the requirements of a standard at 20°C but not at 23°C. However, both temperatures being within the accepted range of thermal comfort, an individual cannot be obliged to live at 20°C simply because at this temperature the standard — if defined for 20°C — would be respected.

This situation is not as hypothetical as it may appear. The case of formaldehyde may be used for illustration. The nomogram depicted in Figure 32.2 shows that the formaldehyde concentration in the air of a room depends critically on temperature and relative humidity. According to the graph an increase or decrease of the room temperature of only 1°C changes the formaldehyde concentration by roughly 10%.

Due to the absence of standardized ventilation requirements for naturally ventilated buildings, which show a wide variety of ventilation rates, similar difficulties would arise in practice if the standard were defined for a specific ventilation rate.

For air conditioned buildings, there are requirements fixing the amount of outdoor air to be provided for a room (e.g., ASHRAE, 1989; Deutsches Institut für Normung, 1994). In deriving these requirements the number of persons for which the room has been conceived is generally taken into account. It is obvious that in practice the occupancy will not always correspond to this number.

32.2.4 Guideline values for indoor air quality

As the term "guideline value" indicates, such a value should provide guidance. Consequently, guideline values are much weaker than standards and have a much less binding character. They are also more likely to be agreed upon by different countries because — in contrast to standards — they generally will not take into account socio-economic or political aspects.

As has been pointed out by the World Health Organization (WHO, 1987), "inhalation of an air pollutant in concentrations and for exposure times below a guideline value will not have adverse effects on health". However, since by nature, the guideline value does not define a sharp borderline and generally does not take synergisms into account, compliance with these values "does not guarantee the absolute exclusion of effects at levels below such values".

Consequently, under some circumstances a strategy that includes two values may be adopted. While the first value would define the level of hazard, the second would indicate a target concentration for the future. In the Federal Republic of Germany, this strategy has recently been applied for tetrachloroethene in the air of rooms next to dry-cleaning shops (Anon., 1988). From toxicological considerations, a value of 5 mg/m^3 was derived below which no immediate action, such as closing the dry-cleaning shop or evacuating nearby flats, was considered to be necessary. However, preventive control measures were recommended to lower the concentration of tetrachloroethene down to a target value of 0.1 mg/m^3.

In a similar way, some countries have published sets of guideline values for average radon concentrations inside buildings differentiating between concentrations at which immediate remedial actions should be taken and concentrations for preventive purposes, i.e. for future housing. Radon gas concentrations of 400 and 200 Bq/m^3 have been recommended in Europe for existing and future housing, respectively (COST 613, 1988). The fact that in the United Kingdom, the upper bound level for radon gas in future housing has been set to only 100 Bq/m^3 proves that the target to be achieved in the future may vary from one country to the next.

Chapter 33 "The World Health Organization Air Quality Guidelines" discusses the WHO Guidelines for air quality that are applicable both to outdoor and indoor air. Other values recommended at national level are discussed in Chapters 36 and 37.

32.2.5 Developing emission standards

There is no doubt that the best way to achieve good indoor air quality is source control. However, unless a compound is not present at all in a product, the acceptable product emission has to be defined. One way to do this is to start from a tolerable indoor concentration level, e.g., a guideline value (see preceding section). With a number of assumptions regarding the average conditions under which the product is used in practice (temperature, relative humidity, air exchange rate, loading factor, etc.) the desired emission rate of a compound can be calculated.

Such a procedure is more easily described than achieved in practice because more than one product may emit the compound under consideration. However, assuming that the apportionment, though difficult, may be possible for continuously emitting surface materials, a simple equation to approximate the emission factor E_i (mg m^{-2} h^{-1}) for a compound i in such a material is:

$$E_i = (c_i \cdot n)/L$$

with c_i = concentration of compound i in the air (mg/m^3); n = air exchange rate (h^{-1}); L = loading factor (m^2/m^3)

More complex equations are needed to take into account the variation of the emission factor with time, the effects of removal processes other than ventilation, the temperature, the relative humidity, etc. Formaldehyde has been mentioned earlier as an example of the important influence of the last two parameters. Many difficulties still need to be solved in emission rate testing. As an example: the approach described briefly for surface materials cannot be used for intermittent sources.

Generally, emission factors are obtained from test chamber measurements. Recent guides published in Europe (COST 613, 1989) and in the USA (American Society for Testing Materials, 1989) show precisely that boundary conditions for such tests have to be described very precisely and maintained carefully during the experiment to obtain comparable data.

Despite all difficulties, to date, source control seems to be the most appropriate way of reducing indoor air pollution. As a first practical step towards this goal, the Council of the European Communities has issued a Council Directive on construction products (European Communities, 1989). The Directive, among other requirements, states that

> "construction work must be designed and built in such a way that it will not be a threat to the hygiene or health of the occupants or neighbours, in particular as a result of any of the following:
> - the giving-off of toxic gas,
> - presence of dangerous particles or gases in the air,
> - the emission of dangerous radiation,
> - pollution or poisoning of the water or soil,
> - faulty elimination of waste water, smoke, solid or liquid wastes,
> - the presence of damp in parts of the works or on surfaces within the works."

The Directive does not specify details but leaves the implementation of these requirements to the European standards organisations which have to

establish harmonised standards for products. Much work remains to be done to make such standards available for the needs of the now existing Single European Market.

32.2.6 Voluntary agreements

It is a fortunate fact that the concentration levels of many pollutants are below what may cause immediate adverse health effects. On the other hand, the available knowledge of the effect of chronic exposure of humans to contaminants at or below no observed effect levels derived from animal studies is, and will probably remain to be, inadequate. In such situation, a further decrease of concentration levels with the idea of prevention in mind can only be based on consensus reached by all parties of a democratic society.

To achieve such consensus, the dissemination of information beyond scientific circles is urgently needed. The difficulties that scientists and governmental institutions are facing in communicating environmental risk to the public are well recognized (Renn, 1989; Covello et al., 1987; Covello, 1989) and faulty behaviour of officials and industry have certainly contributed to having created these difficulties in the past. As has been pointed out recently (Fülgraff, 1989), "scientists tend to neglect that they may be experts for risk assessment, but not at all experts for acceptability, adequacy, reasonability, justifiability, etc."

Special incentives may induce a kind of self-regulation of the market. Good results have been obtained in the Federal Republic of Germany with an environmental label, the so-called "Blue Angel" which can be attributed to products representing a lower burden to the environment than others. As things stand today, an educated public would prefer products for indoor use if an official label indicated that these products do not contain, e.g., known human carcinogens and allergens.

32.3 THE COST OF REGULATION

Although the financial aspect of regulation may be considered a matter of its own right beyond the scope of this text, some remarks with regard to costs seem to be appropriate. As Ashford (1981) has pointed out, the prediction of the cost of regulation is generally very imprecise since regulating agencies need to rely on data from industry to calculate them. Furthermore, the ability of regulated industries to produce more cost-effectively after a period of adaptation is generally not taken into account in calculating such costs.

It is often argued that the costs linked to a regulation or provision cannot be borne by industry or society. However, on a long-term basis, the decision to

lower the emission of pollutants has often a positive effect on the industrial development. The following is one example out of many. When in the early eighties, the forest decline in Europe initiated governments to lower the emissions of sulphur dioxide, it was estimated that the reduction rates foreseen by the West German government would cost many billions of marks. The critics of the abatement measures argued that this money was impossible to be risen by German industry. Today, we know that the regulation has not only substantially contributed to lowering SO_2 levels in ambient air, but also to guaranteeing West-German industry a world-leading position in the flue gas desulphurisation business.

In a similar way, regulations prohibiting the use of materials that are widely used indoors may stimulate new technological developments. However, any substitute needs very careful testing before being released to the market on a larger scale.

One very important aspect of cost-benefit calculations which is especially important for considerations related to the indoor environment is that costs on the one hand and benefits on the other are often not assigned to the same parties. As an example, the lessor of a building who does not maintain or clean the ventilation system of his building makes a benefit as he saves money, whereas it is the lessee and the social security who pay for the costs of office workers' sick-leave if the building turns out to be a sick building as a result of the lessor's lack of care. The result is doubly negative: the occupants of the building will suffer from injuries to their health and society will have to cover costs which by far exceed the lessor's benefit.

One drawback of cost-benefit analysis in health matters is that the benefits of a regulation as opposed to its costs are not easily sorted out although many attempts have been made to give numerical estimates of illness or even death. However, agreement on the value of health and well-being or comfort is not easily reached. The now widely used indicator, the loss in productivity, can only be a partial surrogate in such calculations as it is only applicable to office spaces and not to private rooms (on this subject, see also Chapter 38).

32.4 CONCLUSIONS

Although setting air quality standards has been instrumental in reducing the pollution of outdoor air, the special character of the indoor environment makes it unlikely that the same kind of regulation would be an adequate way of fighting indoor air pollution. Rather than standards, guideline values should be developed. These guideline values may be set two at a time. Whereas one value would indicate a toxicologically derived action level applicable to

cases of already existing pollution, the other would define a target concentration to be reached in the future. This strategy would both permit to protect the public from acute danger and promote the idea of prevention. Furthermore, such guideline values, together with appropriate modelling, could serve in the process of limiting the emissions of products, especially surface materials.

A number of questions are still open which cannot be answered in a generally valid form because judicial and administrative structures vary widely from one country to the next. Nevertheless, it is hoped that this discussion contributes to the understanding of all parties involved that regulating indoor air is a complex process in which setting air quality standards appears to be the least appropriate tool. As long as science has not found definite answers to the many open questions, societal consensus is needed to make the best use of the available financial resources.

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Chapter 33

The World Health Organization Air Quality Guidelines[1]

33.1 CRITERIA USED IN ESTABLISHING GUIDELINE VALUES

Relevant information on the pollutants was carefully considered during the process of establishing guideline values. Ideally, guideline values should represent concentrations of chemical compounds in air that would not pose any hazard to the human population. However, the realistic assessment of human health hazards necessitates a distinction between absolute safety and acceptable risk. To aim at achieving absolute safety, one would need a detailed knowledge of dose–response relationships in individuals in relation to all sources of exposure, the types of toxic effects elicited by specific pollutants or their mixtures, the existence or nonexistence of "thresholds" for specified toxic effect, the significance of interactions and the variation in sensitivity and exposure levels within the human population.

However, such comprehensive and conclusive data on environmental contaminants are not always available. Very often the relevant data are scarce and the quantitative relationships uncertain, scientific judgement and consensus therefore play an important role in establishing acceptable levels of population exposure. Value judgements are unavoidable, because terms such as "adverse" and "sufficient evidence" are not in themselves totally objective, their meaning being based on generally agreed judgements.

Although it may be accepted that a certain risk can be tolerated or is simply unavoidable, the risk within a population may not be equally distributed. There may be subpopulations which are at considerably higher risk from the same exposure. Therefore, groups at special risk in the general population must be taken specifically into account in the risk management process. Even if knowledge about groups with specific sensitivity is available, unknown factors may exist that change the risk in an unpredictable manner.

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1 The text of this chapter has been extracted from the volume "Air Quality Guidelines for Europe", published by the World Health Organization, Regional Office for Europe, 1987.

33.1.1 Criteria common to carcinogens and non-carcinogens

Sources, levels and routes of exposure

Available data are provided on the current levels of human exposure to pollutants from all sources, including the air. Special attention is given to atmospheric concentrations in urban and in non-polluted rural areas and in the indoor environment. Where appropriate, concentrations in the workplace are also indicated for comparison with environmental levels. To provide information on the contribution from air in relation to all other sources, data on uptake by inhalation, ingestion from water and food, and dermal contact are given where relevant. However, for most chemicals, data on total human exposure are lacking to some extent.

Kinetics and metabolism

Available data on the toxicokinetics of distribution in humans and experimental animals are indicated for inter- and intra-species extrapolation, especially to assess the magnitude of body-burden from long-term, low-level exposures and to characterize better the mode of toxic action. Data concerning the distribution of an agent in the body are important in determining the molecular or tissue dose to target organs. High-to-low-dose and inter-species extrapolations are more easily carried out using equivalent tissue doses. Metabolites are mentioned, particularly if they are known or believed to exert a greater toxic potential than the original agent. Additional data of interest include the rate of excretion and the biological half-life.

33.1.2 Criteria for endpoints other than carcinogenicity

For those compounds reportedly without carcinogenic effects (or for which data on carcinogenicity were lacking or insufficient), the starting-point for the derivation of guideline values was to define the lowest concentration at which effects are observed in humans, animals and plants. In doing so, an attempt was made to define a lowest-observed-adverse-effect level. The question whether the lowest-observed-effect level or the no-observed-effect level should be used instead is mainly a matter of availability of data. If a series of data fixes the lowest-observed-effect level and the no-observed-effect level, either of those levels might be used. The gap between the lowest-observed-effect level and the no-observed-effect level is among the factors included in judgements concerning the appropriate margin of protection. However, a single, free-standing no-observed-effect level which is not defined in reference to a lowest-

observed-effect level or a lowest-observed-adverse-effect level is not conclusive. Opinions on this subject differ, but the working consensus was that the level of concern in terms of human health is more relatable to the lowest-observed-adverse-effect level; this level was therefore used whenever possible. In the case of irritant and sensory effects on humans, it is desirable where possible to determine the no-observed-effect level.

On the basis of the evidence concerning adverse effects, judgements about the protection factors (safety or uncertainty factors) needed to minimize health risks were made. Averaging times were included, since the time of exposure is critical in determining toxicity. Criteria applied to each of these key factors are described below.

33.1.3 Criteria for selection of a lowest-observed-adverse-effect level

The distinction between adverse and non adverse effects poses considerable difficulties (WHO, 1978). Any observable biological change may be considered an adverse effect under certain circumstances. The definition of an adverse effect has been given as "any effect resulting in functional impairment and/or pathological lesions that may affect the performance of the whole organism or which contributes to a reduced ability to respond to an additional challenge" (U.S. EPA, 1980). Even with such a definition, a significant degree of subjectivity and uncertainty remains. Ambient levels of major air pollutants frequently cause subtle effects that are typically detected only by sensitive methods. This makes it exceedingly difficult, if not impossible, to achieve a road consensus as to which effects are adverse. To resolve this difficulty, data should be ranked in three categories.

1. Observations, even of potential health concern, which are single endings that have not been verified by other groups. Because of the lack of verification by other investigators, such data could not readily be used as a basis for guideline values. They do, however, indicate the need for further research and may be considered in evaluating a margin of protection.
2. A lowest-observed-effect level: such a level is represented by data which have been supported by other scientific information. When the results are in a direction that might result in pathological change, there is a higher degree of health concern. Scientific judgement based on all available health information is used to determine how effects in this category can be used in determining the pollutant level that is to be avoided so that excessive risk can be prevented.
3. A substantial change in the direction of pathological effects: these endings have had a major influence on guideline considerations.

33.1.4 Criteria for selection of protection factors

In previous evaluations by WHO, protection factors, usually called safety factors, have been applied to derive guidelines from accepted criteria for adverse effects on health (WHO, 1984; Vettorazzi, 1980). The rationale has been that such a factor allows for a variety of uncertainties, for example, about possibly undetected effects on particularly sensitive members of the population, synergistic effects of multiple exposures, and the adequacy of existing data. Traditionally, the safety factor has been used to allow for uncertainties in extrapolation from animals to humans and from a small group of individuals to a large population (WHO, 1978).

In these guidelines, the terms "protection factor" and "margin of protection" have been used in preference to "safety factor" or "margin of safety" because the word "safety" may convey to the public the impression of absolute freedom from risk; this goes beyond what is intended by scientists when they refer to safety factors. These factors are applied in guidelines for the protection of human health. They are not applied to ecological guidelines, because they already include a kind of protection factor with regard to the variety of species covered.

A wide range of factors for protecting human health is used in this book, based on scientific judgements concerning the interplay of various criteria. The decision process for developing protection factors has been complex, involving the transformation of mainly non quantitative information into a single number expressing the judgement of a group of scientists.

Some of the factors which are taken into account in deciding the margin of protection can be grouped under the heading of scientific uncertainty. Uncertainty occurs because of limitations in the extent or quality of the data base. One can confidently set a lower margin of protection (i.e. use of a smaller number), when a large number of high quality, mutually supportive scientific experiments in different laboratories using different approaches clearly demonstrate the dose–response, including a lowest-observed-effect level and a no-observed-effect level. In reality, difficulties inherent in studying air pollutants and the failure to perform much needed and very specific research usually preclude this situation.

Where a protection factor was adopted in the air quality guidelines, the reasoning behind this factor is given in the scientific background information. As previously mentioned, exceeding a guideline value with an incorporated protection factor does not necessarily mean that adverse effects will result. However, the risk to public health will increase, particularly in situations where the most sensitive population group is exposed to several pollutants simultaneously. It is therefore necessary to exercise some kind of judgement regarding the size of the protection factor.

Groups within a population respond differently to pollutants (WHO, 1983). Individuals with pre-existing lung disease, for instance, can be at higher risk from exposure to air pollutant than healthy people. Differences in response can be due to factors other than pre-existing health factors, such as age, sex level of exercise taken or to unknown factors. Thus, the population must be considered very heterogeneous in respect of response to air pollutants. Existing information does not allow adequate assessment of the proportion of the population that has enhanced response. However, an estimate of even a few per cent of the total population entails a large number of people.

Effects observed in laboratory animals in the absence of human studies generally require a larger protection factor, because humans may be more susceptible than laboratory animal species. Negative data from human studies will tend to reduce the magnitude of the protection factor. Also of importance are the nature and reversibility of the reported effect. A pollutant level producing slight alterations in physiological parameters requires a smaller protection factor than a pollutant level producing a clearly adverse effect. Scientific judgement about protection factors will also take into account the toxicology of pollutants, including the type of metabolites formed, variability in metabolism or response in humans suggesting hypersusceptible groups, and the likelihood that the compound or its metabolites will accumulate in the body. It is also important to consider the exposure level used in health studies and to make appropriate conversions to environmental situations.

It is obvious, therefore, that diverse factors must be taken into account in proposing a margin of protection. The protection factor cannot be assigned by a simple mathematical formula; it requires experience, wisdom and judgement.

33.1.5 Criteria for selection of averaging times

The development of toxicity is a complex function of the interaction between concentration and time of exposure. A chemical may cause acute, minor, reversible effects after brief exposure and irreversible or incapacitating effects after prolonged exposure. Our knowledge is usually insufficient to delineate these concentration-time interrelationships. Therefore, expert judgement must be applied, based on the weight of the evidence available (WHO Technical Report, 1972). Generally, when short-term exposures lead to adverse effects, short-term averaging times are recommended. The use of a long-term average under such conditions would be misleading, since the typical pattern of repeated peak exposures is averaged over time and the risk manager has difficulty in deciding upon effective strategies. In other cases, exposure–response knowledge is sufficient to recommend a long-term average. This frequently occurs for chemicals that accumulate in the body overtime, thereby

resulting in adverse effects. In such cases, the integral of exposure can have more impact than the pattern of peak exposure.

It should be noted that these averaging times are based on effects. Therefore, if the guidelines are used as a basis for regulation, the regulator needs to select the most appropriate and practical standards in relation to the guidelines, without necessarily using the guidelines directly.

33.1.6 Criteria for consideration of sensory effects

Some of the substances selected for evaluation have malodorous properties at concentrations far below those at which toxic effects occur. Although odour annoyance cannot be regarded as an adverse health effect in a strict sense, it affects the quality of life (WHO, 1979). Therefore, odour threshold levels for such chemicals have been indicated where relevant and used as a basis for separate guideline values.

For practical purposes, the following aspects and respective levels were considered in the evaluation of sensory effects:

a) intensity, where the detection threshold level is defined as the lower limit of the perceived intensity range (by convention the lowest concentration that can be detected in 50% of the cases in which it is present);
b) quality, where the recognition threshold level is defined as the lowest concentration at which the sensory effect, e.g. odour, can be recognized correctly in 50% of the cases;
c) acceptability and annoyance, where the nuisance threshold level is defined as the concentration at which not more than a small proportion of the population (less than 5%) experiences annoyance for a small part of the time (less than 2%); since annoyance will be influenced by a number of psychological and socioeconomic factors, a nuisance threshold level cannot be defined on the basis of concentration alone.

33.1.7 Criteria for carcinogenic endpoint

Cancer risk assessment is basically a two-step procedure, involving a qualitative assessment of how likely it is that an agent is a human carcinogen, and a quantitative assessment of the cancer rate the agent is likely to cause at given levels and durations of exposure (Peakall et al., 1985).

Qualitative assessment of carcinogenicity

The decision to consider a substance as a carcinogen is based on the qualitative evaluation of all available information on carcinogenicity, ensuring

that the association is unlikely to be due to change alone. Here the classification criteria of the International Agency for Research on Cancer have been applied (IARC, 1982). These classify chemicals for carcinogenicity in the following way.

Group 1 — Proven human carcinogens. This category includes chemicals or groups of chemicals for which there is sufficient evidence from epidemiological studies to support a causal association between the exposure and cancer.

Group 2 — Probable human carcinogens. This category includes chemicals and group of chemicals for which, at one extreme, the evidence of human carcinogenicity is almost sufficient, and those for which, at the other extreme, it is inadequate. To reflect this range, the category is divided into two subgroups according to higher (Group 2A) and lower (Group 2B) degrees of evidence.

Group 2A — This group is usually used for chemicals for which there is at least limited evidence of carcinogenicity in humans and sufficient evidence for carcinogenicity in animals.

Group 2B — This group is usually used for chemicals for which there is inadequate evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals. In some cases the known chemical properties of a compound and the results of short-term tests have allowed its transfer from Group 3 to Group 2B, or from Group 2B to Group 2A.

Group 3 — Unclassified chemicals. This group includes chemicals or groups of chemicals which cannot be classified as to their carcinogenicity in humans.

With regard to this classification scheme, it is likely that in the future some chemicals in Group 3 can be classified as non-carcinogenic.

It was concluded that the qualitative evaluation applied by IARC should serve as the baseline in establishing the air quality guidelines for carcinogens, and that the IARC categorization scheme should be used if no new divergent evidence was available. In this respect, it was decided that all chemicals categorized in Groups 1 and 2A, i.e. proven human carcinogens and carcinogens with at least limited evidence of human carcinogenicity, should be treated as human carcinogens, and guidelines should be formulated accordingly, indicating only risk estimates. For chemicals classified in Group 2B (inadequate evidence in humans, sufficient evidence in animals) it was decided that, until new evidence appeared, guidelines would point out the carcinogenic effects in laboratory animals and cite risk estimates (if such estimates could be reasonably obtained) in the health risk evaluation part of the scientific background information. However, these risk estimates, based on animal data only, would not be incorporated in the guideline recommendations because of various uncertainties in this connection. Guideline values based on non-carcinogenic endpoints would be given for these pollutants.

Quantitative assessment of carcinogenic potency

The aim of risk assessment is to apply information available from very specific study situations (mainly occupational studies) to the general population in order to calculate the possible risk to the letter. Therefore, quantitative risk assessment or, more specifically, dose–response assessment generally includes the extrapolation of risk from relatively high dose levels (characteristic of animal experiments or occupational exposures), where cancer responses can be measured, to relatively low dose levels, which are of concern in environmental protection and where such risks are too small to be measured directly, either in animal studies or in epidemiological studies (WHO, 1981).

The choice of the extrapolation model depends on the current understanding of the mechanisms of carcinogenesis (Anderson, 1985). No single mathematical procedure can be regarded as fully appropriate for low dose extrapolation. Methods based on a linear, non-threshold assumption have been used at the international level (WHO, 1981; IARC, 1982) and the national level (various health assessment documents produced by the US Environmental Protection Agency (EPA) and the National Institute of Public Health, Netherlands) more frequently than models which assume a safe or virtually safe threshold.

In these guidelines the risk associated with lifetime exposure to a certain concentration of a carcinogen in the air has generally been estimated by linear extrapolation and the carcinogenic potency expressed as the incremental unit risk estimate. The incremental unit risk estimate for an air pollutant is defined as "the additional lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed continuously from birth throughout their lifetimes to a concentration of 1 $\mu g/m^3$ of the agent in the air they breathe" (U.S. EPA, 1985).

Calculations expressed in unit risk estimates provide the opportunity to compare the carcinogenic potency of different agents and can help to set priorities in pollution control according to the existing exposure situation. By using unit risk estimates, any reference to the "acceptability" of risk is avoided. The decision on the acceptability of a risk should be made by national authorities in the framework of risk management.

For those substances for which appropriate human studies are available, the method known as the "average relative risk model" (U.S. EPA, 1983) has been generally used and is therefore described in more detail below.

For animal cancer bioassays several methods have been used to estimate the incremental risks. Two general approaches have been proposed. A strictly linearized estimate has generally been used by U.S. EPA (Anderson, 1985). Nonlinear relations have been proposed by others where either the concentra-

tion–tumour response was found experimentally or where metabolism is of limited capacity. Accordingly, risk estimates based on animal bioassays are considered separately.

Quantitative assessment of carcinogenicity based on human data

The quantitative assessment using the average relative risk model includes four steps: (a) selection of studies; (b) standardized description of study results in terms of relative risk, exposure level and duration of exposure; (c) extrapolation towards zero dose; and (d) application to a general (hypothetical) population.

First, a reliable human study must be identified, where the exposure of the study population can be estimated and the excess of cancer incidence is statistically significant. If several studies exist, the best representative study should be selected or several risk estimates evaluated.

When a study is identified, the relative risk (R) as a measure of response must be calculated. It is important to note that the 95% confidence limits around the central value for the relative risk can be wide and should specifically stated and evaluated. The relative risk is then introduced in the following formula (average relative risk model) which combines steps (c) and (d) and allows the unit lifetime risk (UR) (i.e. risk associated with a lifetime exposure to 1 $\mu g/m^3$) to be calculated:

$$UR = \frac{P_0(R - 1)}{X}$$

where P_0 = background lifetime risk (this is taken from age/cause-specific death or incidence rates found in national vital statistics tables using the life table methodology, or it is available from a matched control population); R = relative risk, being the ratio between the observed (O) and expected (E) number of cancer cases in the exposed population (the relative risk is sometimes expressed as the standardized mortality ratio SMR = (O/E) × 100); X = lifetime average exposure (standardized lifetime exposure for the study population on a lifetime continuous exposure basis); in the case of occupational studies, X represents a conversion from the occupational 8-h, 240-day exposure over a specific number of working years and can be calculated as X = 8-h TWA × 8/24 × 240/365 × (average exposure duration [in years])/(life expectancy [70 years]), where TWA is the time-weighted average ($\mu g/m^3$).

It should be noted that the unit lifetime risk depends on P_0 (background lifetime risk), which is determined from national age-specific cancer incidence or mortality rates. Since these rates are also determined by exposures other

than the one of interest and may vary from country to country, it follows that the UR may also vary from one country to another.

Necessary assumptions for average relative risk method

Before any attempt is made to assess the risk in the general population, numerous assumptions are needed at each phase of the risk assessment process to fill in various gaps in the underlying scientific data base. Therefore, as a first step in any given risk assessment, an attempt should be made to identify the major assumptions that have to be made, indicating their probable consequences. These assumptions are as follows.

1. The response (measured as relative risk) is some function of cumulative dose or exposure.
2. There is no threshold dose for carcinogens. Many stages in the basic mechanism of carcinogenesis are not yet known or are only partly understood. However, taking available scientific findings into consideration, several scientific bodies (WHO, 1981, 1984; IARC, 1982; National Research Council, 1977; U.S. Department of Health and Human Services, 1985) have concluded that there is no scientific basis for assuming a threshold or no-effect level for chemical carcinogens. This view is based on the fact that most agents that cause cancer also cause irreversible damage to deoxyribonucleic acid (DNA). The assumption applies for all non-threshold models.
3. The linear extrapolation of the dose–response curve towards zero gives an upper-bound conservative estimate of the true risk function if the unknown (true) dose–response curve has a sigmoidal shape. The scientific justification for the use of a linear non threshold extrapolation model stems from several sources: the similarity between carcinogenesis and mutagenesis as processes which both have DNA as target molecules; the strong evidence of the linearity of dose–response relationships for mutagenesis; the evidence for the linearity of the DNA binding of chemical carcinogens in the liver and skin; the evidence for the linearity in the dose–response relationship in the initiation stage of the mouse 2-stage tumorigenesis model; and the rough consistency with the linearity of the dose–response relationships for several epidemiological studies (WHO, 1981). This assumption applies for all linear models.
4. There is constancy of the relative risk in the specific study situation. In a strict sense, constancy of the relative risk means that the background age/cause-specific rate at any time is increased by a constant factor. The advantage of the average relative risk method is that this needs to be true only for the average.

Advantages of the method

The average relative risk method was selected in preference to many other more sophisticated extrapolation models because it has several advantages, the main one being that is seems to be appropriate for a fairly large class of different carcinogens, as well as for different human studies. This is possible because averaging doses, i.e. averaging done over concentration and duration of exposure, give a reasonable measure of exposure when dose rates are not constant in time. This may be illustrated by the fact that the use of more sophisticated models (US EPA, 1983, 1984, 1985) results in risk estimates very similar to those obtained by the average relative risk method.

Another advantage of the method is that the carcinogenic potency can be calculated when estimates of average level and duration of exposure are the only known parameters besides the relative risk. Furthermore, the method has the advantage of being simple to apply, allowing non-experts in the field of risk models to calculate a lifetime risk from exposure to the carcinogens.

Limitations of the method

As pointed out earlier, the average relative risk method is based on several assumptions which appear to be valid in a wide variety of situations. However, there are specific situations in which the method cannot be recommended, mainly because the assumptions do not hold true.

The cumulative dose concept, for instance, is inappropriate when the mechanism of the carcinogens suggests that it cannot produce cancer throughout all stages of the cancer development process. Also, specific toxicokinetic properties, such as higher excretion rate of a carcinogen at higher doses or a relatively lower production rate of carcinogenic metabolites at lower doses, may diminish the usefulness of the method in estimating cancer risk. Furthermore, supra linearity of the dose–response curve or irregular variations in the relative risk over time which cannot be eliminated would reduce the value of the model. However, evidence concerning these limitations either does not exist or is still too preliminary to make the average relative risk method inappropriate for carcinogens evaluated here.

A factor of uncertainty, rather than of methodological limitations, is that data on past exposure are nearly always incomplete (IARC, 1982; US Department of Health and Human Services, 1985). Although it is generally assumed that in the majority of studies the historical dose rate can be determined within an order of magnitude, there are possibly greater uncertainties, even of more than two orders of magnitude, in some studies. In the risk assessment process it is of crucial importance that this degree of uncertainty be clearly

stated. This is often done simply by citing upper and lower limits of risk estimates (IARC, 1982). Duration of exposure and the age-and-time-dependence of cancer caused by a particular substance are less uncertain parameters, although the mechanisms of relationship are not so well understood (Peakall, 1985).

Risk estimates from animal cancer bioassays

Animal bioassays of chemicals provide important information on the human risk of cancer from exposure to chemicals. These data enhance our confidence in assessing human cancer risk on the basis of epidemiological data.

Several chemicals considered in this volume have been studied using animal cancer bioassays. The process is continuing and new information on the potential carcinogenicity of chemicals is rapidly appearing. Consequently, the status of chemicals is constantly being reassessed.

During the preparation of this book, dichloromethane was classified by IARC as showing sufficient evidence of carcinogenicity in animals, on the basis of studies in rats and mice (IARC, 1986). Detailed studies of the kinetics of metabolism of dichloromethane have also recently been completed, indicating that the capacity of mammals to metabolize dichloromethane is limited. Thus, the extent of metabolism of dichloromethane at high doses where cancer bioassays are conducted is less than at levels of environmental exposure. The linearized cancer risk models may therefore represent considerable overestimates of the carcinogenic potential of dichloromethane in humans at levels likely to occur in the environment. As with dichloromethane, there are also considerable uncertainties in establishing human risk estimates derived from animal data for formaldehyde and 1,2-dichloromethane. Therefore, the significance of such estimates is still very problematic.

There is little doubt of the importance of animal bioassay data in reaching an informed decision on a chemical. The collection and use of data such as those on saturation mechanism, absorption, deposition and metabolic pathways, as well as on interaction with other chemicals, are important and should be continued. Regrettably, these data were not available for the above-mentioned chemicals at the time of this evaluation of guidelines for air pollutants. The process of evaluating guidelines and the impact of human exposure to these chemicals should continue and be revised as new information becomes available.

Interpretation of risk estimates

The risk estimates presented in this book should not be regarded as being equivalent to the true cancer risk. Quantitative risk estimates can provide

policy-makers with rough estimates of risk which may serve well as a basis for setting priorities, balancing risks and benefits, and establishing the degree of urgency of public health problems among subpopulations inadvertently exposed to carcinogens (Anderson, 1985).

33.1.8 Ecological effects

The importance of taking an integrated view of both health and ecological effects in air quality management was recognized from the beginning of the project. Ecological effects may have a significant indirect influence on human health and well-being. For example, most of the major urban air pollutants are known to have adverse effects at low levels on plants, including food crops. A consultation group was therefore convened to consider ecological effects of sulphur oxides, nitrogen oxides and ozone/photochemical oxidants on terrestrial vegetation. These substances are important both because of the high anthropogenic amounts produced and because of their wide distribution. They deserve special attention because of significant adverse effects on ecological systems in concentrations far below those known to be harmful to humans.

The pollutants selected for consideration here form only part of the vast range of air pollutants that have ecological effects. The project timetable permitted only an evaluation of adverse effects on terrestrial plant life, although effects on animal and aquatic ecosystems are also of great concern in parts of Europe. Nevertheless, even this limited evaluation clearly indicates the importance attached to the ecological effects of such pollutants in the European Region.

33.2 SUMMARY OF THE GUIDELINES

The term "guidelines" in the context of this book implies not only numerical values (guideline values), but also any kind of guidance given. Accordingly, for some substances the guidelines encompass recommendations of a more general nature that will help to reduce human exposure to harmful levels of air pollutants. For some pollutants no guideline values are recommended, but risk estimates are indicated instead. Table 33.1 summarizes the different endpoints on which guideline values and carcinogenic risk estimates have been based for organic and inorganic substances, showing that all relevant biological effects (endpoints) were evaluated and sometimes more than one endpoint was considered for guideline recommendations.

TABLE 33.1
Established guideline values and risk estimates

Substance	IARC Group classification	Risk estimate based on carcinogen endpoint	Guideline values based on:		
			Toxicological endpoint	Sensory effects or annoyance reaction	Ecological effects
Organic substances					
Acrylonitrile	2A	X			
Benzene	1	X			
Carbon disulfide		X	X		
1,2-Dichloromethane	a		X		
Dichloromethane	a		X		
Formaldehyde	2B		X		
Polynuclear aromatic hydrocarbons (Benzo[a]pyrene)	b	X			
Styrene	3		X	X	
Tetrachloroethylene	3		X	X	
Toluene			X	X	
Trichloroethylene	3		X		
Vinyl chloride	1	X			
Inorganic substances					
Arsenic	1	X			
Asbestos	1	X			
Cadmium	2B		X		
Carbon monoxide		X			
Chromium (VI)	1	X			
Hydrogen sulfide		X	X		
Lead	3		X		
Manganese			X		
Mercury		X			
Nickel	2A[c]	X			
Nitrogen dioxide		X		X	
Ozone/photochemical oxidants			X		X
Radon			X		
Sulfur dioxide and particulate matter			X		X
Vanadium			X		

[a] Not classified but sufficient evidence of carcinogenicity in experimental animals.
[b] Not classified, but sufficient evidence of carcinogenicity of PAH in humans in some occupational exposures (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 34). Sufficient evidence of carcinogenicity for benzo[a]pyrene is present as a component of the total content of polycyclic aromatic hydrocarbons in the environment (IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 32).
[c] Exposures from nickel refineries are classified in Group 1.

The numerical guideline values and the risk estimates for carcinogens (Tables 33.2–33.5) should be regarded as the shortest possible summary of a complex scientific evaluation process. Scientific results are an abstraction of real life situations, and this is even more true for numerical values and estimates based on such results. Numerical guideline values, therefore, are not to be regarded as separating the acceptable from the unacceptable, but rather as indications. They are proposed in order to help avoid major discrepancies in reaching the goal of effective protection against recognized hazards. Moreover, numerical guidelines for different substances are not directly comparable. Variations in the quality and extent of the scientific information and in the nature of critical effects result in guideline values which are only comparable between pollutants to a limited extent.

Owing to the different bases for evaluation, the numerical values for the various air pollutants should be considered in the context of the accompanying scientific documentation giving the derivation and scientific considerations. Any isolated interpretation of numerical data should therefore be avoided and guideline values should be used and interpreted in conjunction with the information contained in the appropriate sections.

It is important to note that guidelines are for individual chemicals. Pollutant mixtures can yield differing toxicities, but data are at present insufficient for guidelines relating to mixtures (except that of sulphur dioxide and suspended particulates) to be laid down.

33.2.1 Guideline values based on effects other than cancer

The guideline values for individual substances based on effects other than cancer and outdoor are given in Table 33.2. Guideline values for combined exposure to sulphur dioxide and particulate matter are indicated in Table 33.3.

The emphasis in the guidelines is placed on exposure, since this is the element that can be controlled to lessen dose and hence lessen response. As stated earlier, the starting-point for the derivation of guideline values was to define the lowest concentration at which adverse effects are observed. On the basis of the body of scientific evidence and judgements of protection (safety) factors, the guideline values were established.

However, compliance with the guideline values does not guarantee the absolute exclusion of undesired effects at levels below the guideline values. It means only that guideline values have been established in the light of current knowledge and that protection factors based on the best scientific judgements have been incorporated, though some uncertainty cannot be avoided.

For some substances, a direct relationship between concentrations in air and possible toxic effects is very difficult to establish. This is especially true of

TABLE 33.2

Guideline values for individual substances based on effects other than cancer or odor/annoyance

Substance	Time-weighted average	Averaging time	Chap.
Cadmium	1–5 ng/m^3	1 year (rural)	19
	10–20 ng/m^3	1 year (urban)	
Carbon disulfide	100 μg/m^3	24 h	7
Carbon monoxide	100 mg/m^3	15 min	20
	60 mg/m^3	30 min	
	30 mg/m^3	1 h	
	10 mg/m^3	8 h	
1.2-Dichloroethane	0.7 mg/m^3	24 h	8
Dichloromethane (methylene chloride)	3 mg/m^3	24 h	9
Formaldehyde	100 μg/m^3	30 min	10
Hydrogen sulfide	150 μg/m^3	24 h	22
Lead	0.5–1.0 μg/m^3	1 year	23
Manganese	1 μg/m^3	1 year	24
Mercury	1 μg/m^3	1 year	25
Nitrogen dioxide	400 μg/m^3	1 h	27
	150 μg/m^3[1]	24 h	
Ozone	150–200 μg/m^3	1 h	28
	100–120 μg/m^3	8 h	
Styrene	800 μg/m^3	24 h	12
Sulphur dioxide	500 μg/m^3	10 min	30
	350 μg/m^3	1 h	
Sulphuric acid	e		30
Tetrachloroethylene	5 mg/m^3	24 h	13
Toluene	8 mg/m^3	24 h	14
Trichloroethylene	1 mg/m^3	24 h	15
Vanadium	1 μg/m^3	24 h	31

[a] Information from this table should not be used without reference to the rationale given in the chapters indicated.

[b] Exposure at these concentrations should be for no longer than the indicated times and should not be repeated within 8 hours.

[c] Due to respiratory irritancy, it would be desirable to have a short-term guideline, but the present data base does not permit such estimations.

[d] The guideline value is given only for indoor pollution, no guidance is given on outdoor concentrations (via deposition and entry into the food chain) that might be of indirect relevance.

[e] See Chapter 30.

Note: When air levels in the general environment are orders of magnitude lower than the guideline values, present exposures are unlikely to present a health concern. Guideline values in those cases are directed only to specific release episodes or specific indoor pollution problems.

TABLE 33.3

Guideline values for combined exposure to sulphur dioxide and particulate matter

	Averaging time	Sulphur dioxide ($\mu g/m^3$)	Reflectance assessment: black smoke [b] ($\mu g/m^3$)	Gravimetric assessment	
				Total suspended particulates (TSP)[c] ($\mu g/m^3$)	Thoracic particles (TP)[d] ($\mu g/m^3$)
Short term	24 h	125	125	120[e]	70[e]
Long term	1 year	50	50	–	–

[a] Not direct comparison can be made between values for particulate matter in the right-and left-hand sections of this table, since both the health indicators and the measurement methods differ. While numerically TSP/TP values are generally greater than those of black smoke, there is no consistent relationship between them, the ration of one to the other varying widely from time to time and place to place, depending on the nature of the sources.
[b] Nominal $\mu g/m^3$ units, assessed by reflectance. Application of the black smoke value is recommended only in areas where coal smoke from domestic fires is the dominant component of the particulates. It does not necessarily apply where diesel smoke is an important contributor.
[c] TSP: measurement by high volume sampler, without any size selection.
[d] TP: equivalent values as for a sampler with ISO-TP characteristics (having 50% cut-off point at 10 μm), estimated from TSP values using site-specific TSP/ISO-TP ratios.
[e] Values to be regarded as tentative at this stage, being based on a single study (involving sulphur dioxide exposure also).
Sulphur oxides point to the need for a significant reduction of emissions in some areas.

those metals for which a greater body-burden results from ingestion than from inhalation. For instance, available data show that the food chain is, for most people, the critical route of non-occupational exposure to lead and cadmium. On the other hand, airborne lead and cadmium may contribute significantly to the contamination of food by these metals. Complications of this kind were taken into consideration and an attempt was made to develop air quality guidelines which would also prevent those toxic effects of air pollutants that resulted from uptake through both ingestion and inhalation.

For certain compounds, such as organic solvents, the proposed health-related guidelines are orders of magnitude higher than current ambient levels. The fact that existing environmental levels for some substances are much lower than the guidelines by no means implies that pollutant burdens may be increased up to the guideline values. Any level of air pollution is a matter of concern, and the existence of guideline values never means a license to pollute.

The approach taken in the preparation of the air quality guidelines was to use expert panels to evaluate data on the health effects of individual com-

TABLE 33.4

Rationale and guideline values based on sensory effects or annoyance reactions, using an averaging time of 30 min

Substance	Detection threshold	Recognition threshold	Guideline value
Carbon disulfide in viscose emissions			20 g/m^3
Hydrogen sulfide	0.2–2.0 g/m^3	0.6–6.0 g/m^3	7 g/m^3
Styrene	70 g/m^3	210–280 g/m^3	70 g/m^3
Tetrachloroethylene	8 mg/m^3	24–32 mg/m^3	8 mg/m^3
Toluene	1 mg/m^3	10 mg/m^3	1 mg/m^3

pounds. As a part of this approach, each chemical is considered in isolation. Inevitably, there is little emphasis on such a factor as interaction between pollutants that might lead to additive or synergistic effects and on the environmental fate of pollutants (e.g. the role of solvents in atmospheric photochemical processes leading to the formation or degradation of ozone, the formation of acid rain and the propensity of metals and trace elements to accumulate in environmental niches). These factors militate strongly against allowing a rise in ambient pollutant levels. Many uncertainties still remain, particularly regarding the ecological effects of pollutants, and therefore efforts should be continued to maintain air quality at the best possible level.

Unfortunately, the situation with regard to actual environmental levels and proposed guideline values for some substances is just the opposite. For substances with malodorous properties at concentrations below those where toxic effects occur, guideline values likely to protect the public from odour nuisance were established; these were based on data provided by expert panels and field studies (Table 33.4). In contrast to other air pollutants, odorous substances in ambient air often cannot be determined easily and systematically by analytical methods because the concentration is usually very low. Furthermore, odours in the ambient air frequently result from a complex mixture of substances and it is difficult to identify individual ones; future work may have to concentrate on odours as perceived by individuals rather than on separate odours substances.

33.2.2 Guidelines based on carcinogenic effects

In establishing criteria upon which guidelines could be based, it became apparent that carcinogens and non carcinogens would require different ap-

TABLE 33.5

Carcinogenic risk estimates based on human studies

Substance	IARC Group classification	Unit risk[b]	Site of tumour
Acrylonitrile	2A	2×10^{-5}	lung
Arsenic	1	4×10^{-3}	lung
Benzene	1	4×10^{-6}	blood (leukaemia)
Chromium	1	4×10^{-2}	lung
Nickel	2A	4×10^{-4}	lung
Polynuclear aromatic hydrocarbons (carcinogen fraction)	9×10^{-2}	lung	
Vinyl chloride	1	1×10^{-6}	liver and other sites

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[a] Calculated with average relative risk model
[b] Cancer risk estimates for lifetime exposure to a concentration of 1 g/m^3.
[c] Expressed as benzo[a]pyrene (based on benzo[a]pyrene concentration of 1 g/m^3 in air as a component of benzene-soluble coke-oven emissions).

TABLE 33.6

Risk estimates for asbestos

Concentration	Range of lifetime risk estimates
500F°/m^3 (0.0005 F/ml)	10^{-6}–10^{-5} (lung cancer in a population where 30% are smokers) 10^{-5}–10^{-4} (mesothelioma)

Note: F° = figures measured by optical methods.

proaches. These are determined by theories of carcinogenesis which postulate that there is no threshold for effects (i.e. that there is no safe level). Therefore, risk managers are faced with two decisions: either to prohibit a chemical or to regulate it at levels that result in an acceptable degree of risk. Indicative figures for risk and exposure assist the risk manager to reach the latter decision. Therefore, air quality guidelines are indicated in terms of incremental unit risks in respect of those carcinogens for which at least limit evidence of carcinogenicity in humans exists (Table 33.5).

Separate consideration is given to risk estimates for asbestos (Table 33.6) and radon daughters (Table 33.7) because they refer to different physical units and are indicated in the form of ranges.

TABLE 33.7

Risk estimates and recommended action level for radon daughters

Exposure	Lung cancer excess lifetime risk estimate	Recommended level for remedial action in buildings
1 Bq/m^3 EER	$(0.7\times10^{-4}) - (2.1\times10^{-4})$	$\geq$ 100 Bq/m^3 EER (annual average)

Unfortunately, the recent reclassification of dichloromethane by IARC has not allowed sufficient time to publish a detailed risk estimate which takes into account important information on the metabolism of the compound. The risk estimate for cancer from the animal bioassay is not used for this reason in the guidelines.

Formaldehyde represents a chemical for which cancer bioassays in rats have resulted in nonlinear exposure response curves. The non linearity of the tumour incidence with exposure concentrations led Starr and Buck to introduce the "delivered dose" (amount of formaldehyde covalently bound to respiratory mucosal DNA) as the measure of exposure into several low-dose extrapolation models (Starr and Buck, 1984). Results showed considerable differences in the ratio between risk estimates based on the administered dose and those based on the delivered dose, with a great variance of ratios between models. Since estimates vary because of the inherent differences in approach, cancer risk estimates are referred to but not used for the guidelines. In addition, such estimates should be compared with human epidemiological data when an informed judgement has to be made.

The evidence for carcinogenicity of 1,2-dichloroethane in experimental animals is sufficient, being based on ingestion data. No positive inhalation bioassays are available. Consequently, an extrapolation from the ingestion route to the inhalation route is needed to provide a cancer risk estimate from the bioassay data. Such extrapolations are the best conducted when detailed information is available on the kinetics of metabolism, distribution and excretion. Two estimates calculated from data on oral studies are provided for the risk of cancer through inhalation of 1,2-dichloroethane, but they lack detailed data for the route-to-route extrapolation and are not used in the guidelines.

It is important to note that quantitative risk estimates may give an impression of accuracy which in fact they do not have. An excess of cancer in a population is a biological effect and not a mathematical function, and uncertainties of risk estimation are caused not only by inadequate exposure data but also, for instance, by the fact that specific metabolic properties of

agents are not reflected in the models. Therefore, the guidelines do not indicate that a specified lifetime risk is virtually safe or acceptable.

The decision on the acceptability of a certain risk should be taken by the national authorities in the context of a broader risk management process. Risk estimate figures should not be applied in isolation when regulatory decisions are being made; combined with data on exposure levels and individuals exposed, they may be a useful contribution to risk assessment. Risk assessment can then be used together with technological, economic and other considerations in the risk management process.

33.2.3 Guidelines based on ecological effects on vegetation

Although the main objective of the air quality guidelines in the direct protection of human health, it was decided that ecological effects of air pollutants on vegetation should also be considered. The effects of air pollutants on the natural environment are of special concern when they occur at concentrations lower than those that damage human health. In such cases, air quality guidelines based only on effects on human health would allow for environmental damage that might indirectly affect human well-being.

TABLE 33.8

Guideline values for individual substances based on effects on terrestrial vegetation

Substance	Guideline value	Averaging time	Remarks
Nitrogen dioxide	95 $\mu g/m^3$	4 h	In the presence of SO_2 and O_3 levels which are not higher than 30 $\mu g/m^3$ (arithmetic annual average) and 60 $\mu g/m^3$ (average during growing season), respectively
	30 $\mu g/m^3$	1 year	
Total nitrogen deposition	3 g/m^2	1 year	Sensitive ecosystems are endangered above this level
Sulphur dioxide	30 $\mu g/m^3$	1 year	Insufficient protection in the case of extreme climatic and topographic conditions
	100 $\mu g/m^3$	24 h	
Ozone	200 $\mu g/m^3$	1 h	
	65 $\mu g/m^3$	24 h	
	60 $\mu g/m^3$	averaged over growing season	
Peroxyacetylnitrate	300 $\mu g/m^3$	1 h	
	80 $\mu g/m^3$	8 h	

It should be understood that the pollutants selected (SO_x, NO_x and ozone/photochemical oxidants) (Table 33.8) are only a few of a larger category of air pollutants that may adversely affect the ecosystem. Furthermore, the effects which were considered are only part of the spectrum of ecological effects. Effects on aquatic ecosystems were not evaluated, nor were effects on animals taken into account. Nevertheless, the available information indicates the importance of these pollutants and of their effects on terrestrial vegetation in the context of the European Region.

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Chapter 34

Air Quality Guidelines for Selected Pollutants[1]

34.1 RECOMMENDED AND REGULATORY RADON LEVELS

34.1.1 Introduction

Regulation of radon in the domestic environment is an issue which has been the subject of much debate by national and international bodies, due to its complexity and peculiarities.

Radon is a natural gas, is ubiquitous and affects homes and the private sphere of individuals. Contrary to what happens with other pollutants whose use could be forbidden if considered necessary, radon can be reduced but not totally eliminated. Moreover, due to the stochastic characteristic of radon health effects and to the hypothesis of a linear relationship between radon exposure and excess lung cancer probability, it is considered that there is no safe value.

It is therefore difficult to choose an "action level" above which remedial actions are recommended, some of which were outlined in Section 31.3. This choice is not totally based on scientific aspects only, such as health effect risk, but takes into account other aspects of the problem. The ICRP recommends that "the best choice of an action level may be that level which defines a significant, but not unmanageable, number of houses in need of remedial work. It is then not to be expected that the same action level will be appropriate in all countries" (ICRP, 1991). Moreover "any action affecting the whole housing stock of a country would be extremely costly, although it might still be cost-effective in terms of the reduction in the national collective dose. It is for national authorities to decide whether the necessary funds would be available and best spent on general radon reduction or other aspects of housing improvement" (ICRP, 1993).

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1 A part of the text of this chapter has been derived from ECA (European Collaborative Action "Indoor Air Quality and its Impact on Man", COST Project 613), 1994. Radon in indoor air, Report No. 15. Luxembourg: Office for Official Publications of the European Communities.

In many countries one or more surveys have been carried out before the choice of their action levels, in order to be able to estimate the impact of the chosen value on the basis of radon concentration distribution.

Moreover, some national and international authorities decided to use different values for existing and future (also referred to as "new") dwellings and workplaces, as suggested by ICRP in 1984, because of the greater difficulties in applying remedial action in existing buildings. Both levels, the action level for existing buildings and "upper bounds" for future ones, are often referred to as "reference levels". Generally, all recommended reference values are to be considered annual radon concentration averages. Please note that in other fields the term "reference level" may have a different meaning.

Until now, in most countries the approach to dealing with indoor radon has been limited to recommendations. It has proved very difficult to transform these into regulatory requirements.

34.1.2 Application to dwellings

The actual recommendations are summarised in Table 34.1 and show a wide spectrum. Due to radon concentration variability, reference levels are generally to be intended as annual average values. Some of the values presented were originally expressed in terms of radon progeny concentration, and are here converted to radon gas values assuming an equilibrium factor of 0.5 (see also Section 3.4.1.1).

Historically, ICRP suggested in its 1984 Publication No. 39 (ICRP, 1984) an action level of 400 Bq/m^3 for existing dwellings as a reference level above which remedial action is suggested, the urgency and severity of the remedial action being related to the amount by which the level is exceeded. Moreover, an upper bound of 200 Bq/m^3 for new dwellings was also proposed.

The Commission of European Communities issued a recommendation in 1990 (CEC, 1990) to limit the radon exposure in buildings which follows strictly the ICRP Publication No. 39.

In the United States in 1986 the EPA recommended 150 Bq/m^3 as a reference level above which remedial action is suggested, the urgency and severity of the remedial action being related to the amount by which the level is exceeded. The same upper bound level was indicated for new dwellings. In the Indoor Radon Abatement Act of 1988, the U.S. Congress set the long-term goal of reducing indoor radon concentration as low as the ambient air. The EPA considers that while this goal is not yet technologically achievable in all cases, it is possible to reduce radon levels to 75 Bq/m^3 in most cases (EPA, 1992).

In Canada the levels suggested by the Federal and Provincial Ministers of Health in 1988 (Létourneau, 1985) for both old and new dwellings is 800 Bq/m^3

TABLE 34.1

Recommended reference levels for radon gas in dwellings (Bq/m^3)

Country/organization	Existing dwellings	Future dwellings
Belgium	250	250
Canada	800	800
CEC	400	200
EPA (USA)	150	150
Finland	400	200
Germany	250	250
ICRP (1984)	400	200
ICRP (1993)	200–600	–
Ireland	200	200
Norway	200	200
Sweden	140 and 400	140
United Kingdon	200	200
WHO	200	200

(McGregor, 1993), but it is recognised that "there is some risk at any level, so that home-owners may wish to reduce levels of radon as low as practicable" (Eaton, 1990).

In the Nordic countries the radiation protection authorities collectively adopted in 1986 the limit value of 800 Bq/m^3 for existing dwellings and the design level of 200 Bq/m^3 for future dwellings. The recommended value for existing dwellings in case of simple remedial actions is 200 Bq/m^3. Individual Nordic countries have since then adopted national reference values.

In Sweden a regulatory action level of 800 Bq/m^3 was established in 1980, and home-owners are recommended to decrease the level to below 200 Bq/m^3 if this is possible with reasonable efforts. In 1990 the limit was decreased to 400 Bq/m^3 and the recommended action level for simple measures was decreased to 140 Bq/m^3 (Snihs, 1992). The design level for new dwellings is 140 Bq/m^3 (Swedjemark, 1990).

In Finland the reference levels are 400 Bq/m^3 for existing dwellings and 200 Bq/m^3 for future dwellings (Castrén, 1993b).

In Norway both the action level for existing dwellings and the upper bound for future dwellings are 200 Bq/m^3 of radon concentration annual average (Strand, 1993).

In the United Kingdom, in 1990 following a nationwide radon survey and using the revision of radiation risk made by ICRP (1991), the action level was

decreased from 400 to 200 Bq/m^3, above which householders should reduce radon concentrations before the occupants receive a further time-integrated concentration of 1500 Bq /year/m^3 (NRPB,1990). In future dwellings the radon concentration levels should be as low as reasonably practical, and at least below the action level of 200 Bq/m^3.

A recommended reference level for both existing and new dwellings in Ireland of 200 Bq/m^3 was adopted in 1990.

In Germany, in 1994, the German Commission on Radiological Protection adopted a recommendation in which no action is suggested for radon concentrations below 250 Bq/m^3, considered "normal", and only simple actions are considered to reduce radon concentration for levels to 1000 Bq/m^3, while stronger actions are recommended only for higher levels. The upper bound for future dwellings is 250 Bq/m^3.

In Belgium a single reference level of 250 Bq/m^3 has been adopted.

The WHO includes radon among the agents that are carcinogenic to humans (WHO/IARC, 1988) and recommends simple remedial action for buildings with annual radon concentrations of 200 Bq/m^3, without delay if the radon concentration is higher than 800 Bq/m^3; the same upper bound of 200 Bq/m^3 is recommended for new dwellings (WHO, 1987).

The ICRP has recently adopted a new recommendation on "protection against radon at home and at work" (ICRP, 1993), in which a range of action level values is proposed for existing dwellings: from 200 to 600 Bq/m^3. The upper bound for future dwellings is now considered unhelpful and is substituted with issuing guidance and codes on construction practices, especially for areas where many high radon concentration values have been measured or estimated.

34.1.3 Application to "normal" workplaces

In the new ICRP recommendations on radon, a range of reference levels are also proposed for workplaces where the occupancy of members of the public is low, e.g. in offices, libraries and theatres: from 500 to 1500 Bq/m^3 (ICRP, 1993). These values differ from those for dwellings because of the shorter period of exposure in workplaces, where 2000 hours per year are assumed, and because of the assumed different radiosensitivity between the general population and workers.

In the UK the law requires employers to limit the exposure of workers to radon daughter, and the action level is 400 Bq/m^3 of radon gas (Gooding and Dixon, 1992).

34.1.4 Limitation of radioactivity concentration in building materials

Up to now, limitations on the use of building materials are generally related to the gamma radiations emitted by the radionuclides that are incorporated into the building materials (NEA/OECD, 1979). However, building materials are considered also as radon sources and the ICRP recommends identifying building materials with high Ra-226 content, and preventing or limiting their use (ICRP, 1993). Some countries have already adopted recommendations on radionuclide content in building materials, e.g. the Netherlands and the Nordic countries.

34.1.5 Conclusions

The spectrum is rather wide. The levels suggested are linked to the special situation of each country: geology of soils, its characteristics, etc., and to general social and economic status.

A basic problem is the cost of remedial actions and who has to sustain the economical burden: the individual citizen or society as a whole.

As outlined above the setting-up of reference levels for indoor radon in dwellings is a political rather than a technical problem. National authorities can offer a careful analysis of the situation (levels detected, number of houses with high concentrations, etc.) and could establish the economical and social cost of remediation. International bodies can only give general guidelines but it is up to the governments to make the final decision.

A major obstacle in reducing the doses from radon to the population by a non-regulatory approach is the general apathy of the public. In recognition of this, a number of organisations such as the World Health Organisation recommend that effective radon risk communication programmes should be an essential part of a comprehensive radon risk management policy (WHO, 1993).

34.2 GUIDELINE VALUES FOR BIOLOGICAL PARTICLES

A working group of the EC Concerted Action 613 "Indoor Air Quality and Its Impact on Man" agreed that it was unsafe to establish reference values for health risk evaluation (COST 613, 1993), because:

1. there is a lack of standardization in sampling methods, and
2. results of sampling are highly dependent on factors such as type of sampler, household or work activity prior to and during sampling, season of year and geographical area.

TABLE 34.2

Levels of housedust mite, cat and dog allergens in house dust (after COST 613, 1993)

Category	Housedust mite (μg/g dust)		Cat Feel d I (ng/g dust)	Dog Can f I (ng/g dust)
	Der p I	Der f I		
Very low	< 0.5	< 0.5	< 100	< 300
Low	< 5	< 5	< 1000	< 10000
Intermediate	< 15	< 15	< 10000	< 100000
High	< 20	< 20	< 100000	< 1000000
Very high	> 20	> 20	> 100000	> 1000000

A final factor which makes assessment of exposure of any individual or group to these pollutants highly problematic is that the usual methods employed, i.e. sampling for viable airborne micro-organisms, seriously underestimate the total airborne burden (including non-viable particles which have the same allergic and toxic characteristics as the corresponding viable particles).

However, the working group categorized levels of contamination in air and dust according to observed values in homes and non-industrial workplaces, without implying health risk. The categories for house dust mite, cat and dog allergens are given in Table 34.2. Although no health risk assessment was given by this group it has been considered by WHO (Platts-Mills and de Weck, 1988) hygienic threshold limits should be: 2 μg Der p I/g dust to be regarded as representing a risk for genetically predisposed individuals in the development of specific IgE to house dust mite allergen; and 10 μg Der p I/g dust to be regarded as a risk factors for acute attacks of asthma. At this higher level most mite-allergic patients will experience symptoms.

Observed values which may be of use in evaluating results of air-sampling for viable fungi and bacteria are given in Table 34.3, but again no health risk evaluation can be based on these values.

Rank order assessment of components of the fungal flora has been in use since 1986 in Government of Canada investigations as a method of interpreting sampling data collected under defined conditions. If a Reuter centrifugal sampler is used to collect a 4-min sample, the following guidelines are applied in Canadian investigations (Nathanson, 1993).

– The presence of certain pathogens (e.g. *Aspergillus fumigatus*) and toxi-

TABLE 34.3

Levels of viable fungi and bacteria in indoor air (after COST 613, 1993)

Category	Fungi (CFU/m^3 air)[a]		Bacteria (CFU/m^3 air)[b]	
	Home	Non-industrial workplace	Home	Non-industrial workplace
Very low	< 50	< 25	< 100	< 50
Low	< 200	< 100	< 500	< 100
Intermediate	< 1000	< 500	< 2500	< 500
High	< 10000	< 2000	< 10000	< 2000
Very high	> 10000	> 2000	> 10000	> 2000

[a] Mixed populations determined using Andersen six-stage sampler in combination with malt extract agar, or N6 Andersen one-stage sampler with this agar or DG 18 agar.
[b] Mixed populations, using Andersen six-stage sampler or slit sampler.

genic fungi (e.g. *Stachybotrys atra*, toxigenic *Aspergillus*, *Penicillium* and *Fusarium* species) is unacceptable.

- The presence of one or more fungal species occurring as a significant percentage in indoor air samples and not similarly present in concurrent outdoor samples is evidence of a fungal amplifier. The "normal" air mycoflora is qualitatively similar and quantitatively lower than that of outdoor air. In Canadian federal government buildings, a 3-year average was approximately 40 CFU/m^3 for *Cladosporium*, *Alternaria* and non-sporulating basidiomycetes.
- The significant presence of fungi in humidifiers and diffuser ducts and on mouldy ceiling tiles and other surfaces requires remedial action, regardless of the airborne spore load.
- More than 50 CFU/m^3 is cause for concern if there is only one species other than *Cladosporium* or *Alternaria* present. Further investigation is necessary.
- Up to 150 CFU/m^3 is acceptable if there is a mixture of species reflective of the outdoor air spores. Higher counts suggest dirty air filters or other problems.
- Up to 500 CFU/m^3 is acceptable in summer if the species present are primarily *Cladosporium* or other tree and leaf fungi. Values higher than this usually indicate failure of filters or contamination within the building.

Such data are considered in relation to two parallel outdoor samples. The

TABLE 34.4

Level of viable fungi in dust as assessed by direct plating of dust and plating of dust suspensions on DG 18 Agar (after COST 613, 1993)

Category	Direct plating[a]	Suspension plating[b]
Very low	< 2000	< 10000
Low	< 6000	< 20000
Intermediate	< 9000	< 50000
High	< 15000	< 120000
Very high	> 15000	> 120000

[a] 30 mg dust spread over surface of medium.
[b] 0.1 ml of dust (100 mg) suspension in peptone solution spread over medium.

presence of species which are absent outdoors indicates indoor amplification sites. This must be regarded as a particularly serious matter in the case of hazardous fungi such as *Stachybotrys*.

Where high counts of bacteria comprise mainly Gram-positive bacteria associated with skin-scales shed by occupants they can be regarded as indicators of inadequate ventilation. However, if Gram-negative bacteria, such as *Pseudomonas aeruginosa*, or thermophilic Actinomycetes are prominent, a potential health risk is implicit. Routine sampling of air for *Legionella* is not recommended, but again where it is found there is an implicit health hazard. *Legionella* instead should be systematically searched for in water reservoirs of HVAC, where the conditions for its growth are met.

Although dust has been shown to be a reservoir of potentially harmful bacteria in hospitals, there is insufficient information to allow counts to be categorized as for fungi (Table 34.4). As with air samples, the presence of certain fungi in dust may be indicative of a potential respiratory hazard, but there is little information on the aerosolization of dust-borne organisms (Flannigan et al., 1992).

Finally, it must again be emphasized that there are at present no methods which can adequately assess exposure to biological particles. Consequently, for non-infectious fungi and bacteria, no quantitative assessment of health risk can be made and no health guidelines values can be given.

However, the presence of visible mould growth in buildings and of certain pathogenic and toxigenic organisms in quantity in indoor air should be regarded as unacceptable.

34.3 VOLATILE ORGANIC COMPOUNDS[1]

The guideline values published (WHO, 1987; Health and Welfare, 1987; COST 613, 1990) have been derived on the basis of available knowledge of direct or indirect health effects of individual substances. Besides the fact that such a substance-by-substance approach is very time-consuming, it does not take synergistic effects into account which are likely to be especially important in the case of mixtures of volatile organic compounds (VOC; "VOC" is used in the sense of a WHO definition (WHO/EURO, 1989) which specifies that VOC include volatile organic compounds with a boiling point range from 50–100 to 240–260°C sampled by adsorption on a solid sorbent).

Although the pioneering work by Mølhave et al. (1986) suggests that it may be possible to define a concentration level at which total volatile organic compounds (TVOC) may start to cause problems (like those associated with sick building syndrome), value-setting remains difficult unless agreement is reached on what is meant by "TVOC". Mølhave et al. (1986) called TVOC a mixture of 22 VOC (excluding any carcinogen) selected on the basis of the occurrence of these compounds in the air of non-industrial indoor environments. TVOC concentrations as measured by others under field conditions represent the sum of a large number of VOC calibrated either individually (Lebret et al., 1986; Krause et al., 1987) or using one common response factor for all VOC (Knöppel et al., 1989). Finally, some authors simply call TVOC what is indicated by a direct-reading instrument such as a flame-ionisation or photo-ionisation detector (Gammage et al., 1989). Results obtained by Knöppel and De Bortoli (1989) give numerical evidence for what any good analyst would have expected, namely that there is no (and cannot be any) satisfactory correlation between the various methods used to measure TVOC if different indoor spaces are compared.

Under these conditions, a convention is badly needed to define what "TVOC" means. In contrast to the definition given by Gammage et al. (1989), which is based on an integrated real-time signal of monitor, this convention should be based on a measuring technique which permits to separate substances. The following basic definition is therefore proposed for TVOC: "Total Volatile Organic Compounds" (TVOC) means the sum of individual VOC separated and quantified by a gas chromatographic technique.

To obtain the sum, the VOC belonging to one of the following chemical classes are ranked according to their measured concentration: alkanes, aromatic hydrocarbons, terpenes, halocarbons, esters, carbonyl compounds (ex-

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1 Text based on B. Seifert: Regulating Indoor Air. Proc. 5th International Conference on Indoor Air Quality and Climate, Toronto, Canada, July 29 to August 3, 1990, Vol. 5.

cluding formaldehyde) and "other". The concentrations of the first ten VOC in each class are then summed up.

With such definition it becomes also possible to set a target guideline value for TVOC which is proposed to amount to 0.3 mg/m^3. Table 34.5 shows how much the different chemical classes of VOC are allowed to contribute to this value. In a first step, the value is intended to be applicable to non-industrial indoor environments outside the private sphere such as offices, schools, kindergartens, etc., in the state of non-occupancy. It is proposed to be valid with the following boundary conditions: no individual VOC should exceed 50% of the concentration allotted to its class and 10% of the TVOC concentration.

It must be emphasised that the proposed target value is *not* based on toxicological considerations but — to the author's best judgement — reflects what could be achieved taking into account the current knowledge on concentration levels found in the literature dealing with VOC (WHO/EURO, 1989). Also, the proposal does not exclude that additional guideline values be set for individual compounds if needed.

The numbers given in Table 34.5 are intended to represent "normal" long-term conditions. It is obvious that under special circumstances, e.g. during and shortly after renovation works, these levels cannot be respected. Consequently, it is further proposed that for up to 1 week and 6 weeks, 50 and 10 times higher concentration levels, respectively, be acceptable. These time intervals have been chosen on the basis of information on removal processes. They will permit renovation works to be carried out under conditions of

TABLE 34.5

Proposed target guideline value for total volatile organic compounds (TVOC) in indoor air (see text)

Chemical class of VOC	Concentration (μg/m^3)
Alkanes	100
Aromatic hydrocarbons	50
Terpenes	30
Halocarbons	30
Esters	20
Aldehydes and ketones (excl. formaldehyde)	20
Other	50
Target guideline value (sum of VOC)	300

Note: The concentration of an individual compound should neither exceed 50% of the concentration allotted to its class nor 10% of the TVOC concentration.

elevated ventilation rates and non-occupancy of rooms (e.g., during school holidays).

To limit analytical work in the field, measurements using a direct-reading instrument may still be appropriate under certain circumstances, e.g. to follow removal processes. As a rule of thumb, one might then assume that the result given by the monitor would agree with that obtained by gas chromatographic analysis within a factor of 2–3. However, since the signal of the monitor depends on the type of instrument used and the mixture actually present in the room this figure cannot be more than indicative.

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Chapter 35

Recommendations of the NATO/CCMS Pilot Study on Indoor Air Quality[1]

35.1 INTRODUCTION

The North Atlantic Treaty Organization's Committee on the Challenges of Modern Society (NATO/CCMS) was established in 1969 to address non-military issues facing NATO nations. The CCMS supports studies on topics already being researched at the national level within NATO nations and has particular interest in issues related to the environment. The aim of CCMS studies is to combine the available expertise and technology on specific issues to the benefit of all member countries.

In 1988, Italy proposed to the CCMS a pilot study on indoor air quality on account of the increasing international importance of the issue. Research has found that individuals typically spend between 80 and 90% of their time indoors. Health studies have found that exposures to a variety of air pollutants indoors can be substantially higher than outdoors, even in large cities. This exposure research suggests a high potential risk associated with indoor environmental factors in modern buildings. Indoor air pollution is a concern both to human health and productivity.

Because a reduction in indoor air pollution is an important international goal, the CCMS pilot study on indoor air quality was designed to foster international cooperation regarding research and strategies for the prevention and mitigation of indoor air pollution.

At its first meeting in Erice, Italy, members of the Pilot Study agreed that the general program of the Pilot Study should target two main areas: (1) regulatory issues, and (2) research and technical issues related to indoor air quality. Participants agreed on two policy objectives for the study. The first objective was to develop a network of the agencies, institutions, and individuals responsible for establishing policy and regulations on IAQ issues within individual NATO nations. The second objective was to examine policy strate-

1 The text of this chapter has been derived from the Pilot Study on Indoor Air Quality-Final Report, No. 195, NATO/CCMS Publications, Eds. Robert Axelrad and Marco Maroni, April 1994.

gies and propose a range of options that could be adopted by NATO nations.

Study members also identified several research objectives: identify current research efforts and develop a registry of research contacts; characterize indoor air quality problems in NATO countries; identify priority problems that pose high risks to human health and materials; and identify, study and recommend mitigation or control methods.

35.2 ACTIVITIES OF THE PILOT STUDY

The Pilot Study included subsequent meetings on six topics related to indoor air quality. A brief summary of the main topics presented and discussed at each of the Pilot Study meetings is provided in the following sections. A book of proceedings is available for each of these workshops.

35.2.1 "Implications of Indoor Air Quality for Modern Society" Erice, Italy, February 1989 (Maroni and Berry, 1989)

The first plenary meeting of the Pilot Study was held in Erice, Italy in February, 1989. The purpose of the Erice meeting was to draft the strategy for a series of seven meetings whose purpose was to identify emerging issues related to indoor air quality. Preliminary discussion among experts at the Erice meeting resulted in a list of relevant topics, including the impact of indoor air quality on health and welfare, strategies for the prevention and mitigation of problems resulting from specific categories of pollutants, population risk assessment for indoor air pollution, and the economic implications of indoor air quality and its regulation and control. The Erice meeting was the preparatory and planning phase for the execution of the Pilot Study.

35.2.2 "Managing Indoor Air Quality Risks", St. Michaels, Maryland, October 1989 (U.S. EPA, 1989)

The meeting "Managing Indoor Air Quality Risks", held in St. Michael's, Maryland, explored programs and policies designed to resolve indoor air quality problems in participating countries. Forty participants representing seven countries shared information about regulatory and non-regulatory approaches to controlling indoor air quality. Participants also discussed indoor air quality pollutant guidelines, ventilation standards and guidelines, and indoor air quality problem diagnosis and mitigation in buildings.

35.2.3 "Energy and Building Sciences in Indoor Air Quality", Sainte-Adèle, Québec, Canada, August 1990 (Institut de Recherche en Santé et en Sécurité du Travail du Québec, 1990)

The third meeting of the Pilot Study focused on indoor air quality in relation to ventilation. Thirty-two participants from ten countries gathered in St. Adèle, Québec, to share information about different ways to construct healthy buildings that are also energy efficient. Presentations were based on four broad topics: Indoor Air Pollution and its Sources, Energy Efficient HVAC, Low Pollution Energy Conserving Design and Operation, and Intervention Strategies. Each presentation ended with discussion. Participants drafted a series of provisional recommendations, which are reported in Tables 35.1–35.3.

TABLE 35.1

Recommendations concerning building design and internal HVAC pollution

Building design	During the planning and design of a building, attention should be paid to the achievement of high standards of indoor air quality and climate and, to the extent possible, to high energy efficiency. To achieve this goal, control should be applied to all the different components and phases of the design and contruction process, taking into consideration local environmental peculiarities or specific unfavorable conditions. Great attention should be paid to the comissioning process of the building which should be preferably performed and certified by an independent agency or company. Guidance to the operators and users of the building should be much more effective than in the past. Manuals describing the design criteria and technology as well as needs for operation and maintenance of the building without adverse impact on IAQ and climate, should be made available to the occupants.
Internal HVAC pollution	HVAC systems should be designed and constructed so as to avoid use of contaminated air, recycling poor quality air and presence of reservoirs of biological contaminants. Regular and proper maintenance and cleaning of HVAC systems are essential with particular concern for filters and drain pans. Accessibility of every component of the HVAC system is necessary. The use of these HVAC system components such as fan coil units, induction units and unit ventilators should be discouraged if adequate maintenance cannot be guaranteed.

TABLE 35.2

Recommendations concerning source control

Material selection	Manufacturers should be obliged to provide sufficient information for the evaluation of their products' safety. Protection of legitimate confidentiality must not impede evaluation. Standardization and Harmonization of emission testing procedures have to continue at international level. In the meantime, research and application of different techniques and methodological intercomparison should be encouraged. Labelling of products and their ranking as far as safety and emission properties are concerned should be encouraged in order to facilitate their appropriate selection and use by the consumers. The choice of the process may vary according to the nature of the risk involved and the local priorities.
Biological contaminants	The presence and growth of moulds and other microorganisms in the indoor environment should be avoided. This can be effectively achieved by elimination and control of moisture. Good housekeeping and sanitation rather than the use of biocides are appropriate control measures also for mites.
Volatile organic compounds (VOCs)	Overall exposure to VOCs should be kept at the lowest possible level primarily by proper control of the emission sources. Pending improved methods, the TVOC concept is a useful tool for a practical assessment of the overall quality of materials and of indoor air. However, at least the type of major VOC species must be known. More research is recommended for the health and sensory effects of VOCs mixtures as well as for some individual compounds typically present in indoor air mixtures for which little toxicological knowledge is available.

35.2.4 "Epidemiology and Medical Management of Building-Related Complaints and Illnesses", Oslo, Norway, August 1991 (Maroni and Levy, 1991)

The National Institute of Occupational Health and the National Institute of Public Health in Norway organized the fourth Pilot Study meeting. The meeting explored different topics under the headings Epidemiology, Medical Diagnosis, Medical Treatment, and Medical and Technical Prevention of Building-Related Complaints and Illnesses. Thirty-seven participants representing medi-

TABLE 35.3

Recommendations concerning guidelines and standards

– The ideal solution for IAQ is to combine health and comfort with energy efficiency. Efficient control of emission sources of indoor pollutants is critical to this target. Should a conflict arise between health and energy conservation, the health target has to prevail.
– The relative importance of the different indoor pollutants as well as of their adverse effects on the population should be assessed in each community in order to identify those conditions that deserve priority, either for the severity of the effects or the extent of the affected population. The first priority in health promotion should be to protect the most sensitive individuals of the population.
– The development of guidelines and/or standards is an essential tool in promoting the achievement of high IAQ and should be encouraged whenever possible and appropriate. In particular, development of guidelines is recommended for pollutant concentration in indoor air, ventilation, emission from materials, and methods for IAQ assessment and control.
– Ventilation standards should take into consideration the total pollutant load present in a building as the result of building constituents, occupants and their activities. The ultimate goal of ventilation should be to provide good IAQ and satisfaction for the large majority of occupants.
– Pro-active interventions on economy domain (market or taxation) as well as information and education of the community should be used to achieve advanced objectives in IAQ. These actions should accompany and complement any regulatory policy on indoor air.

cal and technical expertise from twelve countries shared information about the medical problems connected with modern buildings. Plenary sessions and group discussions attempted to answer questions such as:

- how should Building-Related Illness (BRI) and Sick Building Syndrome (SBS) be defined?
- which clinical, physiological, and laboratory tests can be recommended to diagnose BRI?
- which tests can be recommended to assess exposure in the indoor environment? and
- what medical and technical advice can be given to prevent recurrences and to cure or alleviate illnesses or complaints related to indoor air quality?

The group discussions resulted in suggestions and preliminary recommendations, which are summarized in Tables 35.4–35.12.

TABLE 35.4
Recommendations concerning definitions

The following definitions should be used in the description of indoor related complaints and illnesses:

- *Building-Related Environmental Complaints (BREC):* Complaints of poor indoor air quality (IAQ) or poor indoor air environment. BREC are usually what we register in the complaint (annoyance) part of questionnaire studies.
- *Building-Related Symptoms (BRS) or Building-Related Health Complaints (BRC):* The health complaints (subjective symptoms) reported by *the single individual* as occurring inside a building and usually subsiding shortly after leaving it.
- *Sick Building Syndrome (SBS):* A term denoting a situation where an increased number of the occupants of a building complain of a typical group of general, unspecific and irritative symptoms, including particularly headache, lethargy, dry eyes, blocked nose and sore throat. The symptoms usually fade after the person has left the indoor environment but the specific cause is unidentified.
- *Building-Related Illness (BRI):* Clinical condition with defined symptoms and signs in which the cause (aetiology) is building-related and identifiable.
- *The difference between BRI and SBS* is that the building problems are identified in the former and undiagnosed in the latter. The term "Sick Building" should probably not be used on its own, but might be replaced with the expression "Building with indoor climate problems" or "Problem Building".

TABLE 35.5
Recommendations concerning health effects caused by indoor environment to be considered in Epidemiological Investigations (I). In planning epidemiological investigations on health effects caused by indoor environment, the following organ systems and subjective symptoms should be primarily considered. Whenever possible the subjective symptoms should be confirmed by objective examination using one or more of the available tests.

Target organ system	Subjective symptoms	Available tests
Respiratory system (nose, throat, lungs)	*Upper*: nasal stuffiness; watery nasal discharge; dryness of the throat *Lower*: cough; chest tightness; breathlessness; wheezing	physical examination; lung function tests; chest X-ray; serology; inhalation provocation tests; skin prick tests
Central nervous system (cerebral function)	Tiredness; dizziness; nausea; concentration difficulty; headache; heavy-headedness	neuro-behavioural tests; neurological examination; computer-assisted tomography (CT); positron emission tomography (PET); electroencepha- lography; ophthalmoscopy (mainly for differential diagnostic purposes)
Skin	Dryness; itching; rash; burning sensation	Physical examination; biopsy
Eye	Irritation, itching; feeling of dryness; red eyes; tired eyes; watering eyes	Physical examination; slit lamp examination; tear film break-up time after fluorescin instillation

TABLE 35.6

Recommendations concerning health effects caused by indoor environment to be considered in Epidemiological Investigations (II)

When the outcome is not well defined as in SBS, it is recommended to study single effects which are closely related to SBS. We recommend the following complaints, symptoms or health effect indicators as most rewarding to study, depending upon the relevant health problem:

Complaints of poor indoor air quality:

- feeling of stuffy air; malodorous smell; feeling of dry air

Sick Building Syndrome (SBS) Symptoms:

- Irritation of mucous membranes of the eye, nose and throat; headache; tiredness; dry skin, itching

For the study of building-related illness, the following indicators are suggested:

Infections (legionellosis, etc.):

- episodes of fever and signs of pneumonia; isolation of bacteria from patient and suspected reservoir; immunologic response

Allergy/hypersensitivity:

- asthmatic symptoms; allergic rhinitis; clear nasal secretion, blocked nose

Allergic alveolitis or toxic dust syndrome (humidifier fever):

- repeated episodes of fever; signs of pneumonia; immunologic response; identification of reservoir for infections/toxic agent.

TABLE 35.7

Recommendations on the use of questionnaire for epidemiological investigations of indoor-related health problems

Design criteria

- The assessment of the "sickness" of a building should be based on either the whole working group or random selection of workers stratified by facing, floor and distance from the window and not on volonteer studies.
- Simplicity in the questionnaires is essential; interlinked, complex or multiple-part questions should be avoided.
- Researchers are strongly recommended to use a validated questionnaire before making up new questions.
- Control groups and control buildings should always be included in the study design.
- The question of building- or work-relatedness of symptoms should be specifically addressed even though a unique format is not agreed upon.

Reference populations

- Background reference populations should be constructed on a national basis.
- Within the assessment of the reference population an assessment of exposure should be included, based on a self-administered questionnaire
- It is recommended there be a clear consistency in the questionnaires on a national basis in order to be able to use the national reference population values.
- It is recommended that a working group be identified to construct an international standard IAQ-questionnaire.

TABLE 35.8

Recommendations on diagnosis and medical management

Clinical and laboratory tests available for the investigation of building-related illnesses (BRI) and complaints (BRC)

- The diagnosis of BRI should be primarily based on patient medical history and clinical investigation.
- The physician must be specially trained and aware of the possibility that IAQ factors may be the cause for illnesses and complaints, and be aware of the most probable manifestations of different aetiological factors.
- In cases with pulmonary symptoms with suspected indoor climate aetiology, it is recommended to add immunologic tests (e.g. intradermal, IgG, IgE, RIST, RAST) for the most common indoor allergens (e.g. mites, pets, moulds) to the standard lung function tests.
- Humidifier fever should always be suspected in recurrent cases of fever in environments with spray humidification.
- The uncommonly encountered pneumonia caused by *legionella pneumophila* should be suspected in persons with low resistance to infections who have been close to cooling towers or may have been ill after exposure to low temperature hot water aerosols, or who have resided in buildings where such sources may have contaminated HVAC system.
- It is recommended that radon and/or environmental tobacco smoke (ETS) be taken into consideration for preventive purposes as cause of pulmonary cancer in non-smoking persons living in high radon areas.

Biological tests for illnesses caused by bioaerosols

- People complaining of symptoms possibly related to building factors should be examined by a physician knowledgeable in this field.
- A number of tests are available but recommended only for stepwise use as some are extremely expensive and difficult to interpret.
- Due to the costs and loss of time in doing biological analysis, remedial actions are recommended as the most cost effective measure in the first place if a reservoir for contamination is identified by inspection.
- Laboratory tests are necessary in the diagnosis of *Legionella* infections and of other infections.
- Immunologic tests using environmental allergens and patient serum are recommended as most useful to verify exposure from moulds or thermophilic microorganisms in allergic alveolitis as well as for cases of humidifier fever. Immunologic tests are very informative and recommended also in patients with allergic asthma.
- Every time a building related illness is suspected the possibility of including biological environmental tests in the process of diagnosis should be kept in mind.

TABLE 35.9

Recommendations on the ascertainment of IAQ factors possibly responsible for building-related illnesses and complaints

- For clinical purposes the inspection and visual identification at the premises should be the primary step as it may be sufficient for supporting a preliminary clinical diagnosis.
- The verification of building related factors by objective methods as a cause of suspected building related illnesses should be aimed at confirming the aetiological diagnosis.
- The team approach with cooperation between medical and technical personnel is recommended in every case when it is necessary to find the origin of the most probable cause for the complaints.
- Analysis of volatile organic compounds (VOCs) is usually not recommended in the medical diagnosis of BRI due to the unspecificity and cost, but it is recommended in epidemiologic studies. If, however, VOCs are suspected to be the problem and there will be a follow-up after some changes are made, documentation of VOCs before and after remediations is recommended.
- The diagnosis of BRI should promote actions to avoid more cases of the same illnesses or complaints in the building.
- More investigations should be carried out on changes in the reporting of SBS-symptoms after correction of ventilation or other changes have been made.

TABLE 35.10

Recommendations on medical activities for treatment and prevention of building-related illness and complaints

- The medical doctor should inform the safety and health organization, industrial physician or other health authority in charge when building-related health effects are suspected.
- The investigation of building-related health effects should be a cooperative effort including medical and technical experts who are experienced in the dealing with IAQ-problems.
- In epidemic situations it is recommended to form an epidemiological task group, consisting of representatives from the organization for safety and health, the occupational health service, external expert, the maintenance engineer and management. It is recommended to contact the family doctor for information in both directions.
- A representative group of building occupants in each building should receive regular questionnaires relating to the symptoms of Sick Building Syndrome and environmental assessment. The average scores of these have been used to assess the proper function of the building services.
- In endemic situations, where complaints are present in an acceptable degree, it is recommended to avoid independent actions from consultants, sellers of a diversity of equipment and other "sharks" that will take opportunity to make own benefit.

TABLE 35.11
Recommendations on medical advice for the treatment and prevention of building-related illness

- The prevention of building-related complaints and illnesses should be taken care of at the earliest possible stage of building planning as the costs of remediation are far greater than the costs of proper planning and preventive measures.
- It is recommended that persons with a history of respiratory allergy should be specially cautious regarding excessive humidity that may favour growth of house dust mites and moulds in indoor environments.
- In cases of allergic symptoms proper advice must be given for control of allergens (from mites, mice, moulds and pets) mainly through environmental manipulation as well as for reducing the amount of mucous membrane irritants (formaldehyde, solvents, dust etc.).
- It is recommended that infection sources and mode of transmission be determinated and necessary medical treatment be provided where appropriate.
- In acute or recurrent episodes of fever one should primarily focus on local or central air humidifiers and air conditioners as the reservoir for microbial growth (gram-negative bacteria, algae, legionella).
- It is recommended that persons with hypersensitivity syndromes related to the interior environment be categorized and managed with proper care at an early stage of complaints, that the physical and psychological components be recognized and to actions taken for the removal of relevant sources of the hypersensitivity symptoms.
- It is recommended that research be encouraged to determine the basis of the Multiple Chemical Sensitivities/Chemical Hypersensitivity Syndromes.
- The medical treatment should be the same for BRI as for the corresponding illness due to other causes.
- The physician who makes the diagnosis of building-related illness should take action to prevent an extension of this illness in the indoor environment.

35.2.5 "Methods of Risk Assessment for the Indoor Environment", Kloster Banz, Germany, October 1991 (Seifert, 1991)

During the fifth meeting of the Pilot Study, experts reviewed current methods of risk assessment with special emphasis on indoor air pollutants. Participants agreed that it is difficult to use traditional risk assessment procedures with regard to indoor air pollutants because most developed procedures apply to the case of linear dose–response relationships between pollutant concentrations and health effects. However, non-linear relationships must be assumed for many of the important indoor air pollutants. Often, behavioural and social effects must be accounted for as well. This meeting gave participants an opportunity to discuss the problems associated with risk assessment procedures and identify existing gaps of knowledge in the field.

TABLE 35.12
Recommendations on technical measures to eliminate and/or prevent building-related illness and complaints

– Occupants should be given more opportunity to be informed on the building's IAQ design features and a mechanism for interacting with the HVAC (humidification, ventilation and air conditioning installation) operations staff. For example, ventilation criteria should be posted on walls in various areas (i.e. maximum occupancy in a room is a given number of persons if ventilation supply is to meet standards.)
– The adjustment of the thermal environment and securing cleanliness and mantained efficiency of the ventilation system are recommended as the first goal in preparing for less complaints from the inhabitants.
– Elimination of allergens and dust by standard cleaning procedures is the primary task in the prevention of allergic reactions to indoor air pollution.
– Radon prevention strategies should be implemented in all areas with soil rich in radon to reduce the radon exposure to a low risk level according to recommended limit values for residences.
– The building service management should be trained and supported by higher management and should have regular contact with the building occupants and responsibility to them and should co-operate with the occupational health service.
– Standards such as ASHRAE (7-10 L/s/p) are minimum rates, two to four times these rates are common in offices and should be considered for other spaces.
– The choice of low emitting materials is highly recommended to reduce the need for increased ventilation due to chemical contamination.

35.2.6 "Sampling and Analysis of Biocontaminants and Organics in Non-Industrial Indoor Environments", Chapel Hill, North Carolina, May 1992 (Pierson and Naugle, 1992)

The sixth Pilot Study meeting focused on issues related to sampling and analysis of biocontaminants and volatile organic compounds (VOCs) in non-industrial indoor environments. The sources of biocontaminants, VOCs, or other pollutants need to be identified and characterized in order to implement effective mitigation actions. The meeting consisted of two plenary sessions focusing on the sampling and analysis issues and VOCs, followed by a series of case studies of various measurements that have been used in the field.

35.2.7 "Indoor Air Quality for Health", Civitavecchia, Italy, September 1992 (Maroni et al., in press)

The final meeting of the Pilot Study dealt with the relationship between indoor air quality and human health. Participants at the last meeting also

discussed ideas for the content of the final report and a book on indoor air quality containing a synthesis of presentations and other material.

35.3 FINAL RECOMMENDATIONS OF THE PILOT STUDY

35.3.1 Government policy

Guidance / education

Because of the developing nature of understanding about indoor air quality, government activity regarding many IAQ topics has been focused on raising the awareness of various audiences. This education can take the form of (1) general information and (2) technical guidance and training that contains practical advice on minimizing indoor air pollution, where known. General information presented in the form of documents, videos, and other media can bring indoor air quality issues to the attention of the general public and building professionals. Special focus should be given to the design process, since it is here that professionals can work together to design buildings for acceptable indoor air quality. Targeted technical guidance and training should be provided for audiences that have an influence on indoor air quality or occupant health in various building environments:

- architects and mechanical designers;
- building owners and facility managers;
- home-builders;
- diagnosis and mitigation professionals; and
- physicians.

Research Support

Pollutant Source Characterization. Research on pollutant sources is needed to identify pollutants and emission levels from building materials and other products. Work in this area will serve two purposes:

- Providing measurement protocols and data for use in generally reducing exposures and;
- Providing correlations between research on health effects and pollutant sources of critical contaminant levels.

Health Effects. There are three areas where research is needed to significantly improve understanding of the health effects of indoor pollution:

- low level chemical exposure and pollutant mixtures;
- allergy/hypersensitivity; and

– multiple chemical sensitivity (MCS), also known as environmental illness.

The low levels of pollutants found in non-industrial indoor environments do not elicit health effects according to the same dose–response patterns as higher levels, such as those identified in occupational exposure limits. Research is needed to come to a better understanding of the mechanisms causing the health effects and the differences in response for different individuals or groups of individuals. Research is also needed to explore the effects of low-level exposure to mixtures of pollutants found in non-industrial environments.

Further research is needed to better understand hypersensitivity in individuals as it relates to allergy or other conditions and to determine if medical solutions to hypersensitive reactions can be found.

Finally, research is required to determine the definition and causes of, and solutions to multiple chemical sensitivity. Identifying the physiologic nature of MCS is the first step in understanding whether and how indoor air quality contributes to the syndrome.

Technology Development. Research is needed in the areas of IAQ diagnosis, mitigation, and control. Efforts in the areas of mitigation and control are required to provide economical and practical alternatives. Better means of measuring the effectiveness of ventilation systems are needed. While the ability to measure individual pollutants often exceeds the knowledge of health effects at measurable levels, progress is still needed in measurement of pollutant mixtures. Efforts are also needed to develop diagnostic tools that are sufficiently inexpensive and easy to use to be of help to facility personnel.

There is a particular need for improvement in the methods for assessment of the components of the airborne biological burden, including both the viable and total microorganisms. Research needs to be directed to the development of not only immunological and other methods for the reliable detection and quantification of specific organisms, or their allergens, but also techniques for the assessment of mycotoxins, fungal wall β-glucan and other microbial metabolites which may affect health via non-allergic mechanisms.

SBS and BRI. Efforts to identify causes and solutions to SBS and BRI should be supported. Research in health effects and building diagnostics combined with analyses of data compiled from building investigations are important to gain a better understanding of indoor air quality problems. Research into causes and solutions of IAQ problems should consider the interaction of the many factors that affect IAQ.

Problem assessment and surveys

Governments should support efforts to assess the extent of indoor air quality problems. These efforts will provide accurate information to use when

setting priorities for addressing public health problems.

Building Surveys/Epidemiology. Building surveys, such as those being conducted in the United States and elsewhere, are necessary to provide baseline information about building characteristics and pollutant levels. When combined with occupant health surveys, these studies will help to establish correlations between pollutant levels and health effects. Surveys should be conducted to identify building types or vintages in which problems occur more frequently. The results of these studies will support effective risk reduction programs. Epidemiology studies are needed to aid in differentiating between IAQ-related symptoms and those due to other causes. Moreover, epidemiology studies to assist in quantifying the extent of risk for indoor air pollutants are needed.

Economics. Economic research is needed to measure the costs to individuals, businesses, and society of indoor air pollution and IAQ control strategies. This research includes developing measures of productivity loss and increases in health cost due to indoor air pollution and costing various control strategies, including increasing ventilation, controlling pollutant sources, and air cleaning.

Standard/protocol development

Exposure Guidelines for Indoor Air Quality. Efforts should be made to ensure the protection of workers by setting reasonable exposure limits where the health effects of exposure to particular pollutants are known. In cases where research or risk assessment activities have yet to determine precise dose–response relationships, but where health effects are generally recognized, exposure limits should be set conservatively, weighing risk, economic impact, and feasibility. In addition, efforts should be made to develop exposure limits that recognize non-carcinogenic effects.

Building Codes. Building codes provide an opportunity to incorporate IAQ considerations into the design process. Efforts should be made to incorporate IAQ-related specifications in building codes. Code areas to target include ventilation design, building envelope design, site preparation, materials selection, and commissioning.

Ventilation Standards. Adequate ventilation of the occupied spaces with outside air is necessary to ensure good IAQ. Ventilation is most effective at improving IAQ when source control is practiced concurrently. Research and development are needed for a health-based ventilation standard, either from a consensus group or a governmental body. Encouraging code-setting bodies to adopt ventilation standards set by consensus organizations or governmental bodies will help pave the way for better IAQ in buildings in the future. Existing buildings may need retrofitting to conform to new ventilation standards, which

may prove prohibitively expensive. In such cases, voluntary standards can provide a target for buildings and market forces can ultimately determine whether retrofitting is warranted.

Maintenance Protocols. Efforts should be made to develop standards for maintaining HVAC systems and other maintenance activities that affect IAQ. Easily implementable guidelines for inspecting filters, scheduling pest control, and triggering testing and balancing (for instance, after renovation) would encourage more building professionals to adopt these practices. Regular inspection of a building's HVAC system should be encouraged to ensure proper operation.

Product Labeling. As an incentive to industries to develop and market lower emitting products, product labeling programs may be appropriate. This type of labeling should be done with the intent of providing information to consumers and building designers, not as a sign of safety approval. Because dose–response relationships for many volatile organic compounds are not known, labeling would best serve to achieve general reductions in emissions, rather than requiring manufacturers to meet specific guidelines, except for those cases where undesirable chemicals can be identified.

Accreditation. Efforts should be made to ensure that individuals who are diagnosing and mitigating IAQ problems are qualified. Because of the multi-disciplinary nature of the IAQ field, training should cover IAQ issues from multiple perspectives. By instituting a series of credentials that recognizes and highlights areas of expertise, consumers can be provided with information to make better informed choices when procuring IAQ services.

Emission Standards. Guidelines for product VOC emissions would provide useful information for manufacturers, architects, design engineers, building managers, and others who play a role in selecting products used indoors. However, development of such guidelines is dependent upon additional research establishing a health basis for them.

35.3.2 Design Considerations

Site

Site Investigation. Potential sites should be evaluated to determine whether they may be prone to indoor air quality problems. Sites should be investigated to determine past uses and any sources of contaminants which might remain as a result. Use of adjacent sites should be noted to evaluate the potential for outdoor pollutants being carried to the site.

Research should be done to determine if the site is in a high risk area for radon.

Site Preparation. Accumulation of moisture can be prevented by choosing dry and drained building sites and properly grading the property.

Building envelope design

Tightness. Buildings should be designed airtight to conserve energy and to improve control over infiltration of air and movement of pollutants. Tightness requires that adequate outside air be effectively delivered to occupants through the HVAC system. Natural ventilation should be encouraged whenever it is possible and convenient. Energy conservation can also be achieved by controlling internal loads (e.g., through increased use of natural light).

Ventilation

Outside Air Rates. Outdoor air requirements are calculated as part of the mechanical design process. Guidelines based on occupancy and space usage are important, however, outdoor air rates should also take into consideration the total indoor pollution load and the desired quality of air. Adequate outdoor air flows are important in residential as well as commercial properties.

Breathing Space. A consideration in HVAC design should be the amount of supply air and outside air that actually reaches the occupants of a building. This involves examining the method and efficiency of air distribution. The effectiveness of the HVAC system to dilute and remove indoor pollutants and properly distribute outside air throughout the building is an important aspect of the design. Ventilation rates should be reevaluated when interior spaces are renovated.

Mechanical Ventilation in Houses. Some houses are built tight and adequate outside air does not enter through infiltration. As a result, mechanical ventilation is necessary to introduce a satisfactory flow of outside air and provide adequate dilution and removal of pollutants. This makes the heat recovery from the ventilation air also possible.

Commissioning

New buildings should be commissioned before occupancy. Commissioning should include testing and balancing of the HVAC system, documentation of the system to meet needs during both operations and potential renovations, establishing responsibilities for maintaining and operating the system, and training of staff responsible for operating and maintaining the system. In addition, commissioning should include ventilation specifications for use while the building is new to reduce elevated levels of VOCS.

Material selection

Designers should build in better indoor air quality by specifying building materials that are minimal pollutant sources. These materials include low-emitting products and materials with low fleece factor. Designers should work with manufacturers, if necessary, to obtain these materials. In addition, designers should work to minimize horizontal surfaces on interior finishes and furnishings to reduce the particulate levels in buildings.

Combustion Appliances

Designers and builders should specify and install combustion appliances according to manufacturers' specifications, paying special attention to requirements for combustion air and exhaust venting.

35.3.3 Indoor air pollution control

Management of pollutant sources

Biological Contaminants

Moisture Control. Biological contaminants will flourish wherever there is adequate moisture because adequate nourishment is always available on building surfaces. Because so many building materials can serve as a nutrient source for moulds and other biological contaminants, the most practical means for controlling biological contamination is by avoiding excess moisture wherever possible. Moisture control can be accomplished by dehumidification, ventilation, and increasing the temperature at the building surfaces to prevent condensation. These techniques may be used individually or in combination.

Dehumidification is most important in humid climates. Maintaining a sufficiently warm temperature at interior building surfaces is important in cooler climates and requires proper insulation, including insulated windows. Ventilation can aid in moisture control by increasing air movement.

Maintenance. Biological contamination can be avoided both by sustaining high quality maintenance and monitoring the materials and procedures used in operating and maintaining the building components, including the HVAC system. Proper maintenance of HVAC equipment is critical to prevent microbiological growth and the entry of undesired microbiologicals into the indoor air stream. These components include drainage pans, coils, cooling towers, ductwork, and humidifiers. Poor filter maintenance is a common problem that can lead to microbial growth, which acts as a source of fungal spores, bacterial and other biological particles which can be distributed within the building.

Building management should develop routine maintenance schedules that include filter checking and replacement and drain pan cleaning.

Volatile Organic Compounds (VOCs)

*Source control.*The level of volatile organic compounds within a building can be controlled through careful selection of building materials and products. Building managers should become familiar with the volatile organic compounds found in building components and products used in the building. Designers and building managers should attempt to select the safest, least toxic materials, when they can be identified, or those with the lowest emission rates. Information regarding volatile organic compounds may be found by reviewing product labels, Material Safety Data Sheets (MSDS), and available compendia (e.g. the American Institute of Architects Environmental Resource Guide).

Temporary ventilation after installation / bake out. Off-gassing of VOCs is greatest when VOC-containing materials are new and decreases over time. For building materials, off-gassing is greatest immediately after the installation of VOC sources. In order to minimize occupant exposure to volatile organic compounds, areas that have had new materials installed or have undergone renovation should receive increased outdoor air ventilation and/or local exhaust. In the initial months after building completion, the ventilation should operate 24 h/day and 7 days/week. Installation of new products or renovation work should preferably occur when the space is unoccupied and will remain unoccupied until the strongest VOC off-gassing has occurred. In unoccupied buildings, designers should consider using bake-outs to accelerate the off-gassing of VOCs. Bake-out involves raising the temperature significantly to increase off-gassing and increasing ventilation to remove VOCs from the space.

Use of air cleaning to remove VOCs. Air cleaning is not recommended as a substitute for source control and adequate ventilation for removing VOCs. VOCs can be removed by air cleaners relying on adsorption and absorption methods, however care must be taken to avoid re-emission of the collected VOCs from the filter medium.

Other Pollutants

Radon. In order to prevent the migration of radon gas into a structure, any cracks or openings in the foundation of the lowest level of that structure should be tightly sealed. Ventilation can be introduced to the lowest level in order to dilute and remove the gas. Exhaust fans and piping can create sub-slab depressurization to remove radon and deter it from accumulating in the building.

Combustion Gases. Combustion gases should be prevented from entering occupied spaces. Outdoor air intakes for a building should not be located near exhaust systems or other sources of combustion gases, such as highways. Combustion appliances are also a source of combustion gases within a building and should be used and vented in accordance with the manufacturers' recommendations. Adequate general ventilation air and exhausts for the appliances should be provided to minimize occupants' exposure.

Particulates. Particulates, including asbestos, ETS particles, dust and pollen, are hazardous or troublesome to occupants when they become airborne. Proper housekeeping practices should be followed to keep dust levels low. Housekeeping activities should be done during the off-peak hours to minimize effects on sensitive occupants. High efficiency filtration in the air handling system can also help reduce airborne particulate levels.

Asbestos products should be avoided. When asbestos products are identified in existing buildings, the general recommendation is to manage those materials which are non-friable or friable, yet in good condition, in place. This involves minimizing disturbance of the material and training in-house staff on emergency maintenance handling procedures.

ETS. In order to eliminate exposure of nonsmokers to ETS, buildings should institute smoking bans or provide enclosed, separately ventilated, negatively pressurized smoking rooms with direct external exhaust. These smoking rooms should provide a volume of outdoor air per smoker to meet ASHRAE or other more stringent standards.

Operation and maintenance of ventilation systems

Training of Facility Staff. Building maintenance personnel should be trained to understand the indoor air quality aspects of their work. Many maintenance activities directly affect indoor air quality, and some may reveal indicators of potential problems. The staff should be made aware of indoor air quality considerations and how their work can directly impact the health and comfort of occupants.

Maintenance Logs and Scheduled Filter Changes. Preventive maintenance of a HVAC system is essential for it to operate correctly and provide suitable comfort conditions and good indoor air quality. Detailed maintenance logs should be kept for all equipment, including controls and filters. A scheduled program should be developed for a routine check of equipment, calibration of control system components, and necessary filter replacements.

Design Versus Actual Occupancy and Use. Space is often used for other purposes than those originally intended, especially in older buildings. Changes, such as increased occupant density or altered function of the space

can affect the required outdoor air supply to the space, the necessary exhaust from the space, and consequently the indoor air quality. Design guidelines with respect to outdoor air requirements, may differ drastically from the time of initial design to the time of renovation or space use change. When space is reallocated, renovated, or changed from the original design, an engineer should be consulted to examine the use of the space and determine if adjustments to the HVAC system are warranted. The same procedure is required when new sources of contaminants are introduced.

Air cleaning

Filter Types. Depending on the pollutants of interest, four technologies should be considered for removing contaminants from the air: particulate filtration, electrostatic precipitation, negative ion generation and gas sorption. The first three are devised for the removal of particulate matter, while the fourth is designed to remove gases.

Use of Air Cleaning for Controlling Indoor Pollutants. Air cleaning as a method of indoor air quality control is most effective when used in conjunction with source control and adequate ventilation. Most air cleaning in large buildings is directed primarily at preventing contaminant accumulation in HVAC equipment and enhancing equipment efficiency. There are a variety of filter types even within the four categories.

Filtration is effective only when properly installed and maintained. It is important that filters be changed or cleaned on a regular basis and that leakage around the filters be minimized. High efficiency filtration is most effective at improving indoor air quality.

Indoor Air Quality
M. Maroni, B. Seifert and T. Lindvall (Eds.)

Chapter 36

The Nordic Committee on Building Regulations Guidelines for Indoor Climate and Air Quality[1]

36.1 INTRODUCTION

Since May 1981, NKB Report No 41, "Indoor Climate", has served as a basis to which national regulations have conformed at different rates. Since the publication of the report, developments mainly in the area of air quality have necessitated a revision of this report.

New hygienic problems associated with the built environment have emerged at an accelerating rate. Health problems coupled with the incidence of radon, formaldehyde, mould and diffuse symptoms of an irritational type, the sick building syndrome, occur. There is also an increase in allergies and other hypersensitivity reactions which are traced back to a deterioration in indoor environments, for instance an increased incidence of irritants, house dust mites, mould, bacteria and viruses (Ministry of Health and Social Affairs, 1989). It is difficult to estimate the scope of this problem since there have been, on the whole, few studies. A group of experts in WHO (Report of WHO Meeting, 1989), for instance, has estimated that between 10 and 30% of new or renovated buildings are sick buildings.

A few studies on a larger number of buildings are available. Investigations of offices in Denmark (Skov et al., 1989) and Britain (Wilson et al., 1987) show that many factors are significant for the health problems which have been reported. Sex, psychosocial conditions, social status, work content and different forms of stress are involved in the discomfort experienced by the individual. However, when account has been taken of such factors, there remain considerable differences between buildings as regards reported health problems. The building has an essential role as the cause of human discomfort.

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1 This chapter integrally reports the Nordic Committee on Building Regulation guidelines for indoor climate and air quality, as published by The Nordic Committee on Building Regulations NKB, NKB Publication No. 61E, June 1991.

Developments regarding building technology, building materials and energy technology have been rapid in recent decades. From a construction process characterised by a building season (spring–summer–autumn), long building times, manual craftsmanship, small scales and "natural" materials, development has progressed to a fast, more industrialised, year-round construction with new technology and synthetic materials. Work on quality assurance in construction has not kept pace with this otherwise rapid development. New technology and new materials have often gained ready acceptance since they conferred evident technical and economic advantages. There has been no prior control regarding building hygiene aspects. A similar development has taken place as regards materials for fixtures and fittings, furnishings and furniture and consumer articles. Many new types of pollutants are therefore released into the indoor air.

To this pollutant configuration must be added contaminants given off by people — cooking, tobacco smoking, open fires — and by, e.g., mould, bacteria and house dust mites. The Allergy Enquiry (Ministry of Health and Social Affairs, 1989) in Sweden has pointed out that housekeeping and cleaning have been neglected in the society of today, and this also influences the pollution configuration indoors.

Over the same period, ventilation has been reduced as a consequence of the need for energy management and other factors (Swedish Council for Building Research, 1987). Buildings have become more airtight, with less infiltration/exfiltration as a result. Satisfactory performance of the controlled ventilation is therefore of greater importance now. Experiences gained in several sick building investigations and studies of the performance of ventilation (see e.g. Allhammar et al., 1985; Nielsen et al., 1989) show that many installations do not work as intended.

There is greater knowledge today of the pollution situation indoors. Principally, however, research has been of a survey character and not the kind that permits conclusions to be drawn as to which factors have significance regarding the incidence of ill health. At the present time it is therefore not possible, other than in exceptional cases, to pinpoint "guilty" substances or factors.

In the light of the uncertain state of knowledge, our philosophy is of a general nature. That is to say, what we must do is not only avoid certain substances, factors, but generally to keep the number and intensity of pollution sources at a low level and also to secure a reasonable ventilation standard. The strategy is based on classical elimination techniques, i.e. measures shall be primarily directed at the sources by replacing high emitting processes by low emitting ones, materials or similar. Secondarily, the sources shall be tackled by measures of the encapsulation and local extraction type. When these primary measures do not create a satisfactory air quality, background

ventilation must be increased.

Hygienic conditions in a building depend not only on how the building has been constructed but also on how it is operated and maintained. In order that these aspects also should be covered, the report contains proposals which affect legislation other than conventional building legislation, e.g., working, environmental, occupational safety, and health and safety legislation.

The report deals with the factors and proposes the requirements which have been considered essential for the health of a building. The proposals have been arrived at through an aggregate assessment of experience and scientific documentation. The text contains references to literature listed at the end of the report. Where no references have been available, an assessment has been made on the basis of experience.

The report examines those aspects which directly affect the quality of air indoors in premises other than industrial ones.

Additional guidelines regarding the construction of installations are given in: NKB Publication No. 52, Mechanical Ventilation Installations, Guidelines (Nordic Committee on Building Regulations, 1984); thermal internal climate in NKB Publication No. 40, Internal Climate; and acoustic conditions in NKB Publication No. 32, Guidelines for Building Regulations regarding Acoustic Conditions (Nordic Committee on Building Regulations, 1978).

The report is set out so that each section begins with an introduction which deals with documentation and background. This is followed by recommendations for regulations and guidelines regarding the way in which these can be complied with. Proposals for requirements are formulated with the wording "shall", and guidelines with the wording "should" or merely as informational text.

For the definitions of ventilation and other terms, see Appendix No. I[1] and NKB Publication No. 52, Mechanical Ventilation Installations, Guidelines (NKB Publication, 1984).

36.2 OVERALL OBJECTIVES

Everybody spends most of his or her life indoors. In the home one must rest, sleep and regain one's energy. Known negative effects on health or the risk of these cannot therefore be accepted. The causative relationship between certain pollutants and their effects on health is relatively well known. For instance, WHO has compiled a dose–effect relationship for a number of substances and laid down critical threshold limits. However, the number of potentially harmful or troublesome substances indoors is much greater than this. Furthermore,

1 Appendix I is not reproduced in this book. The reader should consult the original document.

little is known about interaction effects. It is therefore difficult to lay down specific quantified requirements of practical applicability for normal pollution situations indoors. Consideration of sensitive groups (including babies, allergic and other hypersensitive persons, the sick) makes the position even more difficult.

The air indoors consists of air from the outside with pollutants from, e.g., traffic, the ground and industries. During its passage through the supply air system, air may be contaminated by particles, fibres and substances from components such as filters, internal insulation and deposited dirt. When moisture is present in the system, biological growth of, e.g., mould and bacteria may also take place, with contamination of the supply air. Inside the building, there is further pollution released by people and animals, and dirt from activities such as tobacco smoking, cleaning and cooking. To this must be added contaminants from open fires, building materials, fittings, fixtures, furnishings and furniture, office machinery, hobby and cleansing products, etc.

Industrial hygiene threshold limit values or fractions of these are not relevant in this context. These refer to fully healthy adult persons and 8 hours exposure per day. In addition, they have been laid down for situations where the exposure is only to one or a few pollutants. Compared with such threshold limit values, the contents in these environments are low. On the other hand, however, the contents are often high compared with guidelines for the external environment. If the contents indoors and outdoors are compared, the situation is largely as follows:

Particles. Contents are normally equal indoors and outdoors. In conjunction with tobacco smoking, vacuum cleaning or similar, contents are elevated indoors, often by a factor of 10 or more. Radon daughter products, and also radon, normally occur in higher — often considerably higher — concentrations indoors than outdoors. Particles of biological origin such as viruses and bacteria, or of mould and house dust mites, occur in higher concentrations indoors than outdoors if there are internal sources. Such sources are normally present for viruses and bacteria in the form of infected persons. Indoor proliferation of mould, bacteria and house dust mites is of normal occurrence.

Inorganic gases. Contents indoors are normally somewhat lower than, or at the same level as, outdoors. Where there are open fires or tobacco smoking, the contents are higher indoors.

Volatile organic substances. Normally, contents are 2–10 times higher indoors than outdoors. In a newly repainted or completely new building, the contents indoors are often 100 times higher.

Characterisation of the pollutant content of indoor air has so far concentrated on the pollutants and pollutant types to be found in industry or in outdoor air, or on those for which measuring methods had been developed, i.e.

for total dust, inorganic gases and VOC (Volatile Organic Compounds). Most is still not clear. What VVOC (Very Volatile Organic Compounds), SVOC (Semi Volatile Organic Compounds) and POM (Particulate Organic Matter) contents occur? What significance do these have for the health problems encountered? As far as dust is concerned, there is generally no answer to these questions. What is relevant regarding the health of persons, total quantity, the number of particles, their shape, surface, certain size fractions, what the particles consist of, their hygroscopic properties, the contaminants carried by the particles, or what?

There are very few risk estimates with regard to common complaints or symptoms such as irritation of the mucous membranes or skin, or serious diseases such as asthma as a result of airborne pollutants indoors. The same applies for other allergic or hyper-reactive diseases. There is evidence that the incidence of respiratory tract symptoms is generally affected to a high degree by the indoor climate and the airborne pollutants indoors (Ministry of Health and Social Affairs, 1989; Andreas et al., 1988; Åberg, 1988; Bakke, 1989; Johansson, 1990).

A group of experts in WHO (WHO/EURO, 1987) has discussed criteria for the evaluation of the effects of individual organic pollutants in the indoor environment. In its recommendations, the group of experts states, *inter alia*, that the maximum content of an individual substance shall be such as to cause an olfactory sensation in not more than 50% of persons on short-term exposure to air containing the substance, for instance in a visit situation. These values refer to a "normal population" and to an "average situation".

Examples of recommended guidance values for individual substances are given in the WHO Air Quality Guidelines for Europe (WHO/EURO, 1987) which are partly reproduced in Appendix No. II[1]. In all premises a person is at all times exposed to several substances and factors simultaneously. The knowledge is not available to carry out an overall assessment of such a multifactorial low dosage exposure. In the report referred to above, it is stated that "data are at present insufficient for guidelines relating to mixtures to be laid down" (WHO/EURO, 1987). The above criteria for individual substances are therefore not readily applicable.

Criteria for exposure of a composite nature such as e.g. the ASHRAE requirement regarding 80% acceptability (ASHRAE, 1986) cannot be easily compared with the above criteria for individual substances. This is however the most common way in which acceptability is quoted. Acceptability, which is initially generally based on a "first hand impression" when a person enters a room, is a measure of odour impressions and immediate irritation.

1 Appendix II is not reproduced in this book. The reader should consult the original document.

Measurements of acceptability over longer exposure (minutes) show that adaptation normally occurs so that acceptability increases with the period of stay (Gunnarsen, 1990). Other sensory impressions based on exposure over a long period (hours), such as e.g. the perception of "dryness", may provide more relevant information regarding the quality of air with respect to health effects.

The conclusion of the above is that, on the whole, knowledge is not available to lay down quantified criteria for risk assessments regarding the quality of indoor air. For a few substances guidelines have been given in the WHO document (WHO/EURO, 1987), see Appendix No. II, but for the great majority of substances and particularly for a number of substances in interaction, knowledge is insufficient.

The same applies on the biological side. Threshold limit values have been proposed (Public Health Department of the Ministry Social Affairs, 1990) for bacteria (Nevalainen, 1989), mould (Holmberg, 1987) and house dust mites (Platts-Mills, 1987). Further investigations are however required before such threshold limit values are introduced.

Since the state of knowledge does not permit the establishment of threshold limit values based on quantified risk assessments, other than for a few substances, the following recommendation is instead given for overall regulations:

> Buildings inclusive of their installations shall be planned, designed, constructed, maintained and operated so that satisfactory comfort is achieved with regard to air quality and so that danger to health does not arise when rooms are used in the way intended.

The quality of air is considered satisfactory if the great majority of visitors, on entry into the room, perceive the air as acceptable (do not express displeasure), if the air does not cause irritation to skin, mucous membranes or airways, not even in persons who are somewhat more sensitive than normal, if there is no risk of sensitisation and if the risk of health effects after long term exposure is negligible. Nor must the quality of air give rise to disease.

The term "used in the way intended" implies, for instance, that smoking occurs in rooms where tobacco smoking forms part of the design conditions. There must at all times be a margin for short term pollution loads, for instance in the form of openable windows. In dwellings it shall be possible for e.g. hobbies, housekeeping, cleaning etc to be carried out to the normal extent without this causing inconvenience.

In assessing buildings in operation, the experiences and complaints of people can be used in judging the "health" of the building, but other methods are also needed.

36.3 REQUIREMENTS FOR BUILDINGS, FITTINGS AND FIXTURES, FURNISHINGS AND FURNITURE, AND ACTIVITIES IN THE BUILDING

36.3.1 Planning

36.3.1.1 Basic conditions

Experience shows that, broadly speaking, all types of ground can be built on with good results. However, there is a greater risk of the incidence of problems if buildings are constructed on ground that is waterlogged, has a high radon content or may be contaminated, for instance as a result of earlier industry activity (Lindvall et al., 1987). There is little risk if older construction experience is applied, such as that a building should be placed high and dry and that the ground should slope away from the house so that surface water is drained away. Moisture and mould damage as a result of damp from the ground are closely associated with lack of knowledge and defective quality assurance when ground of lesser suitability is chosen.

In certain areas drinking water from deep bored wells may emit radon to the indoor air. In view of this, the following recommendation is made for regulations:

> The choice of building construction shall be made with regard to the nature of the ground so that problems connected with the indoor climate do not arise. Particular account shall be taken of damp and radon. When deep wells are sunk for drinking water in rock of high radium content, the radon content of the water shall be analysed. When a building is constructed on ground contaminated by waste or industrial activity, the pollution source shall be removed or the building shall be constructed so that the pollutants cannot enter.

The ground should be investigated with respect to local groundwater levels, pockets of water, water-bearing strata and the risk of flooding. Construction on abnormally damp ground imposes greater requirements concerning design and construction in order that moisture damage and problems with air quality may be avoided. The radon risk should also be assessed.

The radon content and permeability to air of the ground imposes requirements regarding the airtightness of the building and/or a mechanical ventilation system inside or outside the building as protection against entry of radon. In order that the negative pressure created by the ventilation system in habitable rooms may be kept at a low level, sufficient facilities should be provided for the supply of replacement air.

36.3.1.2 Quality of outdoor air

Pollutants from the outside may be a significant risk factor. In investigations of sick buildings in the USA, it has been found that intake of polluted air from the outside is a not uncommon cause of health problems (NIOSH, 1987). In view of this, the following recommendation is made for regulations:

> In order to secure a satisfactory indoor environment, a building shall be placed in as pure an outdoor environment as possible. The building and its ventilation system shall be designed in view of the quality of outdoor air.

Construction near traffic routes, in town centres or near industries or similar imposes greater requirements on the placing, orientation and design of the building and the planning of its ventilation system. In such cases systems in which air is taken in directly via openings in the building shell should be avoided. It may be necessary to make a survey of the prevailing conditions and to try and evaluate future conditions. These aspects should be take into consideration in e.g. the planning of new housing estates.

Regulations for the outdoor air exist in, e.g., Finland (Minister for Air Quality, 1984) and Sweden (National Swedish Environmental Protection Agency, 1990). See also Appendix No. II for individual guideline values according to WHO (WHO/EURO, 1987).

With regard to the siting of the outdoor air intake, see Clause 4.2.[1]

36.3.2 The design of buildings

The risk of the spread of pollutants can be modified by the design and layout of the building. Open stairways or large rooms with varied activities involve the risk of the spread of pollution. The same applies, on the whole, to a mixture of polluting and non-polluting activities in the same building.

In view of this, the following recommendation is made for regulations:

> A building shall be designed with regard to the pollution which may arise. The ventilation shall be planned so that the risk of the spread of pollution in the building is kept low.

Premises in which there is a large difference in the expected degree of pollution should be separated from one another, for instance as regards both construction and ventilation. They should preferably be located in different buildings.

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1 The clause number is obtained by removing the chapter prefix (36) from the numbered section headings

36.3.3 Construction

Moisture damage often occurs in buildings with flat roofs and with foundation slabs placed directly on the ground. Mould damage often occurs in foundation structures of the type slab laid directly on the ground with insulation on top, ground floors above crawling spaces, and basement walls with internal insulation. On flat roofs even a small leak will give rise to extensive consequential damage.

For instance, problems are often encountered due to icing and cracking in climates with low temperatures and snow. The cause of these problems may be bad planning, defective construction or poor maintenance. As far as damage in general is concerned, neither designers nor others responsible for the construction process have much knowledge of building physics (see, e.g, Samuelsson, 1985).

Leaks from installations can give rise to extensive indirect problems such as a mould growth or chemical degradation of building materials, with deterioration in the quality of air as a consequence.

In view of this, the following recommendations are made for regulations:

> The building structure shall be arranged so that building materials are not exposed to action such that harmful emission of pollutants to the indoor air occurs. The building structure shall be selected, constructed and maintained so that it is satisfactorily dry with respect to the materials used. The structure shall be arranged so that harmful spread of pollution from the outside, the ground, another flat or some other separate part of the building does not occur.

Water supply, drainage and heating installations shall be planned and constructed in such a way that the risk of leaks and consequential damage caused by leaks are prevented, so that negative health effects do not arise.

Materials in structures shall be selected with regard to the temperatures and moisture contents to which they will be exposed.

Structures shall be designed so that harmful ingress of water and moisture (driving rain, snow, moisture from the ground) cannot occur and so that moisture in materials is removed. Roofs shall be designed so that water is drained. Account should be taken of local snow and ice conditions.

Thermal insulation and weathertight layers shall be arranged so that the risk of damage is prevented.

Foundation structures of the type slab laid directly on the ground require particular care in design and construction, for instance with regard to the placing of thermal insulation and waterproofing and the quality of the anti-capillary layer.

Foundation structures of the type where the ground floor construction is above a crawling space are susceptible to moisture damage and should be used only on dry ground and provided with effective ventilation. The materials should be resistant to mould, bacteria and rot.

In rooms where there is a great risk of water leaks there should be either floor gulleys or the floor covering should be arranged so that any leaks can be seen. In bathrooms and similar the floor shall slope towards the floor gulley.

Pipes should be laid so that they are readily accessible for inspection and repair, and arranged so that any leaks can be seen at an early stage.

Since the pressures in different parts of a building, or between the building and the ground, may be different due e.g. to thermal forces, wind or the function of the ventilation system, the structure should be sufficiently airtight with regard to prevailing conditions. The same applies to points where installations penetrate elements of structure. These should be sealed with material resistant to ageing.

During the construction period there should be in existence a quality assurance programme which ensures that unnecessary moisture is not incorporated in the structure. Many problems can be avoided by careful handling of building and building services materials (e.g. by ensuring that materials sensitive to moisture are always covered in the event of rain) and by continuous tests and checks of the moisture content of materials prior to their incorporation in the structure.

36.3.4 Building materials and surface finishes

From the point of view of air hygiene, the aim is that materials in buildings shall not impose extra requirements on ventilation. Ideally, there would then be no need to provide ventilation to remove gases emitted by building materials, fixtures and fittings, furnishings and furniture. At the present day, as a result of lack of knowledge of what materials emit and the significance of these pollutants, this is not realistic. The first step therefore is to choose materials which emit the smallest possible quantities of pollutants. Apart from their own ability to emit pollutants, surface finishes can act as storage areas for pollutants, particulates, gases and vapours from other sources. This storage effect, which may be both positive and negative, is dependent, *inter alia*, on the surface area of the material. For instance, a fleecy surface has a relatively larger storage effect than a smooth one.

Uncritical use of new and hygienically untested materials, and faulty handling of materials, primarily handling that increases moisture load, is a significant cause of sick buildings. One example of this is provided by the chipboards and glued timber structures of the seventies which could emit a lot

of formaldehyde, especially in conjunction with moisture. Another is screeds containing casein from the period 1977–1984 which, in combination with moisture, could give rise to chemical reactions that resulted in significant emission of pollutants. The moisture in this case came from damp concrete or thick courses of screed which were not given enough time to dry out before the floor covering or carpet was laid. Other examples are acoustic tiles of loosely compacted mineral wool, certain paints, and adhesives (see, e.g., Danish Association for Materials Testing and Research, 1988; Ericsson et al., 1984; Gustafsson et al., 1985; Nielsen et al., 1989; Gustafsson, 1990).

Emissions from materials arise from (Ministry of Health and Social Affairs, 1989) solvent residues, (WHO, 1986) remnants of raw materials (e.g. monomers) (Skov, 1989) reaction and decomposition products from the manufacturing process, and additives (Wilson, 1987). The emissions due to the first three factors are greatest in new materials. Within 1–6 months, the emission decreases asymptotically to a level which is characteristic for the material and the type of emission.

Pollutants from materials can in many cases be detected by their odour. Odours in indoor air are therefore a good preliminary indicator of the quality of air in this respect. There is however considerable variation in the odour threshold of pollutants. There are substances which are harmful at levels below the odour threshold, and odour alone cannot therefore be relied upon.

Apart from radon and substances of biological character, it is mainly the volatile organic compounds which distinguish indoor air from outdoor air. These, which often originate from building materials, fixtures and fittings, furnishings and furniture, may be grouped as follows depending on their boiling point (WHO, 1989):

VVOC	very volatile	<0 to 50–100°C
VOC	volatile	50–100 to 240–260°C
SVOC	semivolatile	240–260 to 380-400°C
POM	particulate	<380°C

The more volatile a substance, the faster it is released from its source, e.g., a surface finish. The emission can be at a high level for a short time but will drop after a few weeks or months to normal level. Such emission can be accelerated by increased ventilation, possibly combined with short-term raising of temperature ("outbaking").

Substances with a higher boiling point, which are less volatile, are released at a slower rate. The emission lasts a long time and cannot be accelerated by increased ventilation. It is a characteristic of such substances that they are prone to be absorbed by other materials, especially materials of large specific surface such as textiles.

Measurements of volatile organic substances indoors have mainly concentrated on VOC. Owing to the large variations in the way samples are taken and analysed, it is difficult to compare values measured by different institutions. The measured values of such substances are normally within the following ranges (De Bortoli, 1985; Krause, 1987; Seifert, 1987; Wallace et al., 1987; Jungers et al., 1987; Wolkoff, 1987). For purposes of comparison, the concentrations of airborne dust are also given.

	VOC mg/m^3	Dust mg/m^3
Outdoor air	0.01–0.04	0.005–0.03
Dwellings	0.05–0.4	0.001–0.1
Offices	0.05–1.3	0.1–0.2
Schools, nurseries day		0.05–0.3

A preliminary critical scrutiny of the allergenic properties of sub-stances contained in building materials has been made by the Danish Toxicological Centre (Danish Toxicological Centre, 1989). This is based on the substances included in the Danish Ministry of the Environment list of hazardous substances classified as R42 (may cause allergy on inhalation) or R43 (may cause allergy on skin contact) and a number of other substances which are used in building materials, although far from all. It has been possible to classify a large number of substances as presumably contact allergenic but only a few as allergenic on inhalation. It is stated in conclusion that available knowledge of allergenic properties is often unsatisfactory and that several examples of misclassification have been found. It is stated that this underlines the obligation on the part of manufacturers and importers to produce information themselves and to evaluate the hazard attached to substances and materials. The report states that substances contained in curing systems such as isocyanates, epoxy products, acrylates and amines may be allergenic in their pure form but that after curing they seldom pose any risk of allergy. For a number of substances in general use there is no allergenic effect described in the literature, but by reasoning on the basis of analogy they may nevertheless be presumed to have an allergenic effect.

The volatility of substances plays an important part in the occurrence of the hazard. Highly volatile substances are emitted over a short time, hours or days, while substances of low volatility may take a long time, months or years, to be released. In complex material structures such as sandwich units, foam and jointing compounds, the volatile substances may be held back and emitted over a long period. One common example of wrong use is to expose the material to a high moisture load (Ministry of Health and Social Affairs, 1989). Degradation of materials in a moist environment is therefore a serious problem.

As regards the incidence of radon indoors, building materials can be an essential factor in addition to the soil. Building materials of elevated radon emission have not appeared in the market, other than in exceptional cases, since manufacture of alum shale based lightweight concrete was discontinued in 1975. New materials may however be introduced unless the risk is kept in mind.

In view of this, the following recommendation is made for regulations:

> Building materials and surface finishes shall have the lowest possible emission properties. They shall be manufactured, selected, handled, stored and used so that emission to the room air is the least possible. The material shall be able to stand up to the intended use. The material shall not contain any genotoxic or neurotoxic substances, substances which cause sensitisation or irritation of the mucous membranes or substances harmful in any other way, which pose a health hazard when used as intended in the building.

Intended use includes, e.g., cleaning.

Only materials which have low emission properties and little odour, have been tried and tested, are accompanied by a product description statement and have been subjected to a quality assurance programme, should be used. The client should demand an internal climate related product description of the material. The description may contain data regarding the content of substances in the material, and emission, which affect air quality and may have health consequences or have an effect on comfort, and regarding the intended area of use. Such descriptions should be drawn up by the material manufacturer and importer.

In producing materials, the following should be complied with:

- Only known and tested raw materials should be used, and they should be selected so that they do not carry emitting substances into the material.
- The manufacturing process should be well known and its critical parameters monitored so that unreacted monomer residues are not left in the material and so that decomposition does not occur in the material during the process.
- The components of the material should be correctly selected in view of the intended use of the material, for instance damp conditions. Such components shall not decompose into volatile constituents even when exposed to extreme but possible moisture and temperature conditions during their intended use.
- Additives should be selected so that they are durable in the material and are not lost by emission.

In addition, it is essential that production should be subjected to a well developed quality assurance programme.

Stone, brick, timber, gypsum and high pressure laminates are examples of materials which normally have low emission properties. Materials which may emit gases to an annoying extent over a long period should be avoided. Adhesives, fillers and jointing compounds should therefore be applied in the thinnest possible layers. Moisture sensitive materials should not be exposed to rain or snow, nor should they be incorporated in structures where moisture loading is likely. Materials must in addition be used in the intended manner and, for instance, given sufficient time to dry out before being built in. Before a building is taken into use, it is essential that the materials (e.g. surfaces painted in situ) are given sufficient time for emission to occur. Well tried materials accompanied by a product description should be used in the first place.

Thermal insulation materials should be placed inside the structure or provided with surface finish such that harmful emission of fibres, fire protection chemicals, etc. to the room air is prevented. Mineral fibre products should be designed or incorporated so that release of fibres into the room air is prevented.

Surface finishes in a room should have the lowest possible specific area in order to prevent undesirable storage effects.

36.3.5 Fittings, fixtures, furnishings and furniture

Fittings, fixtures, furnishings and furniture are often just as significant a source of pollution as building materials. See also the background text to Clause 3.4. In view of this, the following recommendation is made for regulations:

> Emission into the room air by fittings, fixtures, furnishings and furniture shall be as low as possible. The materials shall stand up to the intended use. Materials shall not contain any genotoxic or neurotoxic substances, substances which cause sensitisation or irritation of the mucous membranes, or substances harmful in any other way, which pose a health hazard when used as intended in the building.

Fittings, fixtures, furnishings and furniture should be selected, handled and used so that emission to the room air is the least possible. Intended use includes, for instance, cleaning.

Only materials which have little odour, have been tried and tested and are accompanied by a product description statement, should be used for fittings and fixtures.

36.3.6 Processes, activities and handling

In many premises and contexts, processes, activities and handling are dominant pollution sources. One common source is tobacco smoking. In contrast to sources such as building materials, fittings and fixtures, they are often intermittent and mobile (Sundell et al., 1985). Measures to reduce emission to the room air are therefore to be preferred to general measures of the background ventilation type. There is also experience from industry that all polluting processes can be replaced by ones that produce less pollution, can be carried out at times and in places so that few people are affected, can be encapsulated or treated at the source by e.g. local extraction or similar. These experiences should be applied to a greater extent in the premises referred to here, for instance as regards photocopiers and laser printers. In dwellings also exposure to pollutants as a result of e.g. hobby activities should be mainly counteracted by reducing the intensity of source. Consumer, play and hobby products should therefore be controlled and marked in this respect.

In view of this, the following recommendation is made for regulations:

> Processes, activities and handling shall be selected so that they have the lowest possible emission. Where processes, activities and handling which pollute the air are necessary, they shall as far as possible be encapsulated, provided with local extraction, carried out in premises with separate ventilation to the external air, and limited to times when few people are exposed. Where tobacco smoking occurs special measures shall be taken to prevent the risk of passive smoking.

In view of the risk of passive smoking, tobacco smoking indoors should be limited. It is appropriate to provide special smoking rooms with separate ventilation to the external air.

Appliances which emit pollutants, such as certain photocopiers, should similarly be placed in well and separately ventilated rooms. Such appliances should be accompanied by a description containing data on the pollutants emitted and the way these should be dealt with.

In the same way as polluting craft premises and industrial buildings, garages should be located in spaces which are completely separated from other activity as regards ventilation. See also Clause 3.2.

36.3.7 Cleanability and the cleaning of buildings

Ventilation removes the airborne pollutants. Cleaning removes potential airborne pollutants from surfaces in the building.

There are many sources of such pollutants. They may be activities which emit particulates, dirt which enters the building on shoes and clothing, the people themselves, different materials and the outdoor air.

Cleaning of internal surfaces is therefore of great significance for the hygienic conditions including the quality of air in the building.

Owing to the materials used, the design and fittings etc. of a room, it is often difficult to carry out cleaning rationally. This may mean that cleaning is insufficient, but also that aggressive cleaning agents are used, agents which may themselves be emitted into the room air or may decompose surface finishes and add to the airborne pollutants.

Textile floor coverings as well as other materials of large specific area are often not particularly polluting as such, but they become so since it is difficult to keep them clean (Skov et al., 1989; Börjesson, 1985; Nielsen, 1987). They are important storage areas for dirt and pollutants. A relationship has for instance been demonstrated between the use of medication by allergic persons and the incidence of fitted carpets (Bach et al., 1984) and also between the incidence of fitted carpets and the prevalence of the sick building syndrome (Norbäck et al., 1989).

In view of this, the following recommendation is made for regulations:

> Buildings shall be designed so that cleaning of surfaces in contact with supply air or room air is possible. Such surfaces shall be cleaned before the building is taken into use. Surfaces which are likely to become heavily soiled shall be readily accessible and easily cleanable. Surfaces and surface finishes shall be selected so that dirt is not unnecessarily concealed or accumulated.

Apart from walls, floors and ceilings, ducts for supply and extract air, suspended ceilings, radiators etc. are examples of surfaces in contact with supply air or room air.

Surfaces which collect dust or are difficult of access should be avoided. Walls and other surfaces should have a texture that does not collect dirt and dust. An endeavour should be made to keep the proportion of surfaces of high specific area as low as possible.

Internal surfaces should have a texture and colour such that dust and dirt are not concealed.

Cleaning should be carried out so often that stored dust and dirt do not result in deterioration of air quality. Cleaning should be carried out using such methods and agents that cleaning itself causes the least possible deterioration in air quality.

Cleaning should be carried out using agents provided with a product description which have been shown to be of low emission properties and

non-aggressive. Vacuum cleaning should be carried out so that fine grained dust is not returned into the room air.

Radiators should be smooth and easily cleaned even along their rear faces. Radiators should be constructed so that heat emitting surfaces have a low temperature.

36.4 REQUIREMENTS FOR VENTILATION

36.4.1 Outdoor air flow rates

Requirements regarding air flow rates for the ventilation of buildings have varied considerably over time. After Yaglou's studies in the twenties and thirties, thinking in this area and also requirement levels were largely unchanged for a long time. It was not until the energy debate in the seventies and the increased interest in air hygiene that new knowledge regarding the need for ventilation became available. The old "truths" on which NKB Publication No. 41 was based were mainly that man himself was the principal source of pollution which determined the need for air. It was also assumed that the emission of pollutants by building materials, fittings, fixtures, furnishings and furniture and similar also necessitated an air flow rate which was in most cases lower. These separate air flow rate requirements were not added (Nordic Committee on Building Regulations, 1982).

Today we know more about the pollution situation indoors and about the significance of different sources, and also know that the incidence of health problems due to this situation is common. Much knowledge is however still needed before health problems can be related to a certain exposure or a specific pollution source. It is only in exceptional cases that it has been possible to relate symptoms of ill health of the SBS type to the concentrations of specific airborne pollutants or combinations of these. There are very few investigations of SBS in relation to air flow rate. During investigations of a major office complex, however, Jaakkola et al. have found a significant correlation between the outdoor air flow rate and the incidence of SBS symptoms when this was less than 5 l/s per person (Jaakkola et al., 1988). The conclusion was the recommendation that the air flow rate should be not less than 10 l/s per person.

In the study of Jaakkola et al. (1988) and also in a large epidemiological investigation of SBS in the north of Sweden, the real air flow rates were generally far above those specified in codes. In the latter study, the mean value for almost 600 office rooms, situated in 163 buildings, was 16.7 l/s per person (Sundell et al., 1990). Preliminary data from this study indicate that there is no simple relationship between air flow rate and the full SBS syndrome. There is

TABLE 36.1

Three levels of perceived air quality

Perceived air quality (decipol)	Percentage dissatisfied	Required outdoor air flow rate L/S, olf (person)
0.6	10	16
1.4	20	7
2.5	30	4

however a relationship between air flow rate and individual symptoms. The incidence of certain symptoms increases at lower flow rates, while the incidence of others increases at higher flow rates.

A classical measure of air quality is the extent to which odour is perceived as acceptable by visitors directly on entry into the premises. This measure was used by Yaglou in his classical studies, and has in recent years been developed by e.g. Fanger (1988). He has introduced new terms such as olf and decipol to measure the intensity of source and perceived air quality. An olf is equivalent to the perceived air pollution due to the emission (bioeffluents) by a standard person. A decipol is the perceived air quality due to an olf which is ventilated at 10 litres of pure air per second. He quantifies other pollution sources, e.g. building materials, fixtures and fittings, furnishings and furniture, by means of the number of standard persons (olf) which gives rise to the same dissatisfaction as the pollution source being considered. In order that the maximum number of persons dissatisfied with the perceived air quality should be 20% (1.4 decipol), the air flow rate must be at least 7 l/s olf (see Table 36.1). The most important finding in this line of research is that ventilation systems, fixtures and fittings, furnishings and furniture, and tobacco smoking, at least in offices and places of assembly, are sources of pollution which, from the standpoint of odour, are equivalent to, or more important than, man himself.

There is considerable variation in pollution load between buildings (Fanger et al., 1988). According to Fanger, the pollution load from the building, fixtures and fittings, furnishings and furniture, ventilation system etc. is 0.1 olf/m^2 in a "low olf" building, and 0.4 olf/m^2 in a "normal" building, which correspond to 0.7 and 2.8 l/s/m^2 respectively, assuming 1.4 decipol (20% dissatisfied) according to Table 36.1. Too little is known at present about the intensity of sources indoors, measured in olf, for the olf theory as a whole to be applied.

Measurements of the "own odour" of premises have been made to only a small extent apart from Fanger's research. In an investigation of the odour intensity in a classroom, Berglund and Lindvall found that the odour due to

persons exceeded the odour due to the premises at a CO_2 content of 800 ppm, which is equivalent to a flow rate of 10 l/s/person and approx 5 l/s/m^2 (Swedish Council for Building Research, 1987).

In a survey of pupils' degree of satisfaction with the indoor climate in 11 schools, Nielsen et al. (1987) found a large variation in degree of satisfaction between the schools. Significant explanatory variables were outdoor air flow rate per person, quantity of dust and relative humidity. These variables do not however explain the differences between schools. Each school had its own base level for satisfaction with air quality, in relation to which variations in satisfaction occur. In a study of 10 schools, Thorstensen et al. (1990) found a large variation between schools as regards the frequency of complaints. They established a correlation between the content of CO_2, the perception of air quality (in decipol) and reported complaints of the SBS type.

Recent research results regarding odour thus imply that higher air flow rates are required to keep down the "own odour" from the premises and the building, inclusive of the ventilation system. With regard to odour and the emission of pollutants from dust in ventilation ducts, it has been shown (Nielsen et al., 1990) that the contribution of dust, when it is dry and properly ventilated, is negligible.

The humidity of indoor air as a ventilation criterion is a theme of current interest in the Nordic countries, due both to the frequent complaints about "dry air" and to the increasing incidence of moisture and mould damage and allergenic house dust mites (HDM). "Dry air" is a theme of current interest geographically widely different parts of the world with greatly varying climatic conditions, i.e. the coupling to moisture content is by no means a simple one. Studies (referred to in Andersen et al., 1982) have also shown that the perception of "dry air" is due more often to the air being polluted or too warm than to the air being physically dry. Reinikainen et al. found in a study of office workers, however, that the perception of "dry air" and SBS and dryness symptoms was lower in rooms with humidification of air than in rooms without (Reinikainen et al., 1988). A lot remains to be studied, however, for instance with regard to the coupling between the humidity of air and the survival of viruses, bacteria, etc.

Simpler relationships exist between the humidity of air and proliferation of mould and house dust mites. House dust mites thus normally constitute the design criterion. In Danish and later in Swedish dwellings, a rapid increase in the number of mites has been demonstrated. A Danish study by Korsgaard found a forty-fold rise between 1977 and 1986 and a simultaneous increase by a factor of 3–5 in allergic complaints related to this (Korsgaard, 1987). The increased incidence has been ascribed to the fact that buildings have become "wetter" as a result of reduced ventilation. Korsgaard, among others, has shown that mites require humidity, even under winter conditions, of more

than 7 g H_2O/kg of air, i.e. ca 45% RH at 21°C, for survival and increase (Korsgaard, 1981). This is of practical significance only in dwellings and possibly in day nurseries. The moisture load indoors is not sufficiently known, but there are a lot of indications that approximately 0.5 ach is sufficient to keep relative humidity below this value (Nielsen, 1989).

Measurements in residential buildings show that air flow rates today vary appreciably with the type of dwelling and type of ventilation. It has for instance been shown in a Danish investigation that detached houses with natural ventilation have an average flow rate of 0.33 ach, and that, with mechanical ventilation, detached houses have 0.55 ach and blocks of flats 0.59 ach (SBI Report, 1991). A Finnish investigation (Ruotsalainen et al., 1990) comprising 300 dwellings has found no significant difference in air flow rates between natural and mechanical ventilation systems. The average outdoor flow rate (median value) was 0.5 ach, but in 50% of cases the value was below the recommended figure.

The frequent health problems, even in buildings with "standard" ventilation, a few epidemiological studies and new knowledge regarding the "own odour" of premises, all indicate that the old NKB values ought to be increased. There are however large gaps in knowledge as regards e.g. the significance of outdoor air flow rate for the incidence of different symptoms, the significance of the odour criterion for acceptability and health effects in a somewhat longer term, whether direct summation of the different outdoor air flow rate requirements is relevant, the influence of the period of operation (can a lower flow rate be accepted with respect to the load in the premises if the flow rate is continuous), etc. In addition, the available research results are as yet greatly limited in scope. It is not known whether they are representative for a normal stock of buildings and also for other types of properties. In view of the present state of knowledge, minimum air flow rates can be laid down only with a high degree of uncertainty.

The choice of outdoor air flow rate per person who is engaged in sedentary activities can be made on the basis of the odour criterion. For instance, the flow rate 7 l/s/person (Fanger, 1988), which corresponds to 1.2 met (sedentary activity), is obtained for the criterion of 20% dissatisfied (Rasmussen et al., 1985). Higher air flow rates are required at higher levels of activity. From the standpoint of odour, an activity of 1.6 met is equivalent to 12 l/s/person, and 6 met (=gymnastics) to 70 l/s/person (Fanger, 1988; Rasmussen et al., 1985). It should be reasonable for the outdoor air flow rate which is required in view of the occupancy level to be added to the air flow rate required with respect to other pollution sources (building materials, fixtures and fittings, furnishings and furniture, dirt, activities, etc.). There are discussions whether such an addition should be full addition (i.e. 1+1 = 2) or whether it can be supposed

that a hypoaddition (1+1 < 2) occurs. If an addition factor of 0.5 is selected (i.e. 1+0.5×1 = 1.5), a minimum value 0.5×7 l/s/person = 3.5 l/s/person is obtained for the occupancy level.

In view of this, the following recommendation is made for regulations:

> Rooms in which people are present other than occasionally shall be provided with an outdoor air flow rate which is sufficient to dilute and remove the pollutants normally present in the room to contents so low that at least the overriding requirements in accordance with Section 2 are complied with.

36.4.1.1 Choice of outdoor air flow rates

The outdoor air flow rate shall be decided with respect to the required quality of indoor air, the quality of outdoor air, the type and intensity of pollution sources, ventilation and air change efficiency. When new buildings are planned, there are usually no design data available, and empirical values must therefore be used.

The minimum values given below presuppose

- that the supply air maintains good outdoor air quality,
- that air change efficiency is at least equivalent to well mixed ventilation (approx 0.5),
- that building materials, fixtures and fittings, furnishings and furniture, are low emitting,
- that there are no "special" polluting activities,
- that the supply air system is clean, and
- that the standard of cleaning in the building is satisfactory.

With regard to air change efficiency, see Clause 4.3.

The outdoor air flow rate with respect to the release of pollutants from sources other than persons should be not less than 0.7 l/s/m^2. A considerably higher flow rate may be required if the choice of building materials, fixtures and fittings, furnishings and furniture etc. has not been properly thought through, as in many existing buildings.

The additional outdoor air flow rate per person engaged in sedentary activities should be not less than 0.35 l/s/person. For higher levels of activity greater air flow rates are required. However, the total air flow rate, based on occupancy level, should not at any time be less than 7 l/s/person.

For a room for a single person, of 10 m^2 area, the above implies a total outdoor air flow rate of 0.35 l/s/person + 10 m^2 × 0.7 l/s/m^2 = 10.5 l/s.

In rooms where tobacco smoking usually occurs, the outdoor air flow rate should normally be not less than 20 l/s/person.

In the case of dwellings the flow rate will vary depending on the likely occupancy level, extract air flow rates in accordance with Clause 4.4, and the area of the dwelling. The outdoor air flow rate based on the area of the dwelling may probably be somewhat lower than 0.7 l/s/m^2. One of the reasons for this is that the occupants can control ventilation and increase flow rate by opening windows as necessary, so that ventilation in each room during the period of use is in accordance with the requirements in Clause 4.1.

The minimum outdoor air flow rates set out above are based only on the requirement regarding air quality. In the case of many buildings, other factors such as the need to maintain a certain level of temperature constitute the design criterion for the total outdoor air flow rate.

In design, the tolerances for air flow rate should be determined in each individual case with respect to the inaccuracy in measurement and balancing and the expected operational and maintenance conditions. With regard to tolerances, see NKB Publication No. 52, Subclause 3.3.4.

36.4.1.2 Operation

When ventilation is turned off or when flow rate is reduced, pressure conditions in conventional systems can be substantially different from those normally aimed at. Spread of pollution can then occur from rooms with a high pollution load to those with a lower load. Supply air ducts may act as extract ducts, which may result in contamination of the ducts. When flow rates are reduced or the system is turned off, there is a higher concentration of pollutants in the room and a risk of greater "ex-change" through adsorption and desorption of pollutants between different sources and surfaces in the room. This may have the result that the flow rates on full operation, which are normally sufficient, are not enough to keep contents at the required levels.

In view of this, the following recommendations are made for regulations:

When a building is not used in the intended manner, the air quality in the building when this is again put into use shall not be inferior to that in normal operation.

The air handling system shall be designed so that the spread of pollutants in the building, or contamination of the supply air system, does not occur during times when the building is not used.

In view of the fact that the emission of pollutants from new surface finishes, fixtures and fittings, furnishings and furniture is somewhat higher in the beginning, ventilation should normally be run continually at full flow rate

during the first year after new construction or internal renovation. The same may be required after the installation of new fittings, fixtures, furnishings and furniture, and during cleaning.

When supply air flow rates vary, care must be taken to ensure that the installation is able to maintain the intended pressure conditions in the building so that there is no uncontrolled spread of pollutants.

36.4.2 The quality of outdoor air

The outdoor air admitted shall dilute the pollutants which are generated indoors and replace vitiated indoor air by "fresh" air. It is therefore essential that this outdoor air should be as clean as possible. The building should preferably be placed in as clean an environment as possible in accordance with Clause 3.1.2. Where this cannot be done, the outdoor air intake should be situated at the place in the building where the air is best in relative terms. This is normally on the roof and on the side facing the courtyard. Experience shows that outdoor air intakes are often inappropriately sited, for instance towards a street carrying heavy traffic, parking areas or similar, which causes discomfort to the users of the building.

Particulate pollutants can be effectively removed by conventional technology; this should be done so that allergenic and dirty particles are not admitted into rooms and so that the supply air installation is not contaminated.

In view of this, the following recommendation is made for regulations:

> Outdoor air intakes shall be placed where the air admitted is likely to be cleanest and to have the lowest temperature in the summer, in view of the position of the building and the placing of chimneys or flues or other sources of pollution. Where this is required because the quality of outdoor air is unsatisfactory, the supply air shall be filtered to remove dust, pollen and other particulate pollutants.

In more polluted environments, for instance in town centres, the outdoor air intake shall be placed on the roof or towards an internal courtyard at a level such that pollutants from the ground are not drawn in. In supply air systems filters of not less than Class EU 7 (F 85) should be used to filter the outdoor air. Outdoor air intakes should not be placed in the prevailing wind direction downwind from a chimney or other discharge for gas or polluted air.

Vents for direct admission of outdoor air into rooms should be fitted with filters. See also NKB Publication No. 52, Subclauses 3.3.1 and 3.3.4, and (Bergsøe, 1989).

36.4.3 Air flow conditions

It is impossible to prevent some spread of pollutants in rooms without extensive encapsulation and similar. Spread between rooms occurs easily, for instance when persons pass through door openings or when there are differences in temperature. Improperly adjusted installations with imbalances between supply and extract air systems which give rise to unintended pressure conditions and flow directions, are also of common occurrence.

Quantified requirements regarding air change efficiency, other than it shall be satisfactory, i.e. it should be around and preferably over 50%, cannot be reasonably stipulated at present. There are few measurements in rooms under normal operational conditions, and those which are available indicate that in the majority of cases this is around 50% unless there are direct faults such as e.g. greatly elevated inlet temperatures in combination with extract terminals placed in the ceiling (Sandberg, 1984; Sandberg et al., 1988). The principles should however be set out so that they may perhaps be further developed also in a requirement context.

At the Nordic seminar "The Healthy Building" (Dawidowicz et al., 1987) the following statement was made regarding recirculated air:

> "Recirculated air is not recommended in premises open to the public as a general method because of the risk of spreading gases and vapours, and in view of the experiences of faulty technology and lack of maintenance in practice. Air from 'polluted' rooms, for instance smoking rooms, should not be recirculated. In existing installations recirculated air can be used, but filters and the volume of outdoor air admitted must be checked often. It is recommended that in new installations other solutions should be applied to recover heat. Recirculated air systems can be used as a method of mixing outdoor air and room air where this is a practical necessity for e.g. thermal reasons. If recirculated air is used in new buildings, special requirements must be complied with as regards balancing, regular checks on performance and cleaning of the duct system."

Jaakkola et al. (1990) have however found that there is not necessarily any coupling between recirculated air and complaints in offices.

Hot air heating systems have earlier been considered to demand the use of recirculated air in view of the large flow rates required. In the new airtight and low energy buildings of today this is seldom necessary. Buildings have normally been cooled by air, which has also necessitated large air flow rates in the summer and the use of recirculated air during the cold months. Other systems for cooling which do not require such large air flow rates are being used

increasingly, and this reduces the significance of recirculated air.

In view of this, the following recommendations are made for regulations:

The spread of airborne pollutants in the room and to other rooms shall be limited. Spread of airborne pollutants to other rooms where these are not normally generated shall be prevented.

Air shall be designed to flow from a room with a more stringent requirement regarding air quality to a room with a lower requirement.

Air change efficiency shall be satisfactory.

The basic principle is that pollutants should be captured as near the place of generation as possible and removed from the room as quickly as possible. In conjunction with concentrated sources of pollution which are not negligible, the concept of ventilation efficiency can be used to describe the ability of ventilation to capture and remove such pollution. This is done by relating the equilibrium concentration in the extract air to the mean concentration in room air under equilibrium conditions. The highest ventilation efficiency is achieved by providing some form of process ventilation in accordance with Clause 4.5.

If there are only diffuse and passive pollution sources such as building materials, fittings, fixtures, furnishings and furniture, or if the sources are weak and mobile, air change efficiency is a better measure. This shows how effectively air in a room is changed. It is defined as the ratio between the mean age of the air in the extract air (exchange time of the ventilation air) and twice the mean age of the air in the room. The highest efficiency, 100%, is obtained under ideal parallel flow conditions, when air passes as a rigid body from e.g. the floor to the ceiling. Efficiency is least when there is short circuiting between supply and extract terminals. When supply and extract terminals are placed in the ceiling and the supply air is heated, there is a risk of such flow, especially if the ceiling is high and there are few sources of disturbance in the room. An air change efficiency of 50% represents "perfect mixing".

In order to secure acceptable direction of flow between rooms in a building, it is necessary to ensure that pressure conditions are correct. For instance, rooms which are more polluted such as garages, smoking rooms, rooms for photocopiers, toilets and kitchens, shall be at a lower pressure than surrounding cleaner rooms. The aim in blocks of flats is to achieve neutral pressure conditions between flats.

These requirements imply that air cannot be recirculated from e.g. rooms where smoking will occur, rooms for photocopiers, between flats in a block of flats, or similar.

36.4.4 Processes and sanitary accommodation

Pollutants should be captured as near the source as possible. The technology for this was developed in the industrial ventilation sector long ago. In the non-industrial sector this technology regarding encapsulation and local extraction is little used and developed. See also Clause 3.6. It is likely that there will be substantial development in this area, for instance with regard to encapsulation and hoods for office equipment and also hobby activities in the home. The cooker hoods in use at present should also be subjected to further development.

Numerical values for kitchens, bathrooms and toilets are empirical and largely correspond to previous recommendations. If flow rates are too low, moisture and mould problems easily arise.

In view of this, the following recommendation is made for regulations:

> Pollution sources which are tied to a certain place and pollution sources or activities of frequent occurrence shall be provided with such process ventilation of the encapsulation, hood or local extraction type that the spread of pollutants is normally prevented.

Pollutants which are of interest from the hygienic standpoint largely follow the air streams. By controlling the flow of air, the pollutants can therefore also be controlled. As regards local extraction and encapsulation, in principle there are three types:

- Encapsulation, which means that the source of pollution is completely enclosed, for instance a fume cupboard or entire room such as toilets, smoking rooms or rooms for photocopiers.
- Entrainment type local extraction works by entraining the pollutants into the air stream towards the extraction device. Examples are weld fume extractors, vacuum cleaners, etc.
- Collection type local extraction gathers up the pollutants and then removes them. Pollutants enter the extraction device under their own power. Cooker hoods are one example of this technology. They are designed so that they have sufficient volume to receive the pollutants which are often generated intermittently, and so that they have sufficient extract air flow so that the hood does not become full and "spill over". The flow rate necessary is considerably lower than in the case of the entrainment type. For instance, a large cooker hood for domestic use requires a flow rate of the order of 20 l/s while a "flat" hood which is intermediate between the two types requires flow rates of approx 100 l/s for the same function. Cooker hoods and extraction hoods are easily

affected by the disturbance caused by air streams due to people moving in their vicinity or to open windows.

Sanitary accommodation such as toilets and bathrooms shall be provided with an extract air flow rate which is both sufficient for the room to act as a kind of encapsulation and enables the room to dry out quickly after use in order to prevent moisture and mould damage. A toilet in a dwelling requires an extract flow rate of not less than 10 l/s, and a bathroom not less than 15 l/s. If the bathroom has no windows, it must be possible for the flow rate to be increased to not less than 30 l/s. It must be possible for such increased rate to be set for times up to a least 2 hours. Toilets in public premises or workplaces should have a least extract flow rate of 15 l/s per toilet stool, urinal, or similar.

Kitchens shall be given a flow rate of not less than 20 l/s. Electric cookers with more than 2 hotplates are to be provided with a hood or local extraction with a collection efficiency of at least 85% for gaseous substances. In the case of gas cookers it is necessary in addition to the above for the collection efficiency and air flow rate to be adjusted to the need to remove combustion products.

36.4.5 Opening of windows

The availability of open windows is protection against malfunction of the ventilation or heating system, and a necessary extra facility for increased extraction in the event of unplanned temporary pollution loads. Bathrooms and shower rooms should also be provided with openable windows. See also Subclause 4.6.1.

In view of this, the following recommendation is made for regulations:

Every workroom and habitable room shall be provided with an openable window for ventilation.

36.4.6 Engineering requirements for air handling installations

36.4.6.1 Ease of use

Jaakkola et al. (1989) draw the conclusion from an investigation of a major office complex that individual control of room temperature increases satisfaction and probably reduces sick building symptoms.

The same conclusion is drawn by Hedge et al. (1989) from a nationwide study of building related diseases in the UK. They put forward the hypothesis that the tolerance threshold of a person to disturbances in the climate is reduced when he or she cannot modify, or control, the climate in his or her surroundings. The varying needs and tolerance thresholds of people appear to

justify the introduction of a requirement that the individual should be given a large measure of control over the climate in his or her surroundings. It has however been considered that this requirement is not so well founded at present that the large additional cost which it would involve can be justified.

In view of this, the following recommendation is made for regulations:

Control devices shall be easy to reach, understand and operate.

The aim should be to develop systems in which the users can easily control the magnitude and direction of the air flow and the temperature of the room and the supply air. The term control device refers to all kinds of arrangements by means of which the temperature, magnitude and direction of the air flow can be controlled, i.e. it includes arrangements for opening and closing vents for the direct intake of outdoor air.

36.4.6.2 Space requirements

In order that inspection and maintenance shall be carried out to the extent required, it is necessary that ease of access and good working spaces shall be provided.

In view of this, the following recommendation is made for regulations:

Components which require attendance and maintenance shall be sited so that they are readily accessible and replaceable, and mounted so that work can be carried out easily and safely.

For guidance reference should be made to NKB Publication No. 52, Subclause 3.2.7.

36.4.6.3 Cleanability

Supply air installations have in some cases been shown to have considerably contaminated the outdoor air on its passage through the installation (Fanger et al., 1988; Berglund et al., 1982). This is due to both pollutants from materials in the installation (filters, sealing compounds, oil from the production of ducts or similar) and to the dirt deposited in the installation. With regard to emissions from dry dust in ventilation ducts, see Fanger (1988).

Cleaning of extract air installations is required mainly to prevent deposits from reducing the air flow rate. It should be noted that an extract installation can at times function as a supply air installation, for instance in the event of recirculated air operation or when the ventilation system is turned off.

Rapid development with respect to system design and cleaning facilities can already be noted.

In view of this, the following recommendation is made for regulations:

It shall be possible for both supply and extract air installations to be cleaned in their entirety. Installations shall be cleaned so often that neither the magnitude of air flows nor the quality of supply air is adversely affected by deposited dirt.

For guidance reference should be made to NKB Publication No. 52, Subclause 3.3.10.

36.4.6.4 Components and materials

Surface finishes on the inside of a supply air installation can release pollutants to the supply air in the form of both particles and fibres (internal insulation) and gates (odorous filter materials, oil from the production of ducts, etc.).

In view of this, the following recommendation is made for regulations:

Components shall be made of materials which stand up to the intended use and maintenance and do not emit pollutants such as particles or gases which may adversely affect the quality of supply air. The choice of materials and construction shall be such that the growth of microorganisms is prevented.

See NKB Publication No 52, Clause 3.3.

36.4.6.5 Airtightness and pressure conditions

Experience shows that ventilation installations are very leaky. This makes it difficult to control air flow in the building, i.e. to deliver the air to the point where it is needed.

A large number of rotary heat exchangers with or without recirculated air dampers have their fans in the wrong place, so that there is considerable recirculation of air even when the damper is closed (often up to or even above 50%). It has been shown that when fans are placed correctly, recirculation of organic gases via rotary heat exchangers may be as much as 10%.

In view of this, the following recommendation is made for regulations:

Installations shall have the required airtightness. Pressure conditions between supply and extract air installations shall be adjusted to the airtightness of these installations so that there is no unintended flow from extract air to supply air.

With regard to the airtightness of ventilation installations, see NKB Publication No. 52, Subclause 3.2.3.

In conjunction with e.g. dampers for recirculated air or heat exchangers, care is to be taken that pressure conditions are such that any leakage takes place from the supply air to the extract air.

36.4.6.6 Humidification of air

In the light of present knowledge, humidification of room air is seldom necessary unless a certain moisture content is required for production engineering reasons or e.g. in certain hospital rooms. A lot of problems associated with dryness can be remedied by humidification when humidity is less than 20% (Nielsen et al., 1987; Jaakkola, 1990).

At the Nordic seminar "The healthy building" (Dawidowicz et al., 1987), the following statement was made regarding humidification:

> "From the hygienic point of view, general humidification of air is not recommended. General humidification may have side effects such as proliferation of house dust mites, Legionnaires' Disease, other allergies. Symptoms of 'dry air' shall be mainly counteracted by methods other than humidification. Selective humidification may be required for special individuals/environments/ processes. In such cases it is essential that a 'safe' technology should be chosen".

See also the background text in Clause 4.1.
In view of this, the following recommendation is made for regulations:

> Where humidification of air is required for special reasons, a type which does not involve the risk that microorganisms or other harmful substances will be released into the air shall be chosen.

Microorganisms multiply in still water or on surfaces which are constantly wet for long periods. See also NKB Publication No. 52, Subclause 3.3.8.

36.4.6.7 Balancing, handing over

Ventilation installations shall be balanced so that the intended flow rates and tolerances are obtained.

When an installation is handed over, it shall be demonstrated that it has been constructed, and functions, in the way intended. The installation shall be handed over in a clean state ready for operation.

The design nominal air flow rates shall be specified together with the assumed reference conditions. See NKB Publication No. 52, Subclause 3.2.5.

Tolerances shall be specified inclusive of inaccuracy in measurement, i.e. inclusive of the probable measurement error. This should at all times be substantially less than the permitted deviation. Reasonable tolerances for individual terminals or rooms are in the range 10–20%. In the case of rooms containing both supply and extract terminals, consideration shall be given in determining the tolerances to whether the room is to be at a pressure higher or lower than its surroundings.

Guidance regarding documentation, balancing and handing over is given in NKB Publication No. 52, Clauses 4.2 and 4.3.

36.5 DOCUMENTATION, MANAGEMENT, OPERATION AND MAINTENANCE

Experience shows that a building and its installations are often insufficiently documented with regard to aspects that are significant for the healthy state of the building. There are often no documents which describe the building or its installations as they are actually constructed. Changes which are essential with regard to air quality may be made at a late stage during the construction of a building, or in conjunction with repairs or refurbishment. Changes in duct layout, change of paint, adhesive, filler or surface finishes are examples of changes which may give rise to problems. In investigating health problems in a building, work is made considerably easier if the building is well documented in all respects.

In order that air quality shall not be adversely affected, it is important that instructions should be available for the operation, maintenance and cleaning of the building and the ventilation installations, and that these should be observed. Experience shows that there are often no such instructions or they are unsatisfactory. In order that an installation may be restored to its "as built" state, it is essential that there should be documentation which sets out the way the installation was intended to function when designed.

In order that operation and maintenance should be carried out effectively, it is necessary for someone to be responsible for the function of the ventilation installation, for this work to be given a reasonably high status, and for the person concerned to be given the necessary training in the way the system works and in operational and maintenance work (Andersson et al., 1986).

In view of this, the following recommendations are made for regulations:

The necessary drawings and specifications shall be produced for a building and the ventilation installation. The materials used, including make and type designation, shall be documented.

Air flow rates through individual terminals or for individual rooms shall be specified together with the reference state and tolerances under the assumed internal and external loads. The same applies to the temperatures of supply air and room air. A written description of the way the installation has been designed, with the assumptions set out, shall be drawn up.

A building and the ventilation installation shall be operated, cleaned and maintained so that the quality of air is not adversely affected due to the operational, cleaning and maintenance conditions.

Instructions for the operation and maintenance of the building and the ventilation installation shall be drawn up and shall be available when the building is put into service. If possible, they shall be drawn up in direct cooperation with the operational and maintenance staff. The instructions for the ventilation installation shall apply only for the installation in question and shall contain all necessary information in order that satisfactory ventilation of the rooms served may be ensured.

User instructions in easily understandable language, which provide information on attendance, cleaning and maintenance, shall be affixed within easy reach of each terminal or appliance which is capable of regulation or cleaning by the users of the room or flat.

In addition to the documents required during the design and construction of the building, documents required during the operational stage should also be drawn up. It is important that in these the function of the installation should be described as clearly as possible. A flow chart indicating the most important functions should be produced, as well as documents which show where measuring and inspection points, cleaning doors etc. are located.

A plan should also be drawn up showing how the building shall be cleaned.

There are many well developed guidelines available for the layout and contents of operational and maintenance instructions. For guidance regarding the production of operational and maintenance instructions for ventilation installations, see NKB Publication No. 52, Clause 5.2. For guidance regarding operational control of ventilation installations, see NKB Publication No. 52, Clause 5.1.

Instructions in the appropriate Nordic language shall be displayed in the vicinity of terminals (appliances) or in some other place convenient for the users of the rooms. The instructions should be written in an easily understood language, and technical terms should be avoided.

36.6 QUALITY ASSURANCE, INSPECTION

The need for better quality assurance during the planning, construction and operation of a building is a theme that is common to conferences such as "Healthy Building 88" (Berglund et al., 1988), public investigations of health problems attributable to buildings such as the Allergy Enquiry in Sweden (Ministry of Health and Social Affairs, 1989) and the Sick Building Group in Sweden (Ministry of Housing and Physical Planning, 1990). There is unanimous demand that there should be much better follow-up and control of especially the air quality aspects at all stages. Extensive literature deals with quality assurance in construction; see, e.g. National Danish Building Agency (1987) with regard to Denmark.

In view of this, the following recommendations are made for regulations:

At all stages of the design, construction and operation of a building, checks shall be made that intended quality is secured.

Buildings shall be regularly inspected with regard to the function of the ventilation and other factors which are significant for indoor climate.

A specification regarding significant requirements for air quality should be drawn up at an early stage in the course of planning.

The way in which the intended qualities are to be secured should be set out in advance and it should be demonstrated on completion that this has been done. During the design and construction of the building, all conditions which are significant for the quality of air should be systematically checked.

A procedure which ensures that checks and inspections are made should be established at all stages during the construction and operation of a building.

In addition to the continuous operational control, ventilation should be checked at appropriate intervals with regard to its function in the rooms served.

Examples of the other factors significant for the quality of air which should be checked during the service period of the building are the use of rooms, the incidence of moisture and mould damage, and the standard of cleaning.

According to the publication "Climatic problems in buildings, investigation methods and remedial measures" (Hagen et al., 1986) of the Nordic Ventilation Group, investigation of building-related health problems may be an appropriate method of checking the overall function of a building with respect to the internal climate. In the simplest form of check, the person responsible for the building asks the safety committee or similar whether there are any problems. If there is any doubt, the check can be extended to a questionnaire survey or a clinical investigation of the users of the building.

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Indoor Air Quality
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Chapter 37

Selected Indoor Air Quality Guidelines and Activities[1]

37.1 THE U.S. EPA GUIDELINES AND ACTIVITIES

37.1.1 EPA's program for dealing with indoor air pollution

Because of the potentially serious impacts on the health of individuals who may experience indoor air quality problems — as well as the dollar costs to society if indoor air pollution is not addressed — EPA has developed a comprehensive program to better understand the indoor air pollution problem and to take decisive steps to reduce people's exposures to indoor air contaminants of all types.

Even in the absence of complete scientific understanding of indoor air pollution, prudent public policy dictates that reasonable efforts be undertaken to reduce people's exposure to potentially harmful levels of indoor air pollutants, using the authority available to the Federal government under current laws.

Pollution prevention — and efficient resolution of indoor air quality problems of all types — must become a routine aspect of the design, construction, maintenance, and operation of public and commercial buildings, homes, health and day care facilities, educational institutions and other special use buildings.

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1 A part of the text of this chapter has been derived from EPA (U.S. Environmental Protection Agency), 1993. Targeting Air Pollution. EPA's Approach and Progress, EPA-R-92-012, Washington, D.C.: U.S. Environmental Protection Agency. March 1993; EPA, DHHS, CDC, NIOSH. Building Air Quality: A guide for building owners and facility managers, Appendix B: HVAC systems and Indoor Air Quality, pp. 137–139, EPA/400/1-91/033, Washington, D.C.: U.S. Environmental Protection Agency. December 1994; Exposure Guidelines for Residential Indoor Air Quality, A Report of the Federal-Provincial Advisory Committee on Environmental and Occupational Health, Environmental Health Directorate-Health Protection Branch, published by authority of the Minister of National Health and Welfare, April 1987; The Nordic Committee on Building Regulations guidelines for indoor climate and air quality, as published by The Nordic Committee on Building Regulations NKB, NKB, Publication No. 61E, June 1991.

An effective research and development program must be conducted to achieve a more complete understanding of the factors affecting indoor air quality, exposure patterns, health effects, and control techniques for improving indoor air quality.

EPA is implementing this program using non-regulatory as well as regulatory tools available under a number of Federal laws to provide information and incentives for action to product manufacturers, architects, engineers, builders, building owners and managers, and building occupants.

The primary objectives of EPA's program are to:

- Establish effective partnerships with organizations representing the range of target audiences for indoor air quality information to communicate specific guidance and information and promote timely action on indoor air quality issues;
- Forge constructive alliances with other Federal agencies to leverage resources and ensure that existing statutory authorities are used most effectively;
- Develop practical guidance on indoor air quality issues utilizing a broad-based consensus approach which includes representatives from industry and public interest groups to ensure that information provided is accurate and practical;
- Design market-based incentives for industries to lower chemical emissions from their products and provide consumers and other decision-makers with information needed to make informed purchasing decisions;
- Sharpen the focus of the chemical screening and risk management program under the Toxic Substances Control Act (TSCA) and the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) to ensure that chemicals that pose unreasonable risks indoors are identified and addressed;
- Identify and fill research gaps in order to provide information to address outstanding indoor air quality policy issues;
- Select appropriate environmental indicators to measure progress in reducing population exposure to indoor air quality problems as the program matures;
- Enhance scientific understanding and public awareness of the complex factors affecting indoor air quality; and
- Bring about substantial reductions in human exposure to the entire range of indoor air pollutants.

37.1.2 Reducing pollutant levels indoors

The building system approach

EPA has set a high priority on improving the way in which buildings are designed and operated, having concluded that people's exposure to indoor air pollutants can be reduced significantly by implementing current knowledge about sound building operation and maintenance practices. Some of the major actions to date include:

- Issuance, in cooperation with the National Institute for Occupational Safety and Health, of comprehensive guidance, entitled *Building Air Quality: A Guide for Building Owners and Facility Managers*, on how to prevent and resolve the full range of indoor air quality problems in public and commercial buildings.
- Publication of *The Inside Story: A Guide to Indoor Air Quality*, to help people identify and correct potential indoor air quality problems in their own homes.

In addition, EPA is developing guidance for school facility managers, new home builders, and architects and design engineers to acquaint them with the most current information on how to prevent indoor air quality problems from occurring or resolve them quickly if they do occur.

The pollutant-specific approach

This emphasis on a "buildings approach" holds the most promise for addressing all the factors — including those related to the ventilation system as well as sources of individual pollutants — that affect indoor air quality. However, the Agency also strongly believes that it must aggressively utilize its combined statutory authorities to identify specific pollutants that present direct health risks in the indoor environment, and to use a variety of means to reduce their levels indoors. The indoor air pollutants that are currently receiving significant Agency attention include:

Radon

The Indoor Radon Abatement Act of 1988 (Title III of TSCA) established a national goal of achieving indoor levels of radon which are no greater than outdoor levels. EPA has undertaken a range of activities directed toward this goal, including revising public information materials, providing financial and technical assistance to States, developing and encouraging the adoption of radon-resistant building practices, establishing training centres, operating industry proficiency programs, conducting studies in schools and Federal

buildings, and performing mitigation research in different building types.

Environmental tobacco smoke

EPA has recently completed a major report on the respiratory health effects associated with environmental tobacco smoke. The report, entitled "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders", concludes that each year secondhand smoke is responsible for about 3000 lung cancer deaths in non-smokers and causes respiratory health problems for hundreds of thousands of young children. EPA is developing an education and outreach program to inform the public about the risks of passive smoking.

Asbestos

Title II of TSCA, the Asbestos Hazard Emergency Response Act (AHERA), passed in 1987, required EPA to establish a regulatory framework for addressing asbestos in schools. The Agency has set standards for state accreditation of personnel involved in asbestos management or abatement in school buildings and will extend accreditation requirements to those who inspect or abate asbestos in public and commercial buildings. EPA is also involved in a range of outreach, grant, and technical assistance activities. Major recent accomplishments include publication, with the Consumer Product Safety Commission and the American Lung Association, of a home-owners' guide to *Asbestos In Your Home*; completion of a public dialogue on asbestos in buildings with industry, real estate interests, unions and the public sector; and publication of a building owner's guide, *Managing Asbestos in Place.*

Toxic substances

TSCA grants EPA broad authority to control chemical substances and mixtures that present an unreasonable risk of injury to health and the environment. EPA has authority to require testing of chemical substances and mixtures; regulate hazardous chemical substances and mixtures by prohibiting or restricting their manufacture, processing, distribution, and disposal; review new chemicals and their intended uses; and impose labeling or notification requirements. TSCA has been used to regulate asbestos, and the Agency is now evaluating groups of chemicals in selected use categories for their effect on people in indoor environments.

Pesticides

FIFRA authorizes EPA to control pesticide exposures by requiring that any pesticide be registered with EPA before it may be sold, distributed, or used in this country. EPA is evaluating the health impacts of indoor products including insecticide sprays, termiticides, and wood preservatives. Major accom-

plishments include the withdrawal from the market of chlordane as a termiticide in homes and mercury used as a mildewcide in many indoor paints.

Lead

Exposure to dust from lead-based paint can pose a serious health threat in homes or apartments where remodelling is taking place. Toddlers and young children are at particular risk because they are more likely to swallow lead dust and the impact on their bodies is more severe. EPA, along with other key Federal agencies, is working to develop a comprehensive strategy to address lead exposures and to develop effective lead testing and abatement procedures.

Indoor air pollutants for drinking water

The Safe Drinking Water Act (SDWA) authorizes EPA to set and enforce standards for contaminants in public water systems to protect against both health and welfare effects. EPA sets standards for volatile organic compounds (VOCs) that can enter the air through volatilization from water used in a residence or other building. Eighteen such standards have been issued to date and three more are planned. EPA is also developing a standard for radon in drinking water.

The carpet policy dialogue: an innovative approach to reduce pollutant emissions

The Agency recently completed a year long "dialogue" with carpet floor covering industries, unions, public interest groups, and other Federal agencies to explore ways of reducing the emission of VOCs from new carpet and related installation materials, such as carpet cushion and adhesives. As a result of this voluntary process, the carpet industry agreed to test new carpet floor covering materials for total VOC emissions and is exploring ways of lowering emissions of VOCs from carpet products. Most importantly, the industry has undertaken an extensive consumer education program in cooperation with other dialogue participants, designed to provide the public with information on the role that carpet products play in indoor air quality and ways that consumers can make informed purchase decisions. EPA expects to conduct similar discussion with other industry groups to determine whether additional reductions in indoor pollutant emissions can be achieved through voluntary actions.

37.1.3 Increasing access to indoor air information

Information dissemination

In addition to publishing a wide range of information materials on indoor air quality, EPA is also developing additional strategies for disseminating

information to key audiences. To ensure that a full range of information about indoor air quality problems and solutions is readily available to both the technical and non-technical public, a National Indoor Air Quality Information Clearinghouse (IAQ INFO) opened in 1992. IAQ INFO will be equipped with toll-free, operator-assisted telephone access, and will be able to provide written information including facts sheets and brochures, perform literature searches, and make referrals to appropriate Federal, State and Regional resources.

Training key indoor air audiences

Because concern about indoor air problems is a relatively recent phenomenon, many of the people who are in the best position to prevent problems or resolve them when they do occur are not sufficiently informed about the issue.

Many indoor air quality problems can be avoided through sound building operation practices, or resolved by knowledgeable building personnel without the need for potentially costly outside assistance. EPA has developed a training course for building owners to acquaint them with the guidance contained in *Building Air Quality: A Guide for Building Owners and Facility Managers* (December 1991). Because many indoor air quality problems are best resolved by responsible government agencies at the State and local level, EPA has developed both a live instructional course on indoor air quality issues, entitled *Orientation to Indoor Air Quality* and a self-placed learning module entitled *Introduction to Indoor Air Quality* (April 1991) for these audiences.

Advancing the science of indoor air quality

EPA is conducting studies to assess indoor air conditions in the nation's existing building stock. Special emphasis is being given to identifying those factors that exert the greatest influence on overall indoor air quality (IAQ) and on occupant health symptoms. The information gained will be used to improve IAQ diagnostic procedures as well as to provide a basis for evaluating the effectiveness of our pollution reduction strategies over time. Another set of studies now underway is designed to quantify the costs of key indoor air pollution control options for typical building structures.

EPA's Office of Research and Development conducts a multi-disciplinary research program on indoor air quality which encompasses studies of the health effects associated with indoor air pollution exposure; assessments of indoor air pollution sources and control approaches; building studies and investigation methods; risk assessments of indoor air pollutants; and a recently initiated program on biocontaminants.

Working with other Federal agencies

More than 20 different Federal agencies have responsibilities associated with indoor air quality, either through their own statutory responsibilities or because they are major property managers. The activities of these agencies are coordinated through a variety of mechanisms, including an interagency Committee on Indoor Air Quality (CIAQ) which meets on a quarterly basis to exchange information on indoor air issues. Five Federal agencies — EPA, the Consumer Product Safety Commission, the Department of Energy, the National Institute for Occupational Safety and Health, and the Occupational Safety and Health Administration — are CIAQ co-chair agencies. In addition, EPA works closely with other agencies on regulatory and information development efforts and jointly sponsors many of its guidance and public information documents with these other agencies to help ensure that Federal actions are well-coordinated.

37.2 THE ASHRAE STANDARD AND GUIDELINES

37.2.1 Standard 62-1989, "Ventilation for Acceptable Air Quality"

ASHRAE 62-1989 is intended to assist professionals in the proper design of ventilation system for buildings. The standard presents two procedures for ventilation design: a "Ventilation Rate" procedure and an "Air Quality" procedure.

With the Ventilation Rate procedure, acceptable air quality is achieved by specifying a given quantity and quality of outdoor air based upon occupant density and space usage. Examples of the tables listing the prescriptive amounts of outdoor air for the Ventilation Rate procedure are presented at the end of this section.

The Air Quality procedure is a performance specification that allows acceptable air quality to be achieved within a space by controlling for known and specifiable contaminants. This procedure is seldom used because source strength is usually not known.

Whichever procedure is utilized in the design, the standard states that the design criteria and assumptions shall be documented and made available the those responsible for the operation and maintenance of the system.

Important features of ASHRAE 62-1989 include:

- a definition of acceptable air quality;
- a discussion of ventilation effectiveness;
- the recommendation of the use of source control through isolation and local exhaust of contaminants;

- recommendations for the use of heat recovery ventilation;
- a guideline for allowable carbon dioxide levels; and
- appendices listing suggested possible guidelines for common indoor pollutants.

37.2.2 Standard 55-1981, "Thermal Environmental Conditions for Human Occupancy"

ASHRAE 55-1981 covers several environmental parameters including: temperature, radiation, humidity, and air movement. The standard specifies thermal environmental conditions for the comfort of healthy people in normal indoor environments for winter and summer conditions. It also attempts to introduce limits on the temperature variations within a space. In addition to specifications for temperature and humidity, guidelines are given for air movement, temperature cycling, temperature drift, vertical temperature difference, radiant asymmetry, and floor temperatures. Adjustment factors are described for various activity levels of the occupants, and different clothing levels.

Important features of this standard include:

- a definition of acceptable thermal comfort;
- a discussion of the additional environmental parameters that must be considered;
- recommendations for summer and winter comfort zones for both temperature and relative humidity;
- a guideline for making adjustment for activity levels; and
- guidelines for making measurements.

It should be noted that space temperatures above 76°F but within the summer comfort envelope have nevertheless been associated with IAQ complaints in offices.

Note: As of summer 1991, a revised Standard 55 was nearly ready.

37.2.3 Standard 52-76, "Method of Testing Air-Cleaning Devices Used in General Ventilation for Removing Particulate Matter"

This standard is intended to assist professionals in the evaluation of air cleaning systems for particle removal. Two test methods are described: the weight arrestance test and the atmospheric dust spot test. The standard discusses differences in results from the weight arrestance test and the atmospheric dust spot test. The atmospheric dust spot test is the test used to determine the "efficiency" of an air cleaner. The values obtained with these two tests are not comparable. For example, a filter with a weight arrestance of 90%

may have an efficiency by the atmospheric dust spot test below 40%. The weight arrestance test is generally used to evaluate low efficiency filters designed to remove the largest and heaviest particles; these filters are commonly used in residential furnaces and/or air-conditioning systems or as upstream filters for other air cleaning devices. For the test, a standard synthetic dust is fed into the air cleaner and the proportion (by weight) of the dust trapped on the filter is determined. Because the particles in the standard dust are relatively large, the weight arrestance test is of limited value in assessing the removal of smaller, respirable-size particles from indoor air.The atmospheric dust spot test is usually used to rate medium efficiency air cleaners. The removal rate is based on the cleaner's ability to reduce soiling of a clean paper target, an ability dependent on the cleaner removing very fine particles from the air. However, it should be noted that this test addresses the overall efficiency of removal of a complex mixture of dust, and that removal efficiencies for different size particles may very widely. Recent studies by EPA, comparing ASHRAE ratings to filter efficiencies for particles by size, have shown that efficiencies for particles in the size range of 0.1–1 μg are much lower than the ASHRAE rating.

Important features of this ASHRAE standard include:

- definitions of arrestance and efficiency;
- establishment of a uniform comparative testing procedure for evaluating the performance of air cleaning devices used in ventilation systems;
- establishment of a uniform reporting method for performance; and
- methods for evaluating resistance to airflow and dust-holding capacity.

No comparable guidelines or standards are currently available for use in assessing the ability of air cleaners to remove gaseous pollutants or radon and its progeny.

37.2.4 Guideline 1-1989 "Guideline for the Commissioning of HVAC Systems"

This guideline is intended to assist professionals by providing procedures and methods for documenting and verifying the performance of HVAC systems so that they operate in conformity with the design intent. The guideline presents a format for documenting the occupancy requirements, design assumptions, and the design intent for the HVAC system. It provides a format for testing the system acceptance by the owner. In addition, the guideline addresses adjustments of the system to meet actual occupancy needs within the capacity of the system when changes in building use are made and recommissioning is warranted.

Important features of this guideline include:
- definition of the commissioning process;
- discussion of the process involved in a proper commissioning procedure;
- sample specification and forms for logging information;
- recommendation for the implementation of corrective measures as warranted;
- guidelines for operator training; and
- guidelines for periodic maintenance and recommissioning as needed.

37.3 THE RESIDENTIAL AIR QUALITY GUIDELINES OF CANADA

37.3.1 Introduction

Criteria for acceptable air quality have existed for many years for the industrial workplace and outdoor environments. Only recently, however, has widespread attention been drawn to potential hazards posed by the presence of airborne contaminants in non-industrial indoor environments. Several factors have contributed to this awareness:
- a trend towards tighter building construction, as incentives are given to conserve fuels;
- the presence of an increasingly complex array of synthetic chemicals used in building and insulating products, furnishings, consumer products and hobby and craft materials;
- the use of alternative heating systems which, if not properly installed or designed, can release combustion by-products;
- a recognition that prolonged exposure even to very low concentrations of chemical contaminants may result in delayed toxic effects; and
- the realization that a very high proportion of the average individual's time is spent indoors.

37.3.1.1 Sources of indoor air contaminants

The quality of indoor air is influenced both by the quality of outdoor air and by the emission characteristics of indoor sources.

Outdoor sources

In almost all inhabited enclosed spaces, there is a continuous exchange of air with the outside. Therefore, all contaminants of outdoor air are likely to be present indoors. Important pollutants in this category include carbon monox-

ide, oxides of nitrogen, oxides of sulphur, particulate matter, ozone (and other photochemical oxidants) and lead.

These pollutants originate, to a large extent, from automobile and factory emissions and other combustion processes. Generally, in the absence of indoor sources of these contaminants, concentrations indoors will be close to or lower than those outdoors.

A natural pollutant is radon-222, a radioactive gas that decays to non-gaseous radioactive species which can be adsorbed onto suspended particulate matter and hence be deposited in the lung. Domestic water, natural gas, underlying soil and groundwater can be significant sources of radon in dwellings.

Indoor sources

Internally generated airborne pollutants fall into one of three categories:

- those formed in combustion processes for heating and cooking;
- those derived from construction materials and furnishings; and
- those related to human activity or presence.

Concentrations of contaminants in the first and last categories tend to vary with time; those in the second are likely to be more constant, assuming that air exchange rates remain constant.

(i) *Combustion processes.* Furnaces and other combustion appliances can be sources of indoor pollutants, notably carbon monoxide, especially if they are not properly vented or routinely serviced. Since combustion in wood-burning stoves is much less complete than in oil gas furnaces, pollutant emissions from them can be greater. Though by-products should be vented to the outside, leaks and improper operation of these appliances can cause emissions to the indoors. Contaminants associated with wood-burning stoves and fireplaces include carbon monoxide, oxides of nitrogen and sulphur, aldehydes and polycyclic aromatic hydrocarbons.

A major potential source of combustion by-products is gas-fired stoves. Emissions from the oven and pilot light are not always vented and can contribute to indoor levels of carbon monoxide, nitrogen oxides and formaldehyde.

Kerosene heaters are becoming increasingly popular for space heating. Since these systems are often unvented, the potential for high contaminant levels again exists. In particular, the improper use of kerosenes with high sulphur contents or of poorly designed units could result in emission of oxides of sulphur as well as some of the other combustion by-products previously mentioned.

(ii) *Building products and furnishings.* Synthetic polymers used in furnishings and decorative materials can slowly degrade, releasing small quantities of the original constituents or reaction by-products. Draperies, rugs and fabrics, the great majority of which contain man-made fibres, are sources of a

variety of organic and, potentially, microbiological contaminants.

Formaldehyde is released from wood laminates and particleboard in which formaldehyde-containing resins have been used. Urea-formaldehyde foam insulation is a significant source of formaldehyde and possibly other gaseous products.

Fibrous materials such as asbestos and fibrous glass are present in some building materials and may be released to the indoor environment especially when such products are disturbed during building-alterations.

(iii) *Human activity*. The variety of contaminants that result from human activity is extremely broad.

Tobacco smoking is a major source of indoor air pollution. While smokers subject themselves to *mainstream* smoke, bystanders can be involuntarily exposed to significant amounts of respirable particles, carbon monoxide and oxides of nitrogen, as well as numerous harmful organic contaminants, in *sidestream* smoke. Over fifty components of cigarette smoke are known to cause adverse health effects, and twelve of these are known or suspected carcinogens.

Human metabolic activity itself influences air quality by reducing the concentrations of oxygen and increasing the level of carbon dioxide. Respiration, perspiration and food preparation add water vapour as well as odour-producing substances to the indoor atmosphere. A large variety of biological agents may be present in the home, for example, micro-organisms from occupants, pets and insects; microbial growths may also occur on moist surfaces or in stagnant water. Pollens, spores, cell debris and insects are present in dust originating both indoors and outdoors.

Air fresheners, furniture waxes, polishes, cleansers, paints, pesticidal formulations, fabric protectors, deodorants and other products frequently used in the home are sources of various inorganic and organic chemicals.

Many substances found in the workplace may also occur in the home as a result of hobby or craft activities. Moreover, workers exposed to chemicals in the workplace may bring these contaminants into the home on their clothing. In some circumstances this may be a means by which significant amounts of potentially harmful substances are introduced into indoor air.

37.3.2 Purpose and scope

37.3.2.1 Objectives

Generally, people can be at home for as much as 70% of their time and, for some segments of the population (the very young, the old and the infirm), this percentage can be much higher. In developing recommendations for the do-

mestic environment, the Working Group considered it prudent to assume that certain segments of population are at home on a continuous basis.

Furthermore, some individuals may be at special risk from indoor air pollution. Such individuals comprise those whose physiological processes are either not fully developed or are deteriorating, or for whom pathological or physiological changes impair the ability to surmount the adverse effects of exposure to a pollutant. Therefore, a primary objective was:

> "to develop guidelines for the concentrations of selected contaminants of *residential indoor air*, taking into account such factors as the sensitivity of groups at special risk and the sources and mechanisms of action of contaminants".

These guidelines may not provide complete protection for the hypersensitive portion of the population which requires extraordinary measures to achieve such protection.

The need to specify ventilation rates for domestic premises that reflect the indoor air quality guidelines was also considered but was deemed to be beyond the expertise of members of the Working Group. Moreover, a prescription for ventilation requirements is only one of several strategies that might be adopted for controlling the presence of airborne contaminants in the home.

Other possibilities include specifications for building design and materials and consumer products. In many instances, measures to minimize exposure to chemical contaminants can be taken by the occupants. Public education is an important strategy for attaining an acceptable quality of indoor air, and the Working Group therefore established as its second objective:

> "to develop, where practicable, other guidelines or recommendations for measures that will preserve or improve air quality in domestic premises".

37.3.2.2 Definitions of indoor air quality

As a guiding principle, air within domestic premises should be sufficiently free from biological, physical and chemical contaminants to ensure that there is a negligible risk to the health and safety of the occupants.

Following an examination of the available information, 17 substances, or groups of substances, were selected for detailed review because of their potential to cause adverse health effects and their possible presence indoors. Guidelines expressed in terms of concentration ranges were developed for nine of these. For the others it was not possible to express the guidelines in quantitative terms, either because the data base was inadequate or because human exposure limits were deemed inappropriate; where possible, however, recom-

mendations are made in this document on measures that can be taken to control indoor exposure to contaminants.

The guidelines in this document therefore encompass two categories of contaminants:

- those for which recommendations are expressed in terms of ranges of concentrations;
- those for which recommendations are specified as practical measures that can be taken to reduce exposure.

Section 37.3.4.1 of this document deals with those contaminants for which exposure limits could be derived exclusively on the basis of health considerations (see section "Non-carcinogen substances"). These are specified for the following substances:

- aldehydes (total)
- carbon dioxide
- carbon monoxide
- nitrogen dioxide
- ozone
- particulate matter
- sulphur dioxide
- water vapour

The potential for adverse effects from long-term exposure as well as from shorter-term higher-level exposure has been considered. Two kinds of exposure limits are therefore specified.[1]

The Acceptable Long-Term Exposure Range (ALTER) is that concentration range to which it is believed from existing information that a person may be exposed over a lifetime without undue risk to health.

The Acceptable Short-Term Exposure Range (ASTER) is that concentration range to which it is believed from existing information that a person may be exposed over the specified time period without undue risk to health.

An important consideration in deriving acceptable exposure ranges is the possibility of interactive effects, since many contaminants are likely to be simultaneously present in the home environment. Where possible, due account was taken of the potential for synergistic and additive effects. However, in most cases there were insufficient or inadequate data to completely resolve this problem.

Section 37.3.4.2 contains recommendations for long-term exposure to formaldehyde, on the basis that it is a possible human carcinogen. For carcinogenic effects, guidelines were not established exclusively on the basis of health

1 Information on radon was also reviewed but no recommendations were developed by the Working Group on Indoor Air Quality.

considerations; rather they were set as low as possible taking into account the cost and technical feasibility of attainment, as well as the associated health risk (see section "Carcinogenic substances").

Section 37.3.4.3 of these guidelines deals with those contaminants for which the formulation of exposure ranges was deemed inappropriate or was not feasible. This group comprises:

- biological agents
- chlorinated hydrocarbons
- fibrous materials
- lead
- pest control products
- polycyclic products
- polycyclic aromatic hydrocarbons
- product aerosols
- tobacco smoke

Recommendations for procedures that would reduce or eliminate exposure in the home are provided for these substances.

In developing the recommendations for these 17 substances or groups, the Working Group critically reviewed the available literature. The criteria and rationale on which these guidelines are based are briefly outlined in this document. A more detailed description of the scientific criteria and reference material is available in a separate publication entitled "Exposure Guidelines for Residential Indoor Air Quality: Supporting Documentation".

It is recognized that the above lists do not fully represent the range of compounds found in the home environment. As new data become available, the need for developing exposure guidelines for additional contaminants or groups and revising the current guidelines, should also be considered.

37.3.2.3 General "indicators" of indoor air quality

Carbon dioxide

Increasingly, complaints made by occupants of some large building are being linked with poor indoor air quality. The occurrence of some of these complaints (headache, fatigue, unpleasant odours, stuffiness and undue warmth) has been associated with elevated concentrations of carbon dioxide.

Whether a concentration range could be prescribed for carbon dioxide that would be indicative of acceptable air quality from the viewpoint of comfort or aesthetic considerations was therefore considered.

Carbon dioxide is produced by human respiration and can be reduced significantly only by ventilation of the building. The concentration of carbon dioxide in indoor air is therefore closely related to the ventilation rate.

The degree of ventilation needed to maintain carbon dioxide at a low level within a building also helps to reduce the levels of other indoor pollutants and to improve overall indoor air quality. Carbon dioxide is useful as an indicator of general air quality only in building where there are significant metabolic and/or combustion sources of carbon dioxide. Otherwise, carbon dioxide levels will be low over a wide range of ventilation rates (for example, in large houses with only one or two occupants and no unvented combustion appliances).

The trend in recent years towards minimizing ventilation within houses in order to reduce energy consumption for heating and cooling can lead to increases in carbon dioxide levels within residences and to a general deterioration in indoor air quality.

In several studies, comfort factors have been correlated with carbon dioxide concentrations. Collectively, these studies suggest that carbon dioxide concentrations above 1800 mg/m^3 (1000 ppm) are indicative that there is an inadequate supply of air, although complaints have been documented at concentrations as low as 1100 mg/m^3 (600 ppm). However, from a review of the direct physiological effects of exposure to carbon dioxide, as opposed to subjective symptoms, a higher maximum exposure concentration is recommended (see section "Carbon dioxide").

It must be noted that these studies were conducted in buildings with mechanical ventilation system and with occupancy rates quite different from those of residences. Moreover, the effects observed are probably not attributable to the presence of elevated concentrations of carbon dioxide, but rather to undesirable concentrations of other substances that result from inadequate ventilation, and for which carbon dioxide provides a suitable surrogate parameter. Therefore caution must be used in interpreting carbon dioxide concentrations as a general indication of residential indoor air quality.

Water vapour

Relative humidity has also been considered as an indicator of indoor air quality. Air-to-air heat exchangers which are designed to bring in outside air and to turn on and off at pre-set relative humidity levels, have been developed. The control on indoor air pollutants other than excessive water vapour by heat exchangers operating in this way may not be satisfactory for the following reasons:

- in a large house with a low occupancy, relative humidity may not rise sufficiently to trigger air exchange, although other pollutants may be present at unacceptable concentrations:
- changes in occupancy throughout the day affect the rate at which water vapour is generated, whereas other pollutants may be emitted continuously;

– the variation in indoor relative humidity with geographical area and season necessitates different control settings for each location and season.

Therefore, relative humidity is not suitable as a general indicator of residential indoor air quality.

37.3.3 Derivation of guidelines and recommendations

37.3.3.1 Data base for derivation of exposure guidelines

Since research into the health effects of residential indoor air quality is at an early stage, there is a dearth of reliable information on the health effects that result from exposure to the low levels and mixtures of contaminants likely to be found. In most cases, therefore, the Working Group relied upon the result of laboratory experiments using animals, clinical studies with human volunteers, and epidemiological investigations of urban air pollution and the occupational environment. The results of epidemiological and clinical studies are the most relevant for establishing acceptable levels of exposure of humans to air pollutants. Nevertheless, the application of each of these types of study involves a number of assumptions and hence uncertainty in the derived dose–response relationship.

Epidemiological studies

Most of the relevant epidemiological studies of populations are observational (non-experimental) in nature; that is, the allocation of individuals into study groups on the basis of exposure is not under the control of the investigator. Such observational studies can be further classified as descriptive (cross-sectional) or analytical (cohort or case-control) studies.

In cohort studies, exposure and outcome are monitored over time; as a result, the quality of evidence obtained from such longitudinal investigations is generally considered to be superior to that from cross-sectional studies, in which populations are monitored at on time. However, the results of all observational studies must be evaluated against the following features of study design;

a) *Estimation of exposure.* In most observational studies conducted to date, pollution data are usually obtained from one or several outdoor monitoring stations; however, the exposure burden can vary greatly between individuals living in the same neighbourhood because of local climatic conditions and special features of the indoor environment. For example, for pollutants generated mainly in the outdoor environment (oxidants, sulphur, dioxide), indoor concentrations will normally be less than those outdoors; however,

indoor levels (from indoor sources) may have greater temporal peaks with concomitant effects on health. Exposure to some pollutants (nitrogen dioxide, carbon monoxide) are not well represented by ambient air measurements if there are significant indoor sources of these pollutants. Only in very recent studies have investigators attempted to take such factors into account in estimating exposure.

b) *Role of confounding variables.* In observational studies of populations exposed to air pollutants, a host of confounding variables (e.g., socioeconomic status, smoking, occupational exposure, meteorological factors) many of which have greater effects than air pollution, must be considered.

c) *Measurement of outcome.* There is substantial variation in the method of measurement of many health-state indicators in the studies conducted to date; such indicators include lung function, hospital admissions and questionnaire, and responses may be biased by the way and conditions under which the questions are asked.

Even in those studies where the design is acceptable, interpretation of the results is complicated. For example, it is often difficult to attribute effects observed in populations exposed to air pollution to a single contaminant. It is possible that the contaminant investigated may serve as an indicator, or surrogate, for the effects of another contaminant or a combination of contaminants. Also, since exposures are not subject to the manipulation of the investigator, it is difficult to determine whether mean or peak concentrations, variability, or some other aspects of air pollution are the most important determinant of health effects.

In summary, those epidemiological studies considered most relevant for developing the guidelines have the following features:

- longitudinal study design;
- adequate control of appropriate confounding factors;
- some attempt to take individual variations in exposure into account, (e.g., area of residence, gas stove use).

Epidemiological investigations of the effects in the general population are considered to be most relevant. Studies of persons exposed to airborne pollutants in the workplace may not reflect potential problems of the general population, since the young, the elderly and other high-risk groups are not accounted for. Moreover, exposure periods and the mixture of pollutants will be different from those in the home.

Clinical studies

Clinical studies are generally, though not always, conducted in controlled laboratory environments. These studies probably provide the most reliable data from which to derive exposure-response relationships that form the basis

for air quality standards. However, clinical studies are restricted for ethical reasons to the examination of mild, temporary effects of short-term exposures in a limited number of subjects. As such, they are most suitable for developing short-term exposure limits.

A good clinical study should control for extraneous variables and experimental bias. In order to satisfy this need, most clinical studies employ a control group. Comparison with this group provides an indication of the effects that the experimental conditions exert on exposed individuals. The inclusion of such a group is very important if the effect of the experimental intervention is to be isolated.

In order to further reduce experimental bias in clinical studies, the allocation of subjects into experimental and control groups should be a random process. That is, all subjects should have an equal chance to be assigned to either the experimental group or the control. Through this process the study will have a good chance of obtaining comparable groups, such that the measured and unknown characteristics of the subjects at the time of group-allocation will be, on the average, evenly balanced between groups. Also, since most statistical procedures are based on normal distributions, the randomization procedure is necessary in order to meet the assumptions of the statistical design.

In order to reduce experimental bias to a minimum, some studies employ designs in which the subjects, and sometimes the investigators, are not aware of the particular intervention to which a subject has been assigned. The advantage of these "blind" designs is that they reduce the possibility that the subjects and/or investigators will favour certain outcomes based on group status.

Only through the employment of blind designs can the investigator be reasonably assured that extraneous variables and experimental bias have been controlled as much as possible. However, it is sometimes impossible to employ such a design for practical reasons. If, for example, the investigators are testing the pulmonary effects of ozone, and the subjects and investigators can detect the treatment condition by the smell of ozone, it is clearly not practical to attempt a blind study. This is a limitation of many clinical studies of airborne pollutants.

Animal studies

Although numerous studies of the effect of airborne pollutants in animal species have been conducted, levels of exposure have, in general, been much higher than those in ambient air. In addition, extrapolation of the results to ambient levels is complicated by the distinct anatomical differences between the respiratory tracts of animals and man. Also, studies are frequently con-

fined to unusually high concentrations of no more than one or two pollutants, rather than to the low concentrations and mixtures of substances generally found in the home. However, the results of such studies are useful in the identification of target organs and systems, in the clarification of mechanism of toxicity and in the assessment of carcinogenicity.

The reliability of carcinogenesis bioassays in animal species is evaluated on the basis of several features of the design and the results of the study.

These features include the size of the experiment (i.e., the numbers of exposed and control animals), the influence of environmental factors (e.g., diet); the route and method of exposure; the doses administered; the species, strain and sex of the animals; the types, site, incidence and time to the development of tumours; and the nature of the exposure–response relationship.

Information concerning the kinetics, metabolism and mechanism of action, and the results of epidemiological studies in human populations are also considered when the relevance of the results of carcinogenesis bioassays for man is assessed.

37.3.3.2 Approach used in deriving exposure guidelines

In order to establish accurate, defensible exposure guidelines it is essential to determinate the quantitative relation between a given pollutant and its effects. The terms "exposure–response" and "exposure–effect" denote this quantitative relationship. Owing to ethical considerations, such quantitative relationships are difficult to determine with precision in human populations. Nevertheless clinical and epidemiological studies combined with laboratory animal studies can provide a substantial amount of quantitative information concerning the effects of exposure to a given pollutant.

Non-carcinogenic substances

Regulatory agencies have traditionally attempted to determine a level of exposure below which there are no apparent detrimental effects. This so-called "threshold level" is closely related to the lowest level at which minimal, or reversible, effects can be observed — the "lowest-observable-adverse-effect level" (LOAEL). A safety factor may be incorporated into the derivation of a regulatory standard or guideline depending upon the number and quality of studies upon which the LOAEL is based. This approach has been used to establish guidelines for a number of indoor air pollutants considered in this document.

The size of the safety factor depends to a large degree on whether human rather than animal data are available, whether studies have been conducted

directly on those segments of the population believed to be at high risk, and the quality of the studies themselves. Ultimately the choice is based on a consensus decision by experts, but strictly has no scientifically defensible basis.

Owing to uncertainty concerning data obtained in observational studies, the World Health Organization has used a safety factor of two in recommending guidelines for daily and annual exposure to air pollutants; this value has been adopted in deriving some of the guidelines specified in this document.

In instances where there are sufficient data from reliable clinical studies of transient changes in groups at risk (for example, changes in pulmonary function in exercising asthmatics), no safety factor is incorporated in the derivation of a short-term exposure guideline.

Because of the wide variation in individual susceptibility to irritants, notably aldehydes, short-term exposure guidelines have been derived by applying a factor of five to the lowest value reported to cause a significant increase in symptoms of irritation.

It has been suggested that occupational hygiene limits could be adapted for the residential indoor environment by applying a safety factor to accommodate differences such as exposure times, pollutant mixtures and population sensitivities. Without a thorough knowledge of the scientific basis for the occupational limits, however, the Working Group considered such an approach to be scientifically indefensible.

Carcinogenic substances

There is evidence that for carcinogenic substances threshold levels may not exist. It follows in such cases that there is no level of exposure at which a hazard does not exist, although at very low concentrations the health risks may be so small as to be undetectable. For carcinogens, the derivation of acceptable exposure limits using experimentally derived LOAELs and safety factors is considered inappropriate.

Ideally, exposure to known or suspected carcinogens should be avoided. However, it is not possible to eliminate certain carcinogens from the environment. The maximum concentrations in the exposure ranges listed in this document for formaldehyde, a suspected carcinogenic substance, are the lowest levels that it is practical to achieve and at which there is no undue public health risk.

To ascertain the level of risk, or probability of adverse response at the low concentrations likely to be encountered in the environment, several statistical procedures have been developed to predict the shape of the dose–response curve at doses below those administered in experimental studies. The shape of the extrapolated dose–response curve can influence considerably the value

of the exposure concentrations at which there appears to be a negligible human health risk with estimates sometimes ranging over several orders of magnitude, depending on the mathematical model employed.

In deriving the long-term exposure guidelines for formaldehyde, the mathematical procedure for determining cancer risk was selected taking into account as far as possible information concerning the mechanisms of action. Nevertheless, it must be recognized that the calculated risk at low levels of exposure is probably overestimated owing to the conservative assumptions upon which the mathematical model is based.

37.3.3.3 Monitoring procedures

Methods for monitoring indoor air quality have not been standardized to this day. Many studies have been carried out with a combination of older methods and instruments (used also for ambient air monitoring) together with those that have only recently been developed. There is now a trend towards developing smaller portable instruments which can serve both as personal monitors and as stationary (area) monitors. Passive and active personal monitors that measure particulate matter (filters) and gases (absorbers and adsorbers) have been developed. The small size and quiet operation of this newer equipment render it suitable for use in indoor environments. Such monitors also offer the possibility of determining pollutant levels near the breathing zone of individuals, thus enabling direct estimates of personal exposure to be made.

Monitoring to ascertain compliance with the short-term exposure guidelines should be conducted when "worst case" situations are anticipated, and procedures should be designed to provide an accurate assessment of occupant's actual exposure. It is recommended that samples be taken when and where maximum concentrations are expected and that the averaging times specified along with the ASTER be used.

In the case of the long-term exposure it is more difficult to specify appropriate monitoring procedures. Sampling should be conduced over periods long enough to encompass any diurnal, seasonal or other temporal fluctuations. The magnitude and frequency of these fluctuations may vary considerably from contaminant to contaminant and be a function of dwelling type, location and the activity of the occupants. In many cases annual averages have served as a basis for formulating long-term air quality standards.

Frequently, annual averages are based on random sampling of 24-hour average readings. Monthly or weekly averages are typically determined using daily averages, although passive monitors now available may have collection periods of 7–90 days.

It is recommended that a minimum sampling period of 24 hours be used and that samples be taken when concentrations are expected to be at their highest. Should concentrations be outside the specified long-term exposure range, additional samples should be taken to ascertain whether levels are likely to remain elevated, or whether fluctuations will result in the long-term (often annual) average concentrations falling within the guideline value.

If contaminant levels are found to be outside any of the recommended exposure ranges, the source of the problem should be identified. Any possible corrective measures should be taken or advice sought from health authorities.

37.3.4 Guidelines and recommendations

37.3.4.1 Substances with exposure guidelines — non-carcinogenic effects

Aldehydes

In cases where more than one aldehyde is detected in indoor air, the sum

$$\frac{C_1}{C_1} + \frac{C_2}{C_2} + \frac{C_3}{C_3}$$

should not exceed 1, where C_1, C_2, and C_3 are the concentrations of formaldehyde, acrolein and acetaldehyde, respectively, as measured over a 5-minute period, and are as follows:

C_1 (formaldehyde): 120 μg/m^3 (0.10 ppm);

C_2 (acrolein): 50 μg/m^3 (0.02 ppm);

C_3 (acetaldehyde): 9000 μg/m^3 (5.0 ppm).

Aldehyde concentrations in indoor air generally exceed those outdoors. The primary sources include gas stoves, space heaters and tobacco smoke.

Identities of all the aldehydes produced during incomplete combustion of organic fuels are not yet known, but measurements in indoor locations have shown that formaldehyde, acetaldehyde and acrolein are the major aldehydes present.

Concentrations of acrolein in indoor air range from 2 to 50 μg/m^3 (0.001–0.02 ppm); limited data available indicate that levels of acetaldehyde average about 17 μg/m^3 and range from 1 to 48 μg/m^3.

The major effect on human health of airborne aldehydes is irritation of the eyes, nose and throat. In recently conducted clinical studies, significant increases in symptoms of irritation have been observed at levels of formaldehyde greater than 1200 μg/m^3 (1 ppm) (exposure periods 1.5–30 min).

Data derived from observational studies of populations exposed to formaldehyde in the occupational environment or in public or residential buildings

are less reliable owing to limitations of the investigations conducted to date. In the best-conducted studies, symptoms of irritation have not been associated with exposure to levels less than 600 μg/m^3 (0.5 ppm) (see section "Formaldehyde").

There are few reliable data available concerning levels of the other aldehydes that induce symptoms. Acrolein is one of the most irritating of the aldehydes identified in indoor air, with most people reporting eye irritation at levels of less than 1 mg/m^3. A significant increase in symptoms of eye irritation has been associated with exposure to levels as low as 210 μg/m^3 (0.09 ppm): in the same study, however, eye irritation at a concentration of 800 μg/m^3 was only slight. Severe irritation results from exposure to concentrations of 1900 μg/m^3 (0.8 ppm). Chronic effects following exposure to acrolein have not been reported and there has been no evidence of carcinogenicity in long-term bioassays with laboratory animals.

Acetaldehyde is considerably less irritating than acrolein; symptoms of irritation have been associated only with exposure to levels greater than 46 mg/m^3 (25 ppm). In a long-term bioassay in rats, significant increases in the incidence of nasal adenocarcinomas and squamous cell carcinomas were observed following inhalation of acetaldehyde: however, the administered dose levels (1400, 2700 and 500 mg/m^3; 750, 1500 and 3000 ppm) and mortality rates during the study were extremely high. Moreover, data given in the published report of the investigation were insufficient to permit meaningful quantitative risk estimation.

The recommended values for C_1, C_2 and C_3 are 5–10 times less than the concentrations reported to induce significant increases in symptoms of irritation. Concentrations satisfying the relationship given above should be low enough to minimize additive irritant effects of the specified aldehydes in the general population.

Carbon dioxide

Based on health considerations, the acceptable long-term exposure range (ALTER) for carbon dioxide in residential indoor air is ≤ 6300 mg/m^3 (≤3500 ppm).

Carbon dioxide is a colourless, odourless and non-flammable gas, which is produced by metabolic processes and by the combustion of fossil fuels. The average concentration of carbon dioxide in the atmosphere is about 620 mg/m^3 (~ 340 ppm), but levels vary widely with time and location. Indoor levels tend to be higher than outdoor levels. Gas stoves and unvented kerosene heaters are major sources of carbon dioxide indoors, but in poorly ventilated rooms, levels may exceed 5400 mg/m^3 (3000 ppm) from human metabolism alone.

An increase in the ambient level of carbon dioxide brings about a rise in the acidity of the blood and an increase in the rate and depth of breathing. Over

prolonged periods, of the order of days, regulation of blood carbon dioxide levels occurs by kidney action and the metabolism of bone calcium. The latter process leads to some demineralization of the bone. Exposure to levels of 27000 mg/m^3 (15000 ppm) or more for several days has induced reversible changes in the lung membrane of guinea pigs. In humans, exposures to carbon dioxide levels of over 90000 mg/m^3 (50000 ppm) have produced effects on the central nervous system such as headache and dizziness and visual distortions; there is some evidence of cardiovascular effects at similar concentrations. Subjective symptoms such as fatigue, headaches and an increased perception of warmth and unpleasant odours have been associated with carbon dioxide levels of 900–5800 mg/m^3 (500–3200 ppm). In some of these studies the symptoms may have been caused by other substances, with the carbon dioxide acting as a surrogate measure of air quality (Section 2.3.1).

The lowest concentration at which adverse health effects have been observed in humans is 12600 mg/m^3 (7000 ppm), at which level increased blood acidity has been observed after several weeks of continuous exposure. A maximum exposure level of 6300 mg/m^3 (3500 ppm) should provide a sufficient margin to protect against undesirable changes in the acid-base balance and subsequent adaptive changes such as the release of calcium from the bones. This level should also provide an adequate safety margin for sensitive groups. At such a level, the effect of carbon dioxide as a ventilatory stimulant is likely to be small and so would not greatly increase the dose received of other pollutants present in the air.

Changes in the acid-base balance and release of calcium from bones occur in response to chronic carbon dioxide exposure rather than to brief excursions in concentration. Thus, a short-term exposure range is not required for this substance.

Carbon monoxide

The acceptable short-term exposure ranges (ASTER) for carbon monoxide in residential indoor air are:

≤ 11 ppm: 8 hour average concentration

≤ 25 ppm: 1 hour average concentration.

Carbon monoxide is a colourless, odourless gas that is produced in the combustion of carbonaceous materials and also in human metabolism. It combines with haemoglobin to form carboxyhaemoglobin (COHb), which reduces the oxygen supply to body tissues. Endogenous levels of carboxyhaemoglobin are approximately 0.5% of the total haemoglobin (written 0.5 COHb%).

Sources of carbon monoxide in indoor air include gas and oil appliances, tobacco smoke, and the infiltration of carbon monoxide in polluted outdoor air. Outdoor levels of 0.05 to 0.9 mg/m^3 (0.04 to 0.8 ppm) have been measured in

rural areas, and levels as high as 57 mg/m^3 (50 ppm) have been found in urban areas, although levels of 1.1 to 11 mg/m^3 (1 to 10 ppm) are more typical. Indoor levels generally follow outdoor levels except in houses with unvented or poorly vented combustion appliances or where there is tobacco smoking; carbon monoxide levels of approximately 115 mg/m^3 (100 ppm) have been found in the kitchens of some houses immediately after gas stoves were used for cooking.

Exposure to carbon monoxide levels leading to carboxyhaemoglobin concentrations of approximately 2.5–10% has been shown to cause adverse effects on the cardiovascular system, to decrease exercise capacity and to impair psychomotor performance. Elevated carboxyhaemoglobin levels in women who smoked during pregnancy have been associated with low birth-weight and educational retardation of their children. Groups that may be at particular risk from the effects of carbon monoxide exposure include those with cardiovascular, cerebrovascular, and peripheral vascular diseases, foetuses, the newborn, pregnant women and individuals living at high altitude.

Experimental results suggest that, in general, such sensitive individuals can tolerate increases in carboxyhaemoglobin levels of up to 1.5 COHb%. The guidelines are intended to ensure that increase due to ambient carbon monoxide remain below this limit. Since carboxyhaemoglobin levels depend on the concentrations of both carbon monoxide and oxygen, levels are expressed only as ratios (parts per million by volume) so that the guidelines will be independent of ambient pressure.

Nitrogen dioxide

The acceptable exposure ranges for nitrogen dioxide in residential indoor air are:

ALTER: ≤ 100 μg/m^3 (< 0.052 ppm);

ASTER: ≤ 480 μg/m^3 (< 0.25 ppm) — 1 hour average concentration.

Nitrogen dioxide (NO_2) is the only oxide of nitrogen which has been shown to be detrimental to human health at concentrations that may be encountered in indoor air.

The primary outdoor sources of nitrogen dioxide are vehicular and industrial emissions. In general, nitrogen dioxide concentrations in urban atmospheres are higher than those in rural atmospheres, reflecting the large contribution of nitrogen dioxide from technological sources. In North America, the background level of nitrogen dioxide in rural areas is less than 19 μg/m^3 (0.010 ppm). In urban centres, nitrogen dioxide levels are at least double this value. During the period 1977–1981 the average of nitrogen dioxide annual means for Canadian urban centres decreased from 60 to 44 μg/m^3 (0.031 to 0.023 ppm). The highest annual mean reported in Canada for nitrogen dioxide (80 μg/m^3; 0.042 ppm) occurred at a commercial site in 1981.

Gas stoves and unvented combustion appliances are major sources of nitrogen dioxide indoors. The indoor/outdoor ratio of nitrogen dioxide concentrations is generally less than unity in dwellings in which there are no major indoor sources, and greater than unity in dwellings with gas stoves and/or other combustion appliances. Families living in rural or low-pollution areas and who use for cooking are exposed to indoor nitrogen dioxide levels of roughly 30 $\mu g/m^3$ (0.015 ppm), although average concentrations of 100 $\mu g/m^3$ (0.050 ppm) have been recorded in some homes.

Interpretation of the results of available epidemiological studies on health effects associated with nitrogen dioxide exposure is rendered difficult by the lack of accurate exposure data and by confounding factors such as exposure to other pollutants. Despite these limitations, the epidemiological studies have provided some useful data concerning exposure–effect relationships. In these studies an increased prevalence of respiratory illness in adults and children chronically exposed to mean levels of near 200 g/m^3 (0.10 ppm) nitrogen dioxide was observed.

The results of clinical studies indicate that normal and asthmatic subjects can experience detrimental respiratory effects when exposed for brief periods to concentrations of approximately 960 $\mu g/m^3$ (0.5 ppm). The short-term effects of nitrogen dioxide exposure below 960 $\mu g/m^3$ (0.5 ppm) have been examined in only a few studies. A "no-adverse-effect level" cannot be clearly identified from the results of these studies; therefore a safety factor of two was applied to arrive at the recommended short-term exposure range.

Ozone (oxidants)

The acceptable short-term exposure range (ASTER) for ozone in residential indoor air is ≤ 240 $\mu g/m^3$ (≤ 0.12 ppm) — 1 hour average concentration.

Infiltration of outdoor air is the principal source of oxidants in indoor air. Ozone, nitrogen dioxide, hydrogen peroxide and peroxyacylnitrates are photochemical oxidants that may be present in indoor air. Nitrogen dioxide is examined in the previous section. Of the remaining oxidants, ozone is the most prevalent. Concentrations of ozone indoors are generally much lower than those outdoors, but may approach outdoor levels if windows are open. Indoor concentrations of ozone follow outdoor fluctuations with a time lag of one hour or less. The average annual outdoor concentration for urban centres in Canada was 30 $\mu g/m^3$ (0.015 ppm) in 1979. Indoor ozone concentrations are typically less than 40 $\mu g/m^3$ (0.02 ppm), although peak levels of 200 to 400 $\mu g/m^3$ (0.1 to 0.2 ppm) have been reported. Ozone can be generated in the home by the arc of electric motors and by improperly installed or maintained electrostatic air cleaners.

Ozone is an irritant that can cause coughs, chest discomfort, and irritation of the nose, throat and trachea. Ozone consistently causes detrimental effects on the lung function of healthy subjects at concentrations at or above 600 $\mu g/m^3$ (0.30 ppm). Furthermore, ozone causes detrimental effects on the lung function of healthy subjects engaged in strenuous physical activity at concentrations lower than 600 $\mu g/m^3$ (0.30 ppm), possibly as low as 240 $\mu g/m^3$ (0.12 ppm). Results of epidemiological studies conducted to date support this finding. The available epidemiological data are, however, insufficient to serve as a basis for establishing an acceptable long-term exposure range.

Individuals exposed to concentrations of ozone between 200 and 800 $\mu g/m^3$ (0.10 and 0.40 ppm) have exhibited an adaptive response, at least in terms of lung function. At present, it is not known if this adaptation is beneficial or detrimental in the long term. Available data are insufficient to serve as a basis for establishing an acceptable long-term exposure range for ozone.

Particulate matter

The acceptable exposure ranges for fine particulate matter ($\leq$ 2.5 m mass-median aerodynamic diameter — MMAD) in residential indoor air are:

ALTER: $\leq 40\ \mu g/m^3$

ASTER: $\leq 100\ \mu g/m^3$ — 1 hour average concentration.

Airborne particulate matter is a mixture of physically and chemically diverse substances, present in air as suspensions of solids and/or liquid droplets varying in size from about 0.005 to 100 μm. The size range of concern when human health effects and indoor air quality are considered is from 0.1 to 10 μm in aerodynamic diameter, particles smaller than this generally being exhaled. Above 15 μm, most particles are too large to be inhaled. Virtually all particles between 10 and 15 μm are deposited in the nasopharyngeal region of the respiratory tract; health effects are associated primarily with the deposition of particles in the thoracic (tracheobronchial and pulmonary) regions. Particles have been further divided into a coarse fraction, normally around 2.5 μm and above, and a fine fraction under this size. It is this latter fraction that can reach the lung alveoli.

Indoor particles come from both indoor and outdoor sources, but the indoor matter differs in both size and chemical composition from that originating outdoors. Indoors, particles occur primarily in the fine fraction, because indoor sources such as combustion appliances and cigarettes tend to produce fine particles, and the building envelope acts as a partial filter to screen out larger particles. Indoor particulate matter contains a much higher fraction of organic matter than that of outdoor air, largely because of household activities such as cooking, cleaning and use of consumer products.

Indoor concentrations of fine particulate matter tend to be higher than those outdoors. Average concentrations of particles under 3.5 μm (respirable suspended particulates or RSP) range between 20 and 30 μg/m^3. Higher concentrations have been noted in "dirty" cities with high outdoor levels, and in homes with smokers or wood stoves. Cigarette smoke appears to be the most significant indoor sources of particulate matter, and the presence of resident smokers has been shown to raise levels of fine particles in homes by between 12 and 40 μg/m^3 per smoker.

Numerous epidemiological studies indicate that human health has improved as concentrations of airborne particulate matter have decreased. Despite the many uncertainties in these studies, they provide some useful information on levels at which adverse health effects might be expected. Increases in mortality have been observed especially among the elderly and those with pre-existing respiratory or cardiovascular disorders, when they were exposed to concentrations of particles (including coarse particles) above 500 μg/m^3 accompanied by high sulphur dioxide levels for periods of one to four days. Increases in hospital admissions and in respiratory clinic visits were also noted at about the same levels, while increased prevalence of respiratory symptoms and discomfort in persons at increased risk because of pre-existing respiratory conditions were first observed at levels in the range 250–350 μg/m^3. In children, marginal decrements in lung function lasting several weeks were also associated with short exposure at about these levels, which were correlated with outdoor and indoor RSP levels estimated to be about 80 μg/m^3.

Clinical studies, while not necessarily representing usual exposure conditions, also indicated that short exposure to concentrations of fine particulates (expressed as sulphuric acid) above 100 μg/m^3 could result in irritation and alterations in respiratory function in asthmatic subjects and in slowing of bronchial clearance in normal individuals.

Chronic exposure for periods of several years to moderate levels of airborne particles estimated to be around 180 μg/m^3 total suspended particulates or 80 μg/m^3 fine particles (respirable suspended particulates or RSP) appear to be correlated with increased prevalence of respiratory symptoms and chronic respiratory disease, accompanied by reduced respiratory-function measurements, in adults and children.

Sulphur dioxide

The acceptable exposure ranges for sulphur dioxide in residential indoor air are:

ALTER: ≤ 50 μg/m^3 (≤ 0.019 ppm)

ASTER: ≤ 1000 μg/m^3 (≤0.38 ppm) — 5 minute average concentration.

Sulphur dioxide is the main oxide of sulphur found in indoor air. Indoor

concentrations are generally lower than those outdoors by a factor of about two, primarily because most sources are outdoors, and sulphur dioxide is readily absorbed by furnishings and fabrics.

Interpretation of the results of available epidemiological studies on health effects associated with exposure to sulphur dioxide is complicated by a paucity of representative exposure data and by confounding factors such as exposure to other air pollutants. However, such studies have provided some useful albeit uncertain data concerning exposure–effect relationships.

Excess mortality, particularly among the elderly and those with pre-existing cardiopulmonary disease, has been observed in populations exposed to 24-hour pollution episodes in which sulphur dioxide concentrations exceeded 300 to 400 $\mu g/m^3$ (0.12 to 0.15 ppm). Increases in hospital admissions and emergency room visits have also been associated with exposure to these levels.

Increased prevalence of acute and chronic respiratory symptoms and impaired pulmonary function have been observed in adults and children exposed for extended periods (< 1 year) to mean levels of 100 $\mu g/m^3$ (0.038 ppm) sulphur dioxide.

Relevant data have also been obtained from clinical studies; however, exposure in such investigations are short and do not necessarily represent usual exposure conditions. In normal subjects, increased airway and nasal flow resistance and a change in the mucociliary flow rate have been observed following exposure to 2600 $\mu g/m^3$ (1.0 ppm) sulphur dioxide; reversible increases in specific airway resistance have been observed in asthmatics exposed by natural breathing for brief periods to concentrations exceeding 1000 $\mu g/m^3$ (0.38 ppm).

Water vapour

Based on health considerations, the acceptable short-term exposure ranges (ASTER) for water vapour in residential indoor air are:

30–80% relative humidity — summer;

30–55% relative humidity — winter (unless constrained by window condensation).

For purposes of indoor air quality, the most useful measure of water vapour levels is relative humidity, the ratio of the concentration of water vapour present to the concentration needed to saturate air at that temperature. Indoor humidity is determined by the humidity and temperature of outdoor air as well as by indoor sources and sinks of water vapour. The main indoor sources are human and animal metabolism, and such activities as bathing, cooking, and the washing and drying of clothes. Small amounts of water vapour are also produced by combustion.

The moisture content of indoor air is reduced by dilution with drier outdoor air, by condensation on cold surfaces, and by absorption or adsorption of water by materials in the home. Homes heated electrically are likely to have higher indoor relative humidities in winter than comparable homes heated by combustion furnaces, since the latter tend to increase the infiltration of dry outside air. Relative humidities in Canadian homes have been found to range from 21 to 68%.

In conjunction with temperature and air flow, relative humidity effects comfort, conditions of 20–60% relative humidity at temperatures between 20 and 25°C usually being judged comfortable. Long periods of low relative humidity are believed to cause dryness of the skin and mucous membranes, which may lead to chapping and irritation. High humidity at high temperatures leads to increased sweating and a loss of electrolytes from the blood; prolonged exposures may lead to heat exhaustion or heat stroke.

Groups that may be at particular risk from high humidity are those suffering from cardiovascular diseases, infants born two or three weeks before term, and the elderly. Arthritis sufferers have been found to experience increased symptoms when a rise in humidity accompanies a drop in atmospheric pressure. Sufferers from asthma develop symptoms of bronchoconstriction after exercise more readily when breathing air at low humidity.

Several species of bacteria and viruses survive best at low or high, rather than intermediate, humidities. Humidity levels above 50% have been found to increase the population size of moulds, fungi and mites that may cause allergies. The evidence suggests that humidity levels should be maintained between 40 and 50% to reduce the incidence of upper respiratory infections and to minimize adverse effects on people suffering from asthma or allergies. Such a range would be hard to maintain, however, and exposure to higher or lower levels is unlikely to affect the health of most people.

37.3.4.2 Substances with exposure guidelines — carcinogenic effects

For substances designated as human, or potential human carcinogens, a continuing effort should be made to reduce exposure to the lowest possible level. It is recognized that attainment of this goal must be considered in light of the cost and feasibility of remedial measures and technological changes. It was, therefore, considered desirable to specify exposure guidelines both in terms of what can be attained now (action level) and what should be striven for as a longer term objective (target level).

Formaldehyde

The exposure guidelines for formaldehyde in residential indoor air are:

Action level: 120 μg/m^3 (0.10 ppm)

Target level: 60 μg/m^3 (0.05 ppm)

Formaldehyde is a colourless gas with a pungent odour. It combines readily with water to form a non-volatile compound and has a tendency to be absorbed on to surfaces and textiles, such as carpets and curtains. An equilibrium is established between formaldehyde in air and that adsorbed on surfaces and within wood products such as particleboard.

Formaldehyde is formed naturally in the environment; outdoor concentrations in remote regions are about 7 μg/m^3 (0.006 ppm). Outdoor levels in Canadian locations are about 10 μg/m^3 (0.008 ppm). Man-made sources of formaldehyde include combustion and the decomposition of formaldehyde resins used in wood, paper, textiles or urea-formaldehyde foam insulation (UFFI). In two large Canadian surveys, average formaldehyde concentrations of 14 and 42 μg/m^3 (0.011 and 0.034 ppm) were found in homes that did not have UFFI; higher average levels were detected in homes with UFFI (66 μg/m^3; 0.054 ppm).

Levels in mobile homes which generally contain a high proportion of urea-formaldehyde resin pressed wood products tend to be even higher than the concentrations in homes with UFFI.

Formaldehyde is a vitally important intermediate in the normal metabolism of cells. It serves as a building-block for the synthesis of purines, pyrimidines and many amino acids and lipids and is a key molecule in one-carbon metabolism. It is present at low levels in body fluids, and exposure to exogenous sources does not lead to any appreciable increase in these levels (see section "Aldehydes").

Formaldehyde gas is a sensory irritant, primarily effecting the nasal passages, respiration and the eyes. In addition, in two well-conducted bioassays and a more limited study, formaldehyde has been carcinogenic in two strains of rats, producing a high incidence of nasal squamous cell carcinomas (38–50%) following administration of approximately 18 mg/m^3 (15 ppm). Formaldehyde is also genotoxic in a number of assays and is weakly mutagenic in cultured human cells as well as in other mammalian cells, Drosophila, fungi and bacteria. Although the epidemiological studies conducted to date provide little convincing evidence that formaldehyde is carcinogenic in human populations, the possibility cannot be excluded owing to limitations of the available data.

Because of the possible carcinogenicity of formaldehyde, it would be prudent to reduce indoor levels as much as possible. The action level of 120 μg/m^3 is the lowest concentration considered to be feasible at the present time.

However, it is recommended that in future, and where remedial measures are taken, every effort be made to reduce concentrations to below the target value (60 μg/m^3).

37.3.4.3 Substances with recommendations for controlling exposure

In examining the need for guidelines for pollutants listed in this part of the document, the scientific literature was reviewed as it was for the pollutants previously considered. The development of quantitative exposure guidelines was, however, considered inappropriate for a number of reasons:

- for some groups of substances the individual components may have widely differing toxicological properties; the complexity of the mixtures precluded establishing a guideline for each constituent or for the group as a whole;
- establishment of air quality guidelines may not be the appropriate strategy for control, especially where inhalation is not the most significant route of exposure;
- there are deficiencies in the scientific data base.

For these substances or groups, information on potential adverse health effects and possible sources has been provided, and recommendations that should help to eliminate or reduce exposure to them developed.

Biological agents

In order to prevent many of the common indoor problems due to biological agents, measures should be taken to ensure that:

- excess humidity and condensation are not present;
- surfaces are kept free of dust;
- stagnant water sources, such as humidifier tanks, are kept clean and occasionally disinfected;
- a high standard of appropriate personal hygiene is maintained.

The indoor environment can present the potential for illness due to exposure to biological agents. These agents may include microorganisms from humans, pests or insects within the home, or from growth on surfaces or in stagnant water. Dust from outside and inside the home includes pollens, spores, cells, cell debris and insects. Such materials in the air may cause infectious diseases or allergic illnesses in sensitized persons in the home. Illnesses must be systematically investigated in order to reveal the cause and to determine possible means of control. Control measures include disinfection and physical removal of the agent where possible and various means of controlling particulate matter and excess humidity. Due to differential individual sensitivity, however, in some cases extraordinary measures my be required to prevent symptoms from occurring.

The variety of biological agents that may occur in air is immense, and their potential for effects on susceptible individuals is unpredictable.

The complexity of the problem and the lack of data from which contamina-

tion levels can be related to disease incidence mean that it is not possible to recommend limits for biological agents in general.

Consumer products

It is recommended that exposures resulting from the use of consumer products be kept to a minimum by ensuring adequate ventilation and observing any other precautionary measures described on the product label, and in any accompanying information.

Pest control products should be used only when absolutely necessary.

(a) *Chlorinated hydrocarbons.* Large quantities of chlorinated hydrocarbons are produced and used annually, worldwide. They are present in the home environment principally as solvents, cleansers and aerosol propellants, and some individuals may be exposed to relatively high levels of them in the pursuit of hobbies. In some cases, chlorinated hydrocarbons may be released continuously from household products; they have also been detected in drinking water.

Chlorinated hydrocarbons are absorbed into the body principally by inhalation, but also through the skin and gastrointestinal tract; they tend to accumulate in fatty tissues such as brain, bone marrow and body fat. Recovery from the acute effects of exposure to the volatile chlorinated hydrocarbons is usually complete, but after repeated exposures, adverse health effects can include depression of, or permanent damage to, the central nervous system, irritation of the eyes and lungs, and damage to the skin, liver and kidneys. In the case of dichloromethane, a metabolite is carbon monoxide, which can cause cardiovascular stress (see section "Carbon Monoxide")

In the home, exposures occur primarily through the use of consumer products and can be of short duration, but levels may be sufficiently high to have the potential for adverse health effects.

(b) *Pest control products.* Pest control products comprise a very large number of diverse chemicals. They are widely used in and around the home environment, both by residents and by professional pest control applicators. These products are employed to control insects in the home, to prevent insect damage to fabrics, to treat house-plants against both insects and fungi, to treat pets, and to disinfect the air, water and surfaces around the home. Occasional inadvertent exposure may occur because of impregnation of building products or household articles with pesticidal preservatives and subsequent release of these into the air. Surfaces in the home may be coated with pesticides after normal use, and food prepared or stored in the room during or after pesticide use can also be contaminated. Pesticides may also infiltrate homes after outdoor application. Many pest control products contain, in addition to the active ingredient(s), non-active ingredients such as solvents, wetting agents and stabilizers. Such ingredients may have much

higher vapour pressures, and hence be present at higher airborne levels, than the active ingredients, and a few have biological activity of their own.

Exposure to pest control products in the home can occur both by inhalation and by absorption through the skin, for example, following contact with pesticide-treated surfaces. Pesticides may also be ingested following injudicious use in the vicinity of foods.

Available data on exposure levels in the home indicate that airborne levels of most pesticides are very low if products are used as directed. Misuse of pesticides, whether through failure to follow the instructions provided, through heavy use in areas where food is stored, prepared or eaten, through use in poorly ventilated spaces, or through misapplication of products designed for outdoor use, provides the greatest potential for exposure in the home, and levels can then be high enough to create a health hazard.

Adverse human health effects from exposure to low levels in the home tend to be non-specific, similar to ailments caused by many other chemical agents: respiratory effects, coughing, burning of eyes and nose, rhinitis, headache, dizziness, tiredness and general malaise. Two major groups of pesticides are anticholinesterase agents, causing augmentation of secretory activity in bronchial, lachrymal, salivary and other glands, and contraction of smooth muscles of the bronchioles. Allergic reactions to a few pesticides occur very occasionally. Most pesticides used in Canada today break down quickly in body tissues. No pesticides available for domestic use in Canada have been assessed as human carcinogens.

Pest control products are subject to extensive review and regulation by several federal and provincial departments before they are released to the market. This ensures that the consumer is provided only with products that, although potentially toxic, are safe if used as directed and that adequate instructions and warnings regarding the use of these products are also provided.

Thus, the consumer is ultimately responsible for the proper use of pesticides in the home.

(c) *Product aerosol.* An aerosol can be a suspension of fine liquid or solid particles in air or other gases; in these guidelines, product aerosols are considered to be consumer products that are dispersed under pressure from disposable containers.

An aerosol product has three main components: the active ingredient or ingredients, the propellant, and miscellaneous additives used to improve the product, such as plasticizers, synthetic resins, surfactants, and emulsifiers. Some products consist of gaseous components only and therefore do not result in the production of an aerosol.

Aerosol propellants used currently in Canada include hydrocarbons, such as propane, butane and isobutane; nitrous oxide; dichloromethane (which is

commonly called methylene chloride); carbon dioxide; dimethyl ether; and nitrogen. Nitrous oxide may also be used as the propellant for food products.

Product aerosols are formulated from a wide variety of chemicals, some of which are potentially toxic at high concentrations. Certain components may be subjected to review, and hence control, under federal legislation such as the Hazardous Products Act and the Environmental Contaminants Act. For example, prior to 1980, chlorofluorohydrocarbons were used as propellants for most aerosol sprays. Chlorofluorohydrocarbons are still used as aerosol propellants in many products, including aerosol cooking sprays, but trichlorofluoromethane and dichlorodifluoromethane have been banned, under the Environmental Contaminants Act, for use as propellants in hair sprays, deodorants and antiperspirants. Generally the use of product aerosols in the home is of short duration and intermittent, ranging from about once or twice a day for deodorant sprays to four times a year for oven cleaners.

Fibrous material

Precautions should be taken to minimize inhalation of, and skin contact with mineral fibres during home renovations and installation operations. Materials and products containing fibres should also be examined periodically for signs of deterioration. Advice should be sought before removing or damaging any materials thought to contain asbestos.

(a) *Asbestos* is the general term for six fibrous silicate minerals that are useful because of their high tensile strength, durability, flexibility and resistance to heat and chemicals. A large proportion of total asbestos production is used in the construction industry in materials such as asbestos-cement sheeting and pipes. Under normal conditions of use, asbestos fibres are not expected to be released from such materials. Fibres may, however, be released from friable surfaces (such as sprayed asbestos-containing insulation or low-density insulation blocks), or from other construction materials in the course of renovation or maintenance.

Available data indicate that, in general, asbestos levels in homes are not significantly greater than those in ambient air. Concentrations as much as three orders of magnitude higher have been measured during such operations as sanding of dry-wall taping compounds. Under the Hazardous products Act, asbestos is now prohibited in most consumer products where respirable dusts are generated during normal use.

Prolonged exposure to elevated levels of asbestos fibres causes asbestosis, lung cancer, mesothelioma and possibly laryngeal cancer and malignancies of the gastro-intestinal tract. The risk of developing asbestosis associated with exposure to levels of asbestos in indoor or ambient air is probably negligible. It is difficult to quantify the risks of lung cancers and mesothelioma associated

with exposure to indoor (and outdoor) levels owing, in part, to problems inherent in using data from historical epidemiological studies for extrapolation, but mainly owing to the complexity of asbestos itself (i.e., variations in risk associated with different sizes and properties of the fibres). Nevertheless, it is likely that these risks are small.

(b) *Man-made mineral fibres* (MMMF) include fibrous glass, mineral wool and ceramic fibres. Fibrous glass accounts for approximately 80% of all MMMF produced and is used mainly as thermal or acoustical insulation. There are few data on levels of fibrous glass in residences; mean levels measured during the installation of glass-fibre insulation have ranged from 0 to 8 fibres/ml. Levels in homes are probably no significantly above ambient levels except during such installations or modifications. Levels in public buildings have been found to range from 0 to 0.008 fibres/ml.

Glass fibres cause transient irritation of skin and eyes in workers occupationally exposed to them. Long-term studies have provided only equivocal evidence of respiratory disease as a result of exposure to glass fibres; however, an excess of lung cancer deaths (not consistently related to dose or duration) has been found in mineral wool workers 20 or more years after first exposure. Smaller excesses have been observed in fibrous glass workers exposed for more than 30 years. The available data indicate that man-made mineral fibres are less pathogenic than asbestos, possibly because of their size distribution and lower tendency to fragment in the lung.

Lead

In order to minimize the exposure of people, and especially children, to lead of airborne origin, it is recommended that surfaces that may be contaminated be cleaned frequently and that a high standard of overall cleanliness be maintained.

Airborne lead is present mainly as inorganic lead compounds in dust particles. More than 90% of the global emissions of airborne lead are from man-made sources, principally the combustion of leaded gasoline, followed by mining and smelting. Atmospheric levels of lead in remote areas are in the range 0.05–8 ng/m^3. Levels in urban areas depend upon proximity to roadways and industrial sources, and upon such factors as traffic density, wind speed and height above the ground. Annual geometric mean lead concentrations measured in Canada declined steadily from 0.74 μg/m^3 in 1973 to 0.27 μg/m^3 in 1982.

The major indoor source of airborne lead is the outside air, and indoor levels tend to be lower than outdoor levels.

People are exposed to airborne lead both directly by inhalation and indirectly by ingestion of lead that has settled as dust. In adults, approximately 10% of the ingested lead is absorbed; for young children the figure may be as

high as 53%. The amount of lead absorbed from the lungs is believed to range from 30 to 50% of the total inhaled lead.

Once lead is absorbed, it is distributed to the soft tissues and the skeleton. Lead in blood reflects current exposure to lead and has a biological half-life of about 16 days. Lead in the skeleton represents long-term accumulation and its half-life is several decades.

Lead can produce many toxic effects in the body. The main symptoms of lead poisoning include anaemia, abdominal cramps, constipation, renal damage and encephalopathy. Children are more sensitive than adults to the harmful effects of lead and may also experience irritability and loss of appetite. Learning impairment and alterations in neurobehavioural responses may occur at low exposure levels.

There is uncertainty in determining total exposure to airborne lead because of indirect exposure to lead of airborne origin that has settled as dust. Therefore it is not possible to derive an acceptable air lead level for the indoor environment.

Although lead is introduced into the domestic environment mainly as an airborne pollutant, the major pathway for exposure is through ingestion of dust once it has settled. Exposure to lead can be controlled to some extent in homes by frequent cleaning of surfaces, including food preparation areas.

Polycyclic aromatic hydrocarbons (PAHs)

Exposure to polycyclic aromatic hydrocarbons indoors should be kept to a minimum by:

- ensuring that any combustion systems, for example wood-and coal-burning stoves, are properly installed and maintained and operated under conditions of satisfactory ventilation;
- adhering to the guidelines and recommendations given in this document for particulate matter and tobacco smoke.

Polycyclic aromatic hydrocarbons (PAHs) are a large class of organic compounds, most of which are non-volatile solids that are very insoluble in water. They are frequently adsorbed onto the surfaces of particulates, and over 100 PAHs have been detected in airborne particulate matter.

Polycyclic aromatic hydrocarbons are produced when materials containing carbon and hydrogen are burned. Coal-burning and the use of internal combustion engines are reported to be major sources, although it has also been claimed that residential wood-burning is the major source of PAHs in the USA. Outdoor levels have been measured in the range 0.1–60 ng/m^3 in urban areas and 0.001–2 ng/m^3 in rural areas. Indoor levels are often dominated by levels in the outdoor air, but cooking (where charring of food occurs), improperly operating wood stoves and open fireplaces and tobacco smoking can add

significantly to indoor exposures. There are few quantitative data on levels of airborne PAHs in houses, and limitations in the available methods for collecting and measuring PAHs may mean that the available data are unreliable.

Exposure to PAHs is possible through skin contact, inhalation and ingestion. Although it has been estimated that ingestion of foods accounts for most of the exposure to PAHs, exposures through the dermal and inhalation routes appear to have more significant effects on human health. In particular, in some instances elevated concentrations of PAHs have been found in air, and the concern over exposure to airborne PAHs centres on the potential of these compounds to cause lung cancer.

There is a paucity of toxicological data (especially inhalation data) for most individual PAHs and almost no data for PAH mixtures. Epidemiological studies of humans are limited by the fact that exposures are usually to low levels of PAH mixtures, and often in the presence of other pollutants. As a result it is impossible to identify the effects of any particular PAH and to reliably quantify the risks to human health.

The lack of reliable data on which to base dose–response relationships and the difficulty of distinguishing the effects of PAHs in the environment from those of other pollutants prevent a guideline for exposure to PAHs in indoor air from being assigned. Since some PAHs are known to be carcinogenic, exposure to these substances should be minimized.

Tobacco smoke

In view of the carcinogenic properties of tobacco smoke, it is recommended that any exposure to tobacco smoke in indoor environments be avoided.

Tobacco smoke is a complex mixture of substances, including carbon dioxide, carbon monoxide, oxides of nitrogen, and a large number of organic vapours and solids. Over fifty of the components are known to cause adverse health effects; twelve (including vinyl chloride, 2-naphthylamine, benzo(a)pyrene and formaldehyde) are known or suspected carcinogens. Carbon dioxide, carbon monoxide, oxides of nitrogen, formaldehyde and particulate matter are among the components of tobacco smoke for which individual air quality guidelines are recommended elsewhere in this document.

The largest amounts of most components are found in the smoke emitted into the environment directly from the burning end of the cigarette. Symptoms reported by non-smokers exposed to such "sidestream" smoke include eye, nose and throat irritation, headache, nausea, dizziness and loss of appetite. Furthermore, the lingering odour and reduced visibility from tobacco smoke are aesthetically unpleasant to many people.

Increased risks of lung cancer have been observed in non-smoking populations exposed to sidestream smoke. Other suspected health effects of tobacco

smoke on non-smokers include the aggravation of such conditions as asthma and angina pectoris, increased risks of spontaneous abortion, congenital malformation or sudden infant death syndrome in the children of smoking mothers, and retarded development of children whose mothers were exposed to tobacco smoke during pregnancy. Increased risks of respiratory diseases have been observed among children whose parents were smokers, and non-smoking wives of smokers have been found to have increased risks of death from nasal sinus cancer and ischaemic health disease.

Estimates have been made showing that non-smokers repeatedly exposed to tobacco smoke are at a significantly higher risk of contracting tobacco-smoke-inducted lung cancer. While these calculations involve a number of assumptions that lead to uncertainty in the actual magnitude of the hazard, it is widely believed that there is no level of exposure to carcinogenic substances below which a risk does not exist.

Summary of exposure guidelines

Contaminants	Acceptable exposure ranges	
	ASTER	ALTER
Aldehydes (total)	$\Sigma C_i/C_i \leq 1$ [a]	—
Carbon dioxide	—	≤6300 mg/m^3 (≤3550 ppm)
Carbon monoxide	≤11 ppm — 8 h [b]	—
	≤25 ppm — 1 h [b]	
Formaldehyde	(c)	(d)
Nitrogen dioxide	≤480 μg/m^3 (≤0.25 ppm) — 1 h	≤100 μg/m^3 (≤0.05 ppm)
Ozone	≤240 μg/m^3 (≤0.12 ppm) — 1 h	—
Particulate matter[e]	≤100 μg/m^3 — 1 h	≤40 μg/m^3
Sulphur dioxide	≤1000 μg/m^3 (≤0.38 ppm) — 5 m	≤50 μg/m^3 (≤0.019 ppm)
Water vapour	30–80% RH — summer	—
	30–55% RH — winter[f]	

[a] C_i = 120 μg/m^3 (formaldehyde); 50 μg/m^3 (acrolein); 9000 μg/m^3 (acetaldehyde), and C_i are respective concentrations measured over a 5-min period.
[b] Units given only in parts per million so that guidelines are independent of ambient pressure.
[c] See Aldehydes (total).
[d] See page 908.
[e] ≤2.5 μg mass median aerodynamic diameter (MMMD).
[f] Unless moist constrained by window condensation.

Summary of exposure control recommendations

Contaminant	Recommendation
Biological agents	In order to prevent many of the common indoor problems due to biological agents, measures should be taken to ensure that: – excess humidity and condensation are not present; – surfaces are kept clear of dust; – stagnant water sources, such as humidifier tanks are kept clean and occasionally disinfected; – a high standard of appropriate personal hygiene is maintained.
Consumer products (chlorinated hydrocarbons, pest control aerosols)	It is recommended that exposures resulting from the use of consumer products be kept to a minimum by ensuring adequate ventilation and observing any other precautionary measures described on the product label and in any accompanying information. Pesticides should be used only when absolutely necessary.
Fibrous materials	Precautions should be taken to minimize inhalation of, and skin contact with, mineral fibres during home renovations and installation operations. Material and products containing fibres should be examined periodically for signs of deterioration. Advice should be sought before removing or damaging any materials thought to contain asbestos.
Lead	In order to minimize the exposure of people, and especially children, to lead of airborne origin, it is recommended that surfaces which may be contaminated be cleaned frequently and that a high standard of overall cleanliness be maintained.
Polycyclic aromatic hydrocarbons (PAHs)	Exposure to PAHs indoors should be kept to a minimum by: ensuring that combustion systems, for example wood- and coal-burning stoves are properly installed and maintained and operated under conditions of satisfactory ventilation; adhering to the guidelines and recommendations given in this document for particulate matter and tobacco smoke.
Tobacco smoke	In view of the carcinogenic properties of tobacco smoke, any exposure to tobacco smoke in indoor environments should be avoided.

37.4 THE EUROPEAN UNION ACTIVITIES

37.4.1 Introduction

European collaboration in the field of indoor air quality started in 1986 as a European Concerted Action which was part of the EC cost-shared Environmental Research Programme. At the end of 1987 the action became a COST activity (COST project 613/1) thereby opening it to the participation of EFTA countries. Since 1991 the European Collaborative Action "Indoor Air Quality and Its Impact on Man" has been continuing this work as part of the Environment Programme of the EC's Joint Research Centre (JRC), Ispra, Italy. Fourteen countries and the JRC's Environment Institute are participating in it. Since its start in 1986 the Indoor Pollution Unit of the JRC's Environment Institute has been supplying the secretariat and scientific and managerial support to the action.

37.4.2 Scope and purpose of the European Collaborative Action "Indoor Air Quality and its Impact on Man"

The European Collaborative Action (ECA) examines Indoor Air Quality (IAQ), defined as all features of indoor air having an impact on man. It deals with all aspects of the indoor environment including temperature, humidity and other environmental factors which may interact with indoor air quality.

The purpose of the action is to help construct and maintain healthy and energy efficient buildings within the EC. The activities focus on sources of pollution and their emission, ventilation, exposure to pollutants and the impact of IAQ on health/comfort in residential, public and non-industrial occupational indoor environments. It will contribute to prenormative research needed by EC services in their mission to prevent pollution and to promote health, comfort and quality of life.

37.4.3 Implementation of the European Collaborative Action

A Steering Committee composed of representatives of all participating countries, the JRC and other Commission Services decides the working programme and discusses and evaluates the results of the work performed.

In view of the fact that the ECA (as the former COST project) has no research funds of its own, the Steering Committee has essentially three means to achieve its objectives:

- development and validation of guidelines and reference methods for indoor related investigations and measurements or for measures to improve indoor air quality;
- collation, synthesis and dissemination of knowledge and data;
- organization of workshops, symposia, seminars and similar venues.

In particular the Steering Committee

- identifies ongoing research within the participating countries and the major research needs;
- establishes working groups for well defined tasks such as the development and/or validation of guidelines;
- provides for exchange of information and collaboration with other international and national organizations active in the field of indoor air quality (e.g. WHO, NATO/CCMS, U.S. EPA).

Typical tasks for Working Groups are to

- develop working instruments like guidelines for measurements and investigations in order to promote the efficiency of research and the comparability of results;
- perform intercomparison exercises for the validation of measurement guidelines;
- assess the status of knowledge in specific areas and propose solutions for indoor air quality problems.

37.4.4 Performed activities

The work performed so far by the European Collaborative Action (and the former COST project) has been summarized in reports published by the Commission of the European Communities: five summary reports on key issues of indoor air pollution, four guidelines on how to perform complex measurements and investigations, a guideline on ventilation requirements, and a report on an interlaboratory comparison experiment. The reports are briefly summarized in the following.

Summary reports

In an attempt to overcome the increasing difficulty of having essential information at hand in a concise form, the Steering Committee, assisted by the Secretariat and by Working Groups, has issued summary reports on single pollutants with high priority. Three such reports have been published: "*Radon in Indoor Air*" (ECA, 1988), "*Indoor Pollution NO_2 in_European Countries*" (ECA, 1989b) and "*Indoor Air Pollution by Formaldehyde in European Countries*" (ECA,1990). In all these reports health effects, IAQ standards or guide-

lines, sources, occurring concentrations, preventive measures and (where applicable) national and Community policies are briefly addressed.

"*Effects of indoor air pollution on human health*" are addressed in a fourth summary report (ECA, 1991b). The report describes in separate chapters broad categories of adverse health effects and relevant indoor exposures. The following health effect categories have been considered: effects on the respiratory system, allergy and other effects on the immune system, cancer and effects on reproduction, effects on the skin and mucous membranes in the eyes, nose and throat, sensory effects and other effects on the nervous system, effects on the cardiovascular system, and systemic effects on the liver, kidney and gastro-intestinal system. For each of these categories effects associated with IAP, the principal agents and sources, evidence linking IAP to the effect(s), susceptible groups, the public health relevance , methods for assessment, and major research needs are briefly discussed.

"*Biological particles in indoor environments*" (ECA, 1993a). This report summarizes knowledge on biological particles in indoor environments and makes some recommendations on how to conduct related indoor investigations, although the authors deemed present knowledge insufficient for a guideline on sampling and analysis of biological particles in indoor environments. The report deals with four major categories of biological particles in the air of private houses, non-industrial workplaces and public buildings (excluding hospitals). These particles are mites and their faeces; dander from pets and other furred animals; fungi, including moulds and yeasts; and bacteria, including actinomycetes. For each of these categories the following items have been considered: health effects; occurrence; available sampling methods; available methods of analysis; recommendations for different studies; and observed values and evaluation of results. Health effects, occurrence and sampling and analysis of Legionella are also discussed briefly.

Guidelines

The following guidelines have been published:

"*Sick Building Syndrome (SBS) — a practical guide*" (ECA, 1989c). This report describes the phenomenon of complaints on poor indoor air quality in large buildings, the extent of this problem, the symptomatology and diagnosis of SBS and related risk factors. Subsequently a procedure for conducting building associated investigation is described which includes 4 steps: technical and hygiene investigations. inspection and guiding measurements, measurements of ventilation, climate indicators and other implicated factors, and medical examination and associated investigations.

"Strategy for sampling chemical substances in indoor air" (ECA, 1989d). This report focuses on the fact (often overlooked) that sampling and analysis of indoor pollutants are only the last steps in a process which primarily needs to establish why, where, when and under which environmental conditions an air sample should be analyzed. The report presents general considerations regarding the dynamics of the indoor environment, the sampling objectives, time of sampling, duration and frequency of sampling, the sampling location, and discusses briefly quality assurance. More specific recommendations are made on sampling strategies for formaldehyde, nitrogen dioxide, suspended particulate matter, asbestos, radon, and volatile organic compounds (VOC).

"*Formaldehyde emissions from wood based materials: guideline for the determination of steady state concentrations in test chambers*" (ECA, 1989a). The guideline describes a method for the determination of formaldehyde emissions from wood based materials using large-scale, walk-in type environmental chambers. The guideline describes essential features of the chambers to be used, such as size, inner wall and sealing materials, tightness, air circulation and position of sensors for temperature and humidity. Moreover values for temperature, relative humidity, air exchange rate, loading factor and air velocity in the chamber are recommended. The guideline also deals with sample preparation and positioning in the chamber, and with formaldehyde sampling and analysis. Questions of quality control are also discussed. The guideline is presently being validated by a CEN[1] working group.

"*Guideline for the characterization of volatile organic compounds emitted from indoor materials and products using small test chambers*" (ECA, 1991a). The guidelines describes and makes recommendations for: small test chambers and ancillary equipment, sample collection and analysis, experimental design and data analysis. The techniques described are useful for both routine product testing and for in depth investigations by indoor air quality researchers. This guideline is presently being validated by several interlaboratory comparison exercises organized by the JRC in the framework of the ECA with the additional participation of North American laboratories.

"*Guidelines for ventilation requirements in buildings*" (ECA, 1992). The guidelines presented in this report introduce a new concept for the assessment of ventilation requirements: it considers not only the occupants but also the building and its equipment as sources of pollution. Ventilation requirements are established in two steps: in the first step ventilation requirements for avoiding adverse health effects are determined. In the second step a decision is made on the level of perceived air quality aimed for in the ventilated space. Three different comfort levels are suggested. Subsequently the pollution load on the air

1 Comité Européenne de la Normalition.

is determined by adding the loads caused by the building and by the occupants. Based on these loads, the available outdoor air quality and the ventilation effectiveness, the ventilation rate required to provide the desired indoor air quality is calculated. The two separately determined ventilation rates required for health and for comfort are compared and the highest value is used for design.

In addition to these guidelines the report "*Determination of VOCs emitted from Indoor materials and products: interlaboratory comparison of small chamber measurements*" (COST, 1993) summarizes the results of three experiments jointly performed by twenty laboratories in Europe and the USA and aimed at validating the guidelines for the characterization of volatile organic compounds emitted from indoor materials and products using small test chambers. The experiments yielded the following main results. Chambers of different materials (glass and stainless steel) and of widely different capacity (0.035–1475 l) appeared equally suitable, however the smaller the chambers the greater is the influence of inhomogeneities of the tested materials. The repeatability of duplicate measurements (including sampling) within each laboratory was good. The test with a known *n*-dodecane source showed, for most laboratories, an unexpected and yet unexplained discrepancy.

The interlaboratory agreement appeared reasonable (coefficient of variation 26–42%) when testing a PVC tile, but for emissions from a wax sample, the scatter was unacceptably high.

37.4.5 Ongoing work and continuation

The ECA is presently engaged in various projects the most important of which is aimed at a procedure for the evaluation of VOC emissions from building materials taking in consideration chemical, sensory, biological, toxicological, exposure and public health aspects.

Recently a working group has been established which, in collaboration with WHO Euro, will endeavour to find a definition and guideline value for TVOC (total volatile organic compounds). The group will analyse questions such as whether the TVOC concept is meaningful from an effect point of view, if so, which is the best combination of VOCs as an indicator of exposure, and how such a combination should be measured.

Further ongoing work is aimed at:

- identifying ways to integrate good IAQ and a rational use of energy;
- the improvement of a small chamber method for the characterization of VOC emissions from indoor materials and products;
- proposing strategies for VOC measurements in indoor air;

37.5 GUIDELINES FOR INDOOR AIR QUALITY IN NORWAY

By commission of the Directorate of Health in Norway, a working group was initiated in 1988 to develop guidelines for non-industrial indoor air quality in Norway. The working group reviewed the literature on some common and important air pollutants found in the indoor environment. The compounds of concern for which criteria documents were developed, were volatile organic compounds, carbon dioxide, carbon monoxide, nitrogen dioxide, formaldehyde, particulates of various types, radon and tobacco smoke. The norms regarding indoor air quality in Norway were issued by the Norwegian Directorate of Health on 10 Sept. 1991. The guidelines are summarized below.

VOC	400 $\mu g/m^3$ (total amount)
Formaldehyde	100 $\mu g/m^3$
Carbon dioxide	1800 $\mu g/m^3$ (maximum value)
Carbon monoxide	10 mg/m^3 (8-h mean)
	25 mg/m^3 (1-h mean)
Nitrogen dioxide	100 $\mu g/m^3$ (24-h mean)
	200 $\mu g/m^3$ (1-h mean)
Suspended particles	40 $\mu g/m^3$ (RSP range 0.1–2.5 μm)
	90 $\mu g/m^3$ (SP 0.1–10 μm)
Asbestos	No free asbestos fibres in indoor environment. No exact levels given, but strict source control and cleaning are essential.
Mites	1 μg *Dermatophagoides pteronyssinus* I-allergen per gram dust, i.e. approx. 50 mites per gram dust.
Microorganisms	No pathogenic microorganisms should exist. The level of others should be kept as low as possible. Mouldy smell should not occur indoors.
Synthetic mineral fibres (MMMF)	Exact levels regarding health effects cannot be given, but free fibres should not occur in indoor air.
Radon	Future buildings: projected to be less than 200 Bq/m^3. Existing buildings: between 200 and 800 Bq/m^3 — simple measures should be taken; above 800 Bq/m^3 — more comprehensive and costly actions are justified, but even simple measures could be effective. Aiming at a level below 200 Bq/m^3 after the measures have been taken.
Tobacco smoke (ETS)	Referring to the law against tobacco smoking and guidelines as a consequence of this.

37.6 THE INDOOR AIR QUALITY PROGRAMME OF THE WHO REGIONAL OFFICE FOR EUROPE

The indoor air quality (IAQ) programme of WHO/EURO was initiated in the mid-seventies when realizing that over 70% of the general population spends its time indoors, such as in homes, office buildings, schools, hospitals, transportation means, etc. The first meeting of experts on health aspects related to IAQ was convened in 1979, probably being the first international meeting on IAQ with participation from Eastern and Western Europe and North America. Eight more meetings followed between 1982 and 1993, discussing the "sick building" syndrome, IAQ research, formaldehyde and radon, organic pollutants, biological contaminants, combustion products, mineral fibres, and health risks from exposure to radon.

Following the establishment of the WHO European Centre for Environment and Health, the responsibility for the WHO/EURO programme on indoor air quality was transferred to the Bilthoven Division. In July 1994, a consultation was held at the Centre to identify priorities for work on health aspects of indoor air pollution. As a first in a series of activities, guiding principles for exposure assessment in indoor environments will be developed in 1994/95.

37.6.1 Introduction

The indoor air environment, as a subject, can be conveniently divided into two components:

(a) indoor climate, which deals with temperature, humidity and ventilation (including air conditioning), and determines human comfort; and

(b) indoor air quality, which includes physical (e.g., radon), chemical and biological pollutants, and relates to human health.

This section describes the development by the World Health Organization Regional Office for Europe (WHO/EURO) in Copenhagen of its programme on IAQ.

WHO/EURO consolidated in the late sixties its traditional environmental sanitation activities, undertaken for many years, and converted it into a new elaborate long-term programme on environmental health. This programme included a component on atmospheric air pollution and control. By 1974 it was recognized that IAQ was another subject with a serious impact on human health yet untouched by WHO. Prior to that time there were very few published reports and research papers available on IAQ, no international forum addressed this subject, and no national health authority paid any attention to it. Yet it had become obvious that, at least in the highly developed and industrialized countries such as in Europe, the majority of the general popu-

lation was spending most of its time indoors ("indoors" in the broadest meaning of the word), within enclosed spaces, such as in homes, in educational and cultural structures (i.e., schools, theatres, museums), general working places (i.e., offices and professional buildings), medical, health and sports facilities (i.e., hospitals, clinics, gyms, closed swimming pools), and most transportation means (i.e., cars, buses, aeroplanes, trains, ships, and respective land, air and sea terminals). When dealing with IAQ from a public health viewpoint (in contrast to the industrial hygiene situation), one is also concerned with people working in enclosed spaces, where the air quality is not directly affected by the nature of their work.

IAQ activities were proposed for inclusion into WHO/EURO's programme of work in 1974 and the first planning meeting took place in 1978.

37.6.2 Health aspects related to IAQ

The planning meeting was followed by the first WHO/EURO working group of experts on health aspects related to indoor air quality, convened in The Netherlands in April 1979. This was probably the first international meeting on IAQ, with the participation of scientists from various European countries — both East and West — and North America. The experts recognized that two major developments have made urgently necessary an assessment of the health aspects of IAQ. The first, rapid increases in the price of energy for heating buildings and the need to reduce energy use had led to measures to reduce the rate of natural and forced ventilation in buildings. To the extent that these efforts were successful, IAQ was bound to deteriorate further. In fact, designers and engineers faced with a maximum rate of air change requirement would tend to aim for even less than such a maximum, further aggravating the problem.

The second development of great concern was the introduction of building materials, furnishings and consumer products which released harmful contaminants to the indoor atmosphere. The Group discussed the effects of soil surfaces and building materials which emit radioactive radon gas, asbestos-containing materials which can release asbestos fibres, and insulation materials and processed wood which release formaldehyde. The Group also considered the wide range of consumer products such as personal hygiene products, cleaning agents, biocides, air fresheners, solvents and adhesives, and hobby and home craft materials, all of which can discharge a large number of harmful contaminants in appreciable quantities and high concentration to the indoor environment.

The Group reviewed certain aspects of IAQ which have always been of concern but which have an even more serious impact with drastically reduced ventilation, such as body odours and those produced by cooking and tobacco

smoking. The health consequences of increased respirable suspended particulate matter (RSP) and CO from tobacco smoke and of increased NO_2, CO and aldehydes from the use of unvented combustion of gas for cooking and water heating were also discussed. CO_2 and water vapour produced by man and his activities in a low ventilation indoor environment can have direct and indirect effects on the health of occupants. Lowered ventilation rates affect the concentration of viable particles indoors and thus the transmission of infectious disease, especially in cases of high density occupancy such as in schools and other public buildings.

A final report, including conclusions and recommendations, was published the same year (WHO/EURO, 1979). The results of that meeting have raised immediate interest in some countries. The first to respond, and soon, was Health and Welfare Canada — the Canadian national health authorities — who had consequently developed a national programme of research on the different aspects of IAQ. Health and Welfare Canada was also the first national health authority in the world to publish in April 1987 a set of national guidelines on IAQ entitled Exposure Guidelines for Residential Indoor Air quality.

37.6.3 Exposure and health effects

The second working group was convened in the Federal Republic of Germany in 1982 to review recent work on exposure to indoor air pollutants and to assess adverse health effects. As more data had become available since the first meeting in 1979, this meeting reexamined the IAQ issues and went into more details. The Group paid particular attention to a problem of great importance called, for no better term, the "sick building" syndrome. Some people in such buildings feel some kind of sickness, a phenomenon which has not yet been well enough understood. The meeting also dealt with the assessment of health effects on the basis of animal toxicological studies, appropriate occupational studies (which serve as a basis for comparison, but refer to much higher concentrations of pollutants), controlled exposure studies in man, designs of sequential studies and epidemiological studies. The methodology and priorities of future studies were also discussed.

The Group assessed the adequacy of current knowledge about the nature and strength of the sources of indoor air pollution and their distribution. It considered the status of available measuring equipment and the adequacy of current knowledge about population exposure. The group also reviewed the adverse health effects that have been reported in conjunction with indoor air pollutants and the current level of knowledge about the exposure–response relationships for each of the pollutant categories of interest.

The state of knowledge on population exposure to a number of pollutants was considered. The pollutants were environmental tobacco smoke (ETS), NO_2, CO, radon and daughters, formaldehyde, SO_2, CO_2, O_3, asbestos, non-asbestos mineral fibres, organic substances and allergens. For each of the pollutants the group estimated the fraction of the population exposed to low and to excessive concentrations. It also rated the adequacy of knowledge about sources, their characteristics and distribution, the adequacy of available measuring equipment and of actual monitoring data. The health effects associated with each of the pollutants were considered, as were the adequacy of knowledge about the exposure-response relationships and about the population exposed. Based on current though inadequate levels of knowledge, an attempt was made to identify the concentration level below which exposures would not be substantial public health concern, as well as the concentration level above which serious public health concern would exist. This assessment was made for most, but no all, of the pollutants. Much of the information accumulated during this exercise was compiled into three tables, becoming a major achievement of the working group, and a basis for repeated reviews and updating in future meetings (see Annex 1).

Attention was given also to methods and priorities for further research on assessing exposure. An evaluation was made of the rapid increase in a number of countries of the "sick building" syndrome in which occupants of large non-industrial buildings complain of a set of symptoms, the underlying cause of which is usually difficult to establish. Consideration was given to the factors that would have to be taken into account if criteria, guidelines and standards for outdoor air, or for the occupational environment, were to be applied to the non-occupational indoor environment. finally, ways of assessing the health effects of indoor air pollutants were considered and priorities for future work on the assessment of exposure–response relationships and on the impact of indoor air pollution on health were identified. a final report, including conclusions and recommendations, was published in 1983 (WHO/EURO, 1983).

37.6.4 Indoor air quality research

The third working group was organized in Stockholm in conjunction with and immediately following the Third International Conference on Indoor Air Quality and Climate, 20–24 August 1984, with the purpose of reviewing recent work in IAQ and examining new developments in research methodology. A full report, including conclusions and recommendations, was published in 1985 (WHO/EURO, 1985).

The Group began with a review of the field as it emerged from the international conference. Substantial progress was being made in the field of IAQ and

especially in the description of its effects on the occupants of buildings. The interest in and the level of activity related to the formaldehyde issue have seen a moderate decline, with an increase of interest in radon, in the issue of "sick buildings", and in volatile organic compounds in the indoor environment. There was also an increased level of interest in, but little corresponding progress in the determination or estimation of the overall impact on public health of different indoor air pollutants.

The Group discussed the development of strategies and policies aimed at effectively reducing exposure to indoor air pollutants. It was concluded that in the indoor environment the reduction of exposure was the responsibility of the occupants, the owners or operators, architects and engineers, suppliers of materials and products and, ultimately, different forms of local and national government. Because of this divided responsibility, the design and implementation of effective control strategies offer special challengers that deserve attention.

The Group reviewed the needs that existed in the development of laboratory research. The characterization of emission rates of materials has a high priority, especially in the area of volatile organic compounds and radon. Inorganic contaminants require relatively less urgent development in the laboratory. The development and validation of biological monitoring methods deserve high priority, as do the development and use of interlaboratory comparisons, which are an important element in any design for quality assurance and quality control of measurements made in the field.

Much of the discussion for the remainder of the meeting was devoted to the current status of research methodology in the field and in the study of associated health effects. The nature of problem makes it desirable to establish "population laboratories" consisting of a large number of buildings with their inhabitants, which should be representative of a much larger region, or of a whole country, in terms of building stock characteristics and the demographic characteristics of the population. Buildings and inhabitants in such a laboratory should be well enough described to allow the selection of subsets with very specific combinations of characteristics, which would permit the effective study of one or a very few risk factors, while minimizing interference by other factors. It was also agreed that field studies, whenever undertaken, should consider as many indoor air pollutants simultaneously as possible, and that health outcomes should be studied and recorded at the same time.

37.6.5 "Sick building" syndrome

Although considerable progress has been made in recent years in this investigation, the methodology is not adequate to clarify the mechanisms of the reactions that occur under such conditions. there is a growing feeling that

the number of instances of "sick building" syndrome is much larger than was originally thought. In fact, quite large populations suffer from the complaints associated with "sick building" syndrome. It was agreed that the application of industrial hygiene measurements or of more sophisticated analytical techniques did not usually increase understanding of the causes of the syndrome. It was agreed that the application of industrial hygiene measurements or of more sophisticated analytical techniques did not usually increase understanding of the causes of the syndrome. A staged sequence of observation and analysis of a problem in a particular building is a more effective approach, starting with a competent evaluation of the architectural and engineering plants, inspection of the site and evaluation of the maintenance and operating procedures. If this does not suggest a solution, a simple but standardized questionnaire and simple measurements should be applied and the results evaluated before much more complex and sophisticated measurements are even considered.

The first "sick buildings" were recognized prior to 1960, and since then there have been increasing numbers of case reports in several countries. The symptoms reported, primarily in the Scandinavian countries and the United States, are of a broad spectrum but have many features in common, such as: eye, nose and throat irritation; sensation of dry mucous membranes and skin; erythema; mental fatigue; headaches, high frequency of airway infections and cough; hoarseness, wheezing, itching and unspecific hypersensitivity; and nausea and dizziness. The number of case reports describing similar symptoms is now so large that it is reasonable to assume that we are dealing with a true environmental health problem.

One category of so-called temporarily "sick buildings" comprises either newly constructed or newly remodelled buildings, where the symptoms decrease in time and mostly disappear after approximately half a year. It is possible that the decline in symptoms is due to evaporation of the volatile compounds in building materials, paints, etc. In the second category of permanently "sick buildings", the symptoms persist for years and are sometimes resistant even to extensive remedial action. Normally no obvious cause is evident in this category, even after extensive investigations of the composition of the air and performance of the ventilation system, and of the building structure itself. However, it appears that such buildings have certain features in common:

1. They almost always have a forced ventilation system, usually serving the whole building, or large sections of it, and relying on partial recirculation of the air. Some buildings have an inappropriately located air intake, while other use heat exchangers that transfer pollutants form the return air into the air supply.

2. The indoor surfaces are to a large extent covered with textiles, including wall-to-wall carpets, and other features of the interior design favour a large interior surface-to-volume ratio.
3. They are energy-efficient, are kept relatively warm, and have a homogeneous thermal environment.
4. They are characterized by airtight building envelopes (windows often cannot be opened).

Indoor air contains a complex pattern of sensory stimuli, but although patterns of pollution vary, patterns of response are almost identical. No single irritant, therefore, is likely to be responsible. Usually, the sensation of dry mucous membranes is not noticeable in the "sick building" syndrome. The onset is gradual and the duration long, compared with the psychogenic illness characterized by hyperventilation, headache, nausea and dizziness, by symptoms related to specific illness, by a sudden onset preceded by a triggering event such as a strange odour and by a duration of days or weeks, although repeated relapses may occur. The symptoms can be differentiated from ubiquitous syndromes found in control buildings; they are the more acute symptoms of discomfort, annoyance and irritation such as eye and throat irritation, odour, sneezing and stuffy or sunny nose. Acute diseases such as colds, productive cough, asthma and fever are also widespread. Psychogenic symptoms may also be present in some cases, and this will require much more investigation.

Thermal factors often play a role, in which case it is recommended that an analysis of the thermal environment is carried out to ensure that temperatures and temperature gradients are not excessive, and that clothing, air humidity and air velocity are appropriate. The homogeneous thermal indoor environment and the homogeneous air quality produced by recirculation may increase the probability of locally generated pollutants spreading throughout the building and exposing all the occupants to them for a long period of time.

As regards airborne irritant pollutants, one should ensure that formaldehyde is not present in adverse concentrations. It should be noted that other irritants may cause symptoms almost identical to those produced by formaldehyde. Such irritants may originate from building and surface materials, furnishings, and human use of household or hobby products. At present very little is known about interactions among low-level irritants. It is possible that, in the case of some compounds at subthreshold concentrations, a summation or potentiation takes place, causing sensory reactions to the mixture of pollutants. It is also possible that chemical reactions take place, converting less irritating compounds to more irritating ones. It is usually not possible to find suitable controls to establish the background prevalence of complaints in a comparable population.

The Group also discussed methodic approaches to be chosen for investigating the problem. It examined the extent of the problem, what population groups would be at risk, how should health outcomes be measured, how to conduct practical investigations and how to design studies, and finally, proposed some remedial measures (WHO/EURO, 1985).

37.6.6 Radon and formaldehyde

The fourth working group met in 1985 in Yugoslavia to assess the risk to health and recommend air quality guidelines for radon and formaldehyde, two pollutants that may adversely affect IAQ. They have already become well-known issues, and a lot of questions have been raised at different governmental levels. The case of formaldehyde from home insulation in Canada is well known, and radon has been identified as a pollutant prevailing in more situations than originally expected.

Aspects discussed with respect to radon were its sources (soil, building material, tap water, and cooking gas), observed levels, roots of exposure (drinking-water, food and air), health effects, evaluation of human health hazards, strategies for identification and control and characterization of sites for future homes. Much of the natural background radiation to which the general population is exposed comes from the decay of radium-226 which produces radon gas and other products. Because radium is a trace element in most rock and soil, radon can be produced indoors by a wide variety of substances, from building materials such as concrete or brick to the soil under building foundations. Tap water taken from wells or underground springs may be an additional source. tests indicate that indoor concentrations of radon and its decay products are often higher than those outdoors. The effects of exposure to radon and its decay products consist predominantly of an increased risk of lung cancer, especially for smokers.

With regard to formaldehyde, the experts dealt, again, with sources, occurrence in air, roots of exposure (drinking-water, food and air), occupational exposure, smoking, skin absorption and blood exchange. Also discussed were the kinetics and metabolism (including absorption, distribution, biotransformation and elimination), health effects, organoleptic properties, and evaluation of human health hazards. Perhaps best known as an embalming fluid, formaldehyde is a common ingredient in foam insulation, furniture, carpets, curtains and other household items. Its commonest use is a component of resins used as bonding agents in plywood and chipboard. Formaldehyde is also a by-product of combustion, e.g., natural gas used in cooking and home heating. Formaldehyde can have effects on health ranging from acute nausea, eye irritation and respiratory impairment to more serious long-term effects.

Formaldehyde levels have been measured at several energy-efficient research houses. Studies have shown that concentrations exceeded 0.14 mg/m^3 in a number of new residential buildings and mobile homes with fewer than 0.3 air changes per hour. Formaldehyde makes its presence known by acute and continuous odour and irritation, causing predominantly discomfort although a cancer risk in humans cannot be ruled out.

In the case of radon, levels are mostly determined by the site characteristics and secondarily by the characteristics of the structure. In the case of formaldehyde, the level is determined by the building characteristics and the presence of sources of formaldehyde in the structure or its furnishings, or by occupant activities. For both pollutants the rate of air exchange with the outdoors is important and, for both, the control or avoidance of the sources is the most effective way of reducing exposure to them. The Group devoted considerable effort also to identifying available remedies for the reduction exposure to both these pollutants. The final report includes conclusions and recommendations (WHO/EURO, 1986).

37.6.7 Organic pollutants

The fifth working group was organized in connection with, and immediately following, the fourth International Conference on Indoor Air Quality and Climate in Berlin (West), held 17–21 August 1987. The Group considered the state of knowledge of organic pollutants in indoor air, and assessed what is known about the adverse effects on health that could result from current levels of exposure. Organic indoor air pollution was a difficult, little known and little understood subject. The meeting examined the chemical characterization of indoor organic pollutants and their distribution, estimated the population exposure, characterization of indoor organic pollutants and their distribution, estimated the population exposure, characterized health effects, discussed sensory effects, and analyzed systemic toxic effects, genotoxicity and carcinogenicity. As a class of contaminants, organic compounds in indoor air are extremely diverse; in the last decade hundreds of such chemicals have been identified. Although most occur in extremely low concentrations, there is appropriate concern about the effect on human health. Some of the compounds are genotoxic and many exhibit toxic, irritant and/or odorant properties.

Committees on indoor pollutants have always mentioned volatile organic compounds (VOC) as an important category of indoor air pollutants. Most of these review listed sources of such pollutants and their possible effects on health, but refrained from a detailed assessment of the total effect on public health. The reluctance to make this type of assessment is public health. The reluctance to make this of assessment is understandable because of the very

large number of chemical species reported, the lack of systematic studies of concentrations, and the distribution of these concentrations over the very large number of indoor areas in which people are exposed. In addition, knowledge of the adverse effects on health associated with such a large number of compounds to which people are exposed is also very limited.

To address the very legitimate questions produced by the increased concern, it was necessary to assemble the combined knowledge and insight of environmental chemists about the species of chemicals encountered, their concentrations and the distribution of such concentrations. Also, the knowledge and insight of toxicologists, experimental psychologists and environmental and occupational epidemiologists needed to be applied, together with those of the chemists, to construct an overall assessment of the public health impact. Since the public health impact consists of the products of the exposure distribution, the exposure–effect relationship and the number of people exposed, its assessment required the simultaneous and coordinated participation of all the relevant disciplines. It was necessary to consolidate the available data about concentration distributions from several larger studies in which the VOC in a number of residential spaces had been measured systematically. Although these studies had been carried out in different countries with somewhat different objectives and protocols, there was quite good agreement between the results. The environmental chemists were thus able to produce systematic and consolidated exposure data for consideration by the whole Group. At the same time, toxicologists, physicians and environmental and occupational epidemiologists reviewed the health effects data for the organic compounds that had been measured in the larger scale surveys, and presented their findings to the Group.

Psychologists, expert in sensory effects, similarly reviewed the existing knowledge about sensory effects of the same compounds and presented these findings to the Group. This simultaneous and coordinated review produced much clearer insights into the total impact, and at the same time led to the identification of areas where important and necessary information was most lacking.

The Group reviewed the findings from a number of systematic studies of personal exposure and of studies of residential indoor environments and integrated and consolidated the results, giving for each compound of significance the distribution of concentrations that had been reported. It was found that the concentrations and the species of VOC were very similar in the studies considered. In each case, these concentrations were higher than the corresponding outdoor concentrations, indicating significant sources and emissions of these compounds in the indoor environment.

The Group also considered the state of knowledge about the health effects associated with each of these compounds, and in each case evaluated these

effects both at high concentrations and at the concentrations reported in indoor environments. These effects cover a wide variety of responses ranging from unwanted sensory effects such as odours and sensory irritation, through toxic effects that modify or interfere with the normal functions of organs or injure tissues, to genotoxic effects. These effects have different time characteristics, ranging from acute perception of odour with subsequent adaptation, through irritant action that grows more severe with prolonged exposure to the delayed expression of the effects of genotoxicity with accumulation of exposure. Similar, sensory effects have a concentration threshold below the response does not occur, and this is also true for most of the toxicity that effects tissues and organ system, but it is generally accepted that genotoxic effects have no such threshold. There are some compounds that are thought to have important effects for which there is inadequate knowledge of population exposure, and there is a set of compounds that has been measured for which one cannot indicate public health impact at present.

Some of the chemicals, such as benzene, have been shown to be carcinogenic in humans while others, such as trichloroethylene, carbon tetrachloride and chloroform, have been demonstrated to be carcinogenic in rodents and are suspected human carcinogens. At the concentrations found in indoor environments, their contribution to causation of cancer in the population is judged negligible to minimal. The Group identified a number of organic contaminants where exposure distributions are inadequately known but which cause potentially more concern. Such contaminations are exemplified by the complex mixture of ETS, soot from unvented combustion, and those associated with some forms of cooking. Similarly, biocides used in and about the home need to be evaluated with respect to the hazard associated with current usage.

Only limited exposure studies have been conducted for organic compounds in ETS, including nitrosamines, aromatic amines, acrolein and aldehydes; these are important and deserve further evaluation. Only limited studies have been conducted for polynuclear aromatic hydrocarbons and for benzo(a)pyrene specifically; they are from various combustion sources, out- and indoors (with an indoor/outdoor ratio sometimes close to 1.0). Other possibly important genotoxic compounds need source strength determinations, as well as exposure assessments, including other non-chlorinated alkenes and epoxy resins, and constituents of polyurethane resins. For the large majority of the compounds involved, the concentrations reported in the indoor environment are several orders of magnitude lower than the concentrations at which adverse health effects have been reported. Many organic compounds found in indoor air have been reported to have toxic effects, but most of these reports concern industrial occupational exposures at concentrations considerably higher than those reported in non-industrial indoor environments. The Group considered

many of the most ubiquitous organic compounds reported in indoor air, and concluded that the reported concentrations in indoor air surveyed were several orders of magnitude below the level at which systemic effects have been reported for each compound.

The same was found to be true for irritation characterized by tissue damage.

For sensory effects, this separation is much smaller and for some compounds it is absent.

Organic compounds in indoor air can produce sensory effects such as perceptions of odour and irritation, which can severely affect human health and well-being. Some organic compounds have been shown to modify behaviour through their effect on the central nervous system at concentrations found in some industrial environments. In non-industrial environments, however, concentrations are several orders of magnitude lower. Although considerable progress was noted in the evaluation of sensory effects of organic compounds in indoor air, this evaluation remains a difficult *and complex problem*. The effects are mostly acute, often transitory and usually reversible, but the total number of people involved can be quite large. As a result, the total public health importance and the effect of reduce well-being on quality of life and productivity is likely to be significant but as yet difficult to quantify.

The Group discussed methods that allow the simultaneous incorporation of exposure distribution data, carcinogenic unit risk estimate or other exposure–effect relationships, background incidence of the illness under consideration, and any workplace or indoor standards and guidelines into an overall impact on the health of the population of current exposure to a specific organic compound. Such standardized approaches are necessary to develop priorities for research and for mitigating exposure.

In the final assessment of the health impact of a compound on a population base, it is necessary to consider the distributed exposure in connection with the adverse effects on health for that exposure. To this end, an approach has been developed to link the exposure distribution to the effect on health in a convenient way. One must bear in mind, however, that there are many uncertainties and sources of variance in the estimations, one of which is the distribution of exposure and another is the distribution of sensitivities among the population. For the Group's assessment, the assumption was made that the distribution of sensitivities is independent of the distribution of exposure at the prevailing levels.

In the United States in 1986 a national committee was assigned the task of preparing an IAQ research programme for the U.S. EPA. The Group's conclusions and recommendations are given in Appendix II.

37.6.8 Biological contaminants

The sixth working group met in 1988 in Finland to consider the state of knowledge on suspended viable particles, aero-allergens and other biologically derived suspended material, to examine the nature and magnitude of their adverse effects on human health and wellbeing, and to evaluate the role of buildings, building systems and contents in producing and disseminating such pollutants. By doing so, the experts dealt again with a little known and little understood IAQ issue, and have broken new ground with the discussion results (WHO/EURO, 1990) and their respective conclusions and recommendations (see Appendix II).

Viable particles suspended in air, and other biologically derived particles indoors, form a distinct class of contaminants. When they are present in indoor air, even in small quantities, they can have a powerful effect on occupants. This effect can be through infection of the occupant by a suspended infectious agent, in which case the organism multiplies in the new host and can produce illness. There can also be allergic or irritant effects characterized by reactions ranging from uncomfortable to disabling.

Some infectious diseases such as tuberculosis, Legionnaires' Disease or measles have been demonstrated to spread through airborne transmission of the infectious agent; airborne transmission may play a contributory role in others. In many allergic conditions, building occupants can develop a hypersensitivity to secretions or to fragments or other products derived from animals and plants. Viruses do not multiply in buildings, but may spread from human and a few animal sources. Buildings can contribute to the airborne spread of viral disease, either through overcrowding or by the spread or airborne viruses through the ventilation system Lack of an adequate supply of outdoor air will also increase the likelihood of airborne transmission of infectious disease, through an increase in the concentration of suspended viruses and bacteria in droplet nuclei. Exposure to airborne biological contaminants contributes to morbidity in the population. There is a wide variety of biologically derived materials in the indoor environment, and these are associated with a range of illnesses. In presenting these agents and their associated illnesses, it was important to rate their frequency and severity as well as the contribution to total incidence attributable to indoor exposure.

There are many diseases that have been associated with problems of IAQ. The following conditions may sometimes be associated with or caused by biologically derived aerosols, as well as having causes unrelated to the building environment: rhinitis, sinusitis, otitis, conjunctivitis, pneumonia, asthma, alveolitis, humidifier fever, bronchopulmonary aspergillosis, contact dermatitis, atopic eczema, contact urticaria, mycotoxicosis, allergy, and pseudo-aller-

gic reactions. These conditions have been defined in the report on the basis of the medical literature.

Much attention has been devoted to pathogenic organisms that multiply in buildings and building systems, such as *Legionella pneumophila* which causes Legionnaires' Disease (legionellosis) and non-pneumonic Legionnaires' Disease (Pontiac fever). Factors in buildings such as crowding and recirculated ventilation air can also promote the spread of airborne pathogens emitted by occupants suffering from tuberculosis, measles, varicella and other diseases. Assessment of the effect of biologically derived aerosols has to be made for the medical management of individual patients, or for populations in the case of an outbreak in an exposed population. The connection between exposure and outcome is often not easy to make. Since the overall process usually begins with a presenting clinical outcome either in an individual or in a population, the first discussion has concerned itself primarily with outcome assessment.

When examining the hazards associated with environmental exposure, a number of issues were reviewed, including infectious and allergic mechanisms, inhalation of mycotoxins and irritation described from bio-aerosols indoors.

Environmental measurement and sampling were carefully reviewed. The discussion of sources and control of microbiological contaminants included reference to ventilation, moisture, temperature, building microbiology, biologically derived particles from animals, pollen and fungi, bacteria and viruses brought indoors, strategies for control, building design and construction materials, ventilation and filtration systems, humidity control and air conditioning, cleaning, maintenance and repair, behavioural factors, and social, economic and regulatory considerations. Ventilation system components such as cooling tower, air chillers and humidifiers and dehumidifiers can support the growth of fungi, bacteria and other microorganisms. These can also grow on the structural parts of a building if the relative humidity inside the building reaches 70% or more, while dust mites can multiply in furnishings. Such microorganisms or products excreted by them or by arthropods and larger animals can be introduced into the indoor air, causing a variety of allergic and irritant reactions in the occupants.

Excessive concentrations of water vapour and accompanying condensation, water leak, failures of equipment drains or lack of cleaning and maintenance can all contribute to the introduction of viable or biologically derived contaminants into building ventilation air. A sizeable proportion of the population is, or is capable of being, sensitized over a lifetime to these forms of biological air contaminants. Other allergenic and toxic contaminants are animal dander, fragments of mites and other arthropods, and aerosols formed from animal faeces and urine. The combined effect of all the biological air contaminants in indoor air is thought to account for a substantial proportion of absenteeism in

schools and workplaces and of the days where activity is restricted. In the general population, five to ten days of restricted activity per head per year is a normal average. By reducing biological air contaminants indoors, acute infections and allergic episodes could be significantly reduced. It was pointed out that, in any building, the cost of losses in productivity due to absenteeism and restricted activity far exceeds the total cost of operating and maintaining the heating, cooling and ventilation systems. Because of the usual division of responsibility and authority in organizations occupying buildings, the relationship between these costs is not often considered.

37.6.9 Combustion products

The seventh working group was convened in Charleston, SEC, USA, in 1989, to consider the state of knowledge of combustion products in indoor air, their distributions in the population, and to assess what is known about the adverse health effects which might be caused by the population exposures.

The Group considered the progress made in recent years in characterizing the distributions of the exposures to combustion products as these had been recorded in recent years. The findings in this area from a number of different systematic studies were reviewed and the findings were integrated into a general assessment of exposures in the industrialized world. The presence of unvented combustion sources in the residential environment was found to dominate the indoor concentrations and exposures to the combustion products they contributed. In indoor environments, in which tobacco is smoked, RSP is determined primarily by the level of smoking rather than by either other indoor sources, or by the contribution from RSP from the outdoor environment. Similarly, when unvented appliances are used for space heating, their contribution to indoor NO_2 concentrations dominates that of all other sources, including that from the infiltration of outdoor NO_2. Unvented space heaters are also major sources of water vapour which can bring about multiplication of fungi, bacteria and insects. (These biological agents can produce a variety of aerosols and odours with biological activity which in turn produces adverse health impacts in occupants, a subject dealt with the previous chapter.)

The Group considered the state of knowledge about the adverse health effects of indoor air contaminants from vented and unvented combustion occurring indoors. These considerations were limited to the range and distribution of the concentrations of different indoor air pollutants produced by indoor combustion. The major pollutants considered were CO, NO_2, SO_2, CO_2, water vapour and RSP. A number of other combustion products produced in lower concentrations were also considered, such as benzene, benzo(a)pyrene, formaldehyde and dimethylnitrosamine. Water vapour occupies a special role

as a combustion product in that the optimum relative humidity in the indoor environment is between 30 and 60%. Relative humidities below 20% will aggravate irritation of mucous membranes of the eyes, nose and throat, and relative humidities above 70% will promote microbiological growth which, in turn, is likely to produce biological air contaminants in the indoor space. Vented combustion appliances will normally introduce only small amounts of combustion products into the indoor space. Under certain conditions, including high wind velocities and mechanical malfunctions, it is possible for even a vented heating appliance to discharge substantial amounts of combustion products into the indoor space it serves. The Group found that the level of protection from such events by current standards, codes and practices required review and, where necessary, improvement.

Exposure to combustion products indoors in the absence of indoor sources is generally lower than outdoors. Whenever unvented combustion sources occur, there is a great likelihood that the total human exposure to combustion products will be dominated by indoor exposures. The Group's conclusions and recommendations are given in Appendix I.

37.6.10 Inorganic fibres and other particulate matter

The Eighth Working Group, which was organized the previous week in Kingston, Ontario, in conjunction with the present International Conference on Indoor Air Quality and Climate in Toronto, Canada, concentrated on particulate matter from asbestos, man-made mineral fibres, rockwool and such like.

The Working Group considered mineral fibres which are of interest in common, non-industrial environments. Man-made mineral fibres (MMMF), most of which are also called man-made vitreous fibres (MMVF), are manufactured from various minerals, including glass and rock. They can be made as a continuous filament, in the form of insulation wool, or as refractory or special-purpose fibres. Asbestos fibres exist in a variety of forms with different qualities, but commercially they have been used mostly in the form of chrysotile. The Working Group evaluated the application of all these fibres in the indoor environment, including the sources and sinks for fibres. Most release of mineral fibres in indoor air is associated with installation, maintenance and retrofitting of fibrous products.

The Group reviewed the analytical methods used in the characterization and measurement of mineral fibres and their concentration. In the indoor environment, fibres of organic origin often dominate the total fibres found, making their recognition very important. The uses of phase-contrast optical microscopy (PCM), scanning electron microscopy (SEM), and transmission

electron microscopy were considered, with TEM having the greatest capacities for resolution and analysis of thin fibres. Sampling strategies can range from sedimentation plates for screening purposes to membrane filter and pump combination for quantitative measurements. The length of the sampling period should reflect the processes which might release fibres, as well as occupancy patterns.

Mean respirable fibre concentrations of MMMF in residences and public buildings have been reported at 40–200 F/m^3 of air. Outdoor air has been reported to contain MMMF at concentrations of 400–1700 F/m^3 in urban environments, and 40 F/m^3 in rural air. Asbestos fibres longer than 5 μm can be found in the indoor air in buildings at mean concentrations in the range of 70–200 F/m^3; if all asbestos fibres of any length are counted, then mean concentrations of $(0.5–1.0) \times 10^6$ F/m^3 can be obtained.

The Working Group considered the state of kowledge concerning the adverse health effects associated with exposure to mineral fibres of different types. Potential effects are interstitial pulmonary fibrosis (IPF), lung cancer and mesothelioma. The Group reviewed the recent toxicological data obtained in animal testing, and also the status of the major epidemiological studies involving cohorts of industrial workers. New toxicological studies using improved methodologies are showing animal carcinogenesis for ceramic fibres and for other MMMFs when the length is more than 5 μm and the fibre diameter is 1 μm or less.

The improved methods include uniform sizing of the fibres and better ways of administering them, which makes it possible to introduce higher lung burdens into toxicological evaluations. New data also support a model in which the durability of the mineral fibre, its length and its diameter determine the toxicological effects in animal models.

Epidemiological studies have been limited in number, and are based on cohorts of workers in different mineral-fibre industries. The exposure/effect relationships are difficult to establish because the exposures are very poorly documented. With the best estimates of exposure it appears that different industrial processes produce different slopes for the exposure/effect relationship for a given mineral fibre. In the asbestos–cement industry, the risk for a given exposure is considerably lower than the same exposure in the asbestos–textile industry. The risk of mesothelioma seems to be much lower for chrysotile and anthophyllite than for crocidolite and amosite, and even for crocidolite the risk appears to depend on the percentage of fibres with a diameter of 0.1 μm or less.

For an asbestos fibre concentration indoors of 100 F/m^3, the lifetime excess risk of cancer is estimated to be in the order of 1 in 10^5 to 1 in 10^7. The Working Group considered different methods of prevention of exposure in indoor environments,

ranging from recommending against the use of carcinogenic mineral fibres in buildings to implementation of detailed administrative procedures to assure effective containment. Such approaches should begin with a documented inventory, inspection and maintenance schedules aimed at preventing releases of fibres, and education and training of responsible personnel in the safeguards required. It is sometimes advantageous to enclose the asbestos-containing material, or to encapsulate it with a bonding agent or a sealer. The most extreme form of asbestos control is to remove it completely from a building using suitably safe methods, and to dispose of it in an approved manner.

Based on this evaluation, the group made the following recommendations:

1. Standard methods for measuring indoor inorganic fibres should be established so that they can be determined and reported in terms of fibre length and diameter, with speciation of fibre type. Indoor samples should be collected over a one-week period.
2. Prior to their use in building products, mineral fibres should be systematically investigated with respect to the exposure and health effects likely to result.
3. All construction and operating documents, including those listing an inventory of inorganic fibre-containing materials in the building should be kept by the building manager, and referred to in the event of maintenance and repair.
4. The use of carcinogenic mineral fibres (such as asbestos and erionite) in construction should be avoided wherever reasonably possible.
5. Exposure to airborne mineral fibres should be kept as low as possible. Particular attention should be given to the management of loose, soft, friable and accessible mineral fibre-containing products.
6. Mineral fibres should be coated or covered with other materials to prevent fibre release and to minimize exposure of the public by inhalation and skin contact. In the event of contamination, mineral-fibre dust should be removed from the affected area before normal activity is resumed.
7. Precautions should be taken to reduce airborne-fibre exposure of people who disturb mineral fibre-containing materials during maintenance and repair.

The Ninth Working Group was convened by the WHO Regional Office for Europe in Eilat, Israel, from 28 March to 4 April 1993, to assess the significance of risks to health associated with exposure to indoor radon, with a view to developing risk management policies and strategies based on a health criteria approach. Twenty-two temporary advisors from eleven European countries, China, Israel, and the United States were invited to participate. Representatives from the WHO International Agency for Research on Cancer

(IARC) and the Regional Office for Europe were in attendance. Seven working papers were prepared for this meeting and eleven additional papers were submitted by participants and some other non-attending scientists for the attention of the Group. The Group examined the indoor radon issue by discussing related risks to health, risk management and communication of risk information to the general public.

The main question concerning risks to health addressed by the Group was to what extend is exposure to radon a public health risk? They reviewed the data available from epidemiological studies of the occupational exposure of miners and residential exposures, animal experiments and other relevant experimental work. Based on these data, radon has been confirmed as a human carcinogen. A number of recent case-control studies of non-occupational public exposure were reviewed. This included the largest and most recently reported study (Stockholm, March 1993), carried out by Pershagen and colleagues in Sweden of 1360 lung cancer cases and two control groups of 2847 people from the same area. One of the control groups was matched on vital status. This study, using comprehensive historical exposure radon measurements, indicates an increased risk of lung cancer from indoor radon exposure. The data also suggest a supra-additive interaction between radon exposure and smoking, which has already been suggested in the past on the basis of data from other epidemiological studies.

Concerning risk management, the question was asked about what guidance could be offered to national authorities, and whether risks from radon should be considered solely within the present radiation protection framework policies or be handled as a part of a more integrated framework of all environmental risks. After defining the rationales and options of a radon control policy applied to different situations, and considering different regulatory tools and vehicles, economic and evaluation instruments, the Group came up with a three-step management scheme. The first step would require the identification of potential sources of elevated radon exposure. If a problem is identified, the second step would follow in the form of detailed surveys for the identification of populations at the highest risk and situations with potential future exposure. The risk management strategy would be the last step, dealing with the severity of the risk to health, the affordability of control measures and the preferred approach for control. It was agreed that the actual setting of indoor radon limits below the region of the acceptable risk should be governed by two approaches (in the given order of preference), namely, the 'integrated risk approach', i.e., comparing the risk to health from radon with other potential radiological and non-radiological health risks, and the 'conventional radiation protection approach', i.e., using the ALARA principle in the comparison between potential risks to health from radon and from other radiation sources.

The quite common apathy of the general public with regard to the indoor radon issue puts a special challenge on the effort to communicate risk information to the public. Hence, the Group took upon itself to discuss the whole problem of risk communication as a part of assisting policy makers to arrive at strategies best suited to their socioeconomic conditions.

The Group agreed on a set of recommendations regarding health risks, risk management and risk communication. Among these, the need for epidemiological case control studies aimed at the precise quantification of the effect of residential radon exposure and for mechanistic studies on lung cancer induction by radon and its progenies was stressed. It was recommended to promote comprehensive radon policies with built-in evaluation programmes and to implement risk reduction strategies. Finally, collection of baseline information on existing attitudes and perceptions about radon followed by the initiation of adequate risk communication programmes was recommended.

37.6.11 Future activities

Following the establishment of the WHO European Centre for Environment and Health with its two operational divisions in Bilthoven, The Netherlands and Rome, Italy, its coordination unit in Copenhagen, Denmark, and its project office in Nancy, France, the responsibility for the indoor air programme of WHO/EURO was transferred to the Bilthoven Division. Recognizing the health impact of indoor air quality, it was considered a major priority to set up a strong programme of work related to this subject. To adequately prepare for this task, an expert meeting on Priorities for Work on Health Aspects of Indoor Air Pollution was held in Bilthoven in July 1994.

At this meeting, it was concluded that indoor air pollution poses an important, yet controllable, risk to human health and comfort. However, much remained to be done to implement the available knowledge on this topic and to further our understanding of indoor exposures and associated risks. WHO could contribute to reducing these risks by undertaking activities in three areas: (1) encouraging country-specific indoor air quality policies; (2) disseminating reliable information on the issue; and (3) helping to identify and fill information gaps (i.e., research needs) that need to be addressed.

To fulfil this role, it was recommended that, in the field of indoor air quality policies, WHO should develop and disseminate a guidance document on strategic approaches to indoor air quality policymaking. This would be an initial step to encourage countries to have a comprehensive and meaningful programme developed to indoor air quality. In the area of dissemination of information, it was considered necessary to develop, based on the WHO Air Quality Guidelines for Europe, a specific publication on Indoor Air Quality

Guidelines, taking into account specific exposures, target groups and control strategies. Besides, it was recommended that WHO-ECEH should consider publishing and disseminating special indoor air quality publications targeted to scientists, regulatory authorities, and the general public. These publications could cover several subjects including acceptable indoor air quality measurement methods, building investigation protocols, and effective and energy-efficient indoor air quality mitigation approaches. Similarly, these publications could address pollutant-specific issues such as environmental tobacco smoke and pesticides. In the area of research, it was recommended that WHO promotes research in the field of IAQ, with special emphasis on exposure assessment research, including personal exposures and activity patterns, on mechanistic and applied studies on the role of IAP in the development and exacerbation of allergic and hypersensitivity reactions, on studies devoted to transmission of infectious diseases, on source characterization research and on research devoted to energy-efficient and cost-effective indoor air quality control techniques.

37.6.12 WHO air quality guidelines

In 1984, WHO/EURO embarked on a project to study and establish air quality guidelines for Europe for close to 30 organic and inorganic air pollutants. The basic principles that guided the project were: the guidelines would describe the latest state of scientific knowledge; the information provided would be condensed, describing only the essential factors leading to the final conclusions; the description of scientific findings would be understandable to a broad and rather heterogeneous group of readers; the rationale for the guideline recommendations would also contain a description of uncertainties in the evaluation process due to missing, inadequate or equivocal data; a basic common structure for the description of pollutants and the rationale for guidelines would be enforced; and the draft guidelines would undergo several intensive reviews.

The guidelines consider various toxic (carcinogenic and non-carcinogenic) substances, and for a few substances also their ecological effects. The guidelines do not differentiate between indoor and outdoor exposure (with the guidelines do not differentiate between indoor and outdoor exposure (with the exception of exposure to mercury) because, although the sites influence the type and concentration of chemicals, they do not directly affect the basic exposure–effect relationship. The guidelines apply to the exposure of the general population, and do not relate to occupational exposures, event though the latter have been considered in the assessment process. For those compounds that were not reported to induce carcinogenic effects and on which data

regarding such effects were lacking to insufficient, a threshold assumption was made and guideline values were proposed. For carcinogenic substances, the guidelines provide an estimate of lifetime cancer risk arising from exposure to those substances.

The project was completed with the publication of the guidelines at the end of 1987 (WHO/EURO, 1987).

Following publication, the Air Quality Guidelines for Europe (AQG) have provided a uniform basis for the development of strategies for the control of air pollution, and have contributed to the maintenance and improvement of public health in several countries. Since then, however, new scientific data in the fields of air pollution toxicology and epidemiology have accumulated and new developments in risk assessment methodologies have taken place, requiring updating and/or revision of the existing guidelines. This fact was recognized during the preparation of the initial workplan of the European Centre for Environment and Health, and it was recommended that the Centre undertake any necessary amendments and extensions to the Air Quality Guidelines.

To initiate the updating procedure, which will be conducted in collaboration with the Commission of the European Communities (CEC) and the International Programme on Chemical Safety (IPCS), a planning meeting was held at the Bilthoven Division of the European Centre for Environment and Health of the World Health Organization on 11–13 January 1993. At that meeting, which set the frame for the updating and revision process, it was recommended to set up a working group to deal with the methodologies to be applied in the assessment of specific pollutants/groups of pollutants as well as with the format of presentation. In addition, it was decided that the project be implemented through working group meetings which require the preparation of working documents on specific air pollutants or mixtures and a final consultation to discuss the updated document. Working groups were set up to assess the health impact of specific groups of air pollutants. The first working group on methodology and format met in September 1993 and gave advice on how the following more specialized groups should tackle assignments and on the format of the second edition of the AQGs. In particular advice on methods of risk assessment was provided. Three working groups dealing with classical air pollutants, inorganic air pollutants and ecological effects were scheduled for September/October 1994. The working groups on particular indoor air pollutants and on PCBs, PCDDs and PCDFs were scheduled for February/March 1995, while the working group on organic air pollutants was planned for autumn 1995. The publication of the second edition of the AQC is expected for 1996.

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Indoor Air Quality
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Chapter 38

Economic Implications of Indoor Air Quality and its Regulation and Control[1]

This chapter discusses the economic costs imposed by indoor air pollution. The costs of both health effects and damages to equipment and materials are considered. The limited available evidence on indoor air pollution effects is used to develop estimates for selected types of costs. These cost estimates are incomplete and are subject to great uncertainty.

However, the available evidence suggests that the costs imposed by indoor air pollution are very high. The costs of controlling indoor air pollution are not covered.

The first section discusses the nature of the economic effects of air pollution. The second section describes methodologies for valuing economic effects, and the third section presents available estimates of economic costs and implications for business managers.

Three major types of economic costs are addressed: (1) materials and equipment damages, (2) direct medical costs, and (3) lost productivity. These are described as follows.

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38.1 NATURE OF THE ECONOMIC EFFECTS OF INDOOR AIR POLLUTION

Indoor air pollution can soil indoor surfaces and can damage equipment and materials of various types[2]. The costs associated with equipment and materials damage include costs incurred to mitigate the effects of contamination (e.g.,

1 A part of the text of this chapter has been derived from the NATO/CCMS Pilot Study on Indoor Air Quality. The Implications of Indoor Air Quality for Modern Society, February 13–17, 1989, Erice, Italy, pp. 89–113.

2 Soiling refers to an effect that is reversible via cleaning, whereas material damage is an irreversible effect.

the costs of cleaning) and the costs of repair or premature replacement of equipment and materials.

People whose health is affected by poor indoor air quality may incur costs for medical services to alleviate the health effects. These medical costs include the costs of visit to the doctor or emergency room, hospital care, surgery, and so on.

Adverse health effects or general discomfort resulting from indoor air pollution may result in lost economic productivity. Lost productivity may occur on a continuum and include: (1) lost productive years due to major illness, (2) lost time due to increased number of risk days taken from one's job, and (3) lost productive efficiency while on the job.

All reduce the nation's capacity to product goods and service of value. Estimates of productivity should include the effects on both income-earning and non-income-earning activities of value. Activities such as child care in the home, home-making, and learning activities in a school or university setting should be included, although often are not because they are difficult to quantify.

These three categories of costs — materials and equipment damage, direct medical costs, and lost productivity — represent only a part of the economic losses due to indoor air pollution. Some economic costs not included in the above categories are:

- other welfare loss associated with pain and suffering due to health effects that are not fully alleviated by medical treatment;
- the value of unpaid time spent by persons taking care of those whose health is affected in the home; and losses due to reduced enjoyment of recreational and other non productive activities affected by indoor air pollution.
- losses due to reduced enjoyment of recreational and other non productive activities affected by indoor air pollution.

No attempt was made to estimate costs in these categories.

In addition, this report does not consider the effects of exposure to commercial or occupational sources of pollutants other than those occurring in white-collar work environments. The categories of economic costs considered in this report therefore represent only a portion of the true losses to society resulting from poor indoor air quality.

38.2 METHODOLOGIES FOR VALUING ECONOMIC EFFECTS

In the absence of market-based data on the effects of pollution, survey techniques are sometimes used to develop the equivalent of market valuations. These surveys seek respondents' views on their "willingness-to-pay" to reduce pollution; in this case, to reduce their exposure to indoor air pollution.

There are difficult methodological problems with such survey, but they have the virtue, like market-based valuations, of including all sources and types of welfare loss due to the pollution. EPA is not aware of any previous contingent valuation survey designed to measure the costs of indoor air pollution.

An alternative to using market-based data or contingent valuation measures is to develop estimates of the costs of individual end-effects of pollution, based on predictions of physical effects and use of various methods to value the effects. As described above, the drawback with the measurement of costs on an effect-by-effect basis is that it is limited to the effects that have been quantified. Quantifiable effects, plus welfare losses that are difficult to quantify, inevitably are excluded. Therefore, summing the costs estimated for individual effects of pollution tends to understate the true social costs of pollution.

However, this is the only approach available at this time to estimate the costs of indoor air pollution.

Two steps are involved in developing an effect/by-effect estimate of indoor air pollution costs. First, it is necessary to estimate "incidence" of each physical effect attributable to indoor air pollution; for example, numbers of cancer cases, levels of soiling, days of lost work, and such. Second, it is necessary to assign an economic value to those physical effects.

In the absence of market-based or contingent valuation estimates of the costs of indoor air pollution, this report relies on damage function procedures to develop cost estimates.

Because of data limitations, it was not possible to develop quantitative estimates of the economic costs associated with equipment and materials damages caused by indoor air pollution, even though these costs are discussed in the text.

For major illnesses, the pollutant/by-pollutant estimates of specific health effects along with estimates of the costs of those effects were used to estimate the economic costs of major illnesses resulting from indoor air pollution. Quantitative estimates of cases attributable to indoor air pollution for a limited number of pollutants and health effects are reported in the general literature. For those health effect quantified (i.e., cancer and coronary heart disease due to exposure to radon, volatile organic chemicals (VOCs) and environmental tobacco smoke (ETS), predicted numbers of cases annually were multiplied by estimates of costs per case to provide national annual cost estimates. Two types of costs are calculated: (1) direct medical care costs for each type of case (i.e., doctor visits, hospitalization, medication, surgery, and such), and (2) the present value of lost lifetime earnings per new case, including imputed earnings for home-making activities. In addition information from a study of emergency room visit for asthmatic children was used to

estimate the costs of exposure to ETS for asthmatic children.

Because of data limitations, it was not feasible to develop reliable estimates of the economic cost of the increased number of sick days or of lost productivity while on the job, due to indoor air pollution. However, available survey were used to provide information on these issues. While potential costs based on this data were calculated, these estimates are excluded from the final tabulations of economic costs because of uncertainties surrounding the data.

Previous chapters have focused primarily on the human health effects of indoor air pollution. High concentrations of contaminants in indoor air can also have adverse effects on materials and equipment. The effects of indoor air pollutants on indoor materials are influenced by a number of factors, including the type of pollutant, its concentration and exposure pattern, the type of material exposed, and other environmental factors. These effects are discussed in some detail in another report (EPA, 1987). Table 38.1 taken from that report summarized the major materials damages according to the materials potentially at risk, the air pollutant(s) associated with the effect, and other environmental factors that can also contribute to materials damage.

The pollutants most often associated with these materials damages include sulphur oxides (SO_x), nitrogen oxides (NO_x), ozone (O_3), particulates, and several acid gases (EPA, 1987). In addition, particulates in the form of water-soluble salts have been associated with damage to electronic equipment. Damages may include corrosion of electronic components and electrical current leakage, which may eventually result in equipment malfunction (EPA, 1987; Walker and Weschler, 1980).

The costs of materials and equipment damage by indoor air pollutants include the maintenance, repair, and/or replacement costs resulting from: (1) soiling or deterioration of a material's appearance, or (2) reduced service life for corroded or degraded appliances, furnishings, and equipment. For example, if house textiles such as draperies fade or change colour as a result of exposure to NO_x pollutants, then costs would consist of the cost of either repair or premature replacement of the draperies. In certain circumstances, such as in art museums or galleries, costs associated with installation of environmental controls such as air, filtering systems also may be incurred.

Few damage functions have been developed for the effects of indoor air pollution on materials and equipment. Studies performed for EPA to support national ambient air quality standards (Manuel, 1983) provide household economic damage functions for soiling due to SO_2 and particulates.

These studies rely on the results of several previous studies to estimate economic benefits due to reduced soiling, and might be applied to estimate the costs of indoor air particulate and SO_2 levels. However, the studies generally include categories of benefits (e.g., reduced washing of outside as well as inside

TABLE 38.1

Air pollution effects on materials

Materials	Type of damage	Principal pollutants	Other environmental factors
Metals	Corrosion, tarnishing	Sulphur oxides and other acid gases	Moisture, air, salt, microorganisms, particulate matter
Paint and organic coatings	Surface erosion discoloration, soiling	Sulphur oxides, hydrogen sulfide, particulate matter	Moisture, sunlight, ozone, microorganism
Textiles	Reduced tensile strength, soiling	Sulphur oxides, nitrogen oxides, particulate matter	Moisture, sunlight, ozone, physical wear
Textile dyes	Fading, color change	Nitrogen oxides, ozone	Sunlight
Paper	Embrittlement, soiling	Sulphur oxides, particulate matter	Moisture, physical wear
Magnetic storage media	Loss of signal	Particular matter	Moisture, heat, wear
Photographic materials	Microblemishes, "sulfiding"	Sulphur oxides, hydrogen sulfide	Moisture, sunlight, heat, other acid gases, particulate matter, ozone and other oxidants
Rubber	Cracking	Ozone	Sunlight, physical wear
Leather	Weakening, powdered surface	Sulphur oxides	Physical wear
Ceramics	Changes surface appearance	Acid gases, HF	Moisture, microorganisms

Source: U.S. Environmental Protection Agency (1987).

window surfaces and cleaning of screens and storm windows) and are based on 1972–1973 consumer expenditure data that are likely to be substantially out of date. For these reasons, the available soiling damage functions were not considered adequate to estimate soiling damages attributable to indoor air pollution

Similarly, no quantitative estimates are available of the effects of indoor air pollution on equipment. Information from Dr. Charles Weschler of Bell Communications Research, however, provide examples of such damages that indicate that the costs may be high (Weshler, 1988). Telephone switching and computing equipment is susceptible to corrosion caused by air particles and

gases. Weschler reported that the seven regional telephone companies have spent large sums to replace, clean, or repair switches and other electronic equipment malfunctioning as a result of indoor air contaminants. Failures are known to have occurred throughout the system and to range in cost from as little as $10,000 to as high as $380,000 per event. Bell Communications Research has developed guidelines for preventing such damages, including the use of high efficiency filtration, the constant use of fans, maintaining minimum air changes per hour, and keeping buildings pressurized to prevent infiltration of outdoor contaminants.

Various studies also have reported that home and office computing equipment and other electronic devices are subject to failure due to indoor air contamination (e.g., see Comizzoli et al., 1986). However, no estimates are available of the extent or costs of such damages.

In addition, it is known that microbial contamination can cause significant damage to buildings and equipment, and there is anecdotal evidence that damage can be so severe as to make a building unfit for human occupation. However, no quantitative data as to the extent of such damage is available.

To estimate the costs of medical expenditures associated with specific health effects, estimates of the present value of direct medical care costs per case were taken from Hartunian et al. (1981). These costs were developed for different types of illnesses from actual case experiences, and costs, occurring in future years were discounted to develop present values. Present value costs calculated at a 6% discount rate were used in this report.

Table 38.2 presents these costs per case taken from the Hartunian study. The average costs per case for all cancers and for coronary heart disease (CHD) were developed in the Hartunian study by weighting costs per case fro different types of cancer or CHD by the relative prevalence of each type in 1985. The Hartunian estimates were inflated to 1986 dollars for use in this study.

TABLE 38.2

Direct medical care costs for major illness

Health Effect	Present value of medical cost per new case ($ 1986)
Lung cancer	21.285
All cancers	24.938
Coronary heart disease	9.684

Source: Hartunian et al. (1981) — Hartunian values in 1975 $ updated to 1986 $ using an inflation factor for medical costs of 2.57 (1986 index of 433.5 divided by 1975 index of 168.6,; index of medical care prices from U.S. Bureau of Labor.
Statistics: CPI Detailed Report.

38.3 ESTIMATES OF ECONOMIC COSTS AND IMPLICATIONS FOR BUSINESS MANAGERS

Table 38.3 presents estimates of the annual national costs of medical care resulting from the major indoor air pollution health effects identified in the literature. As shown, estimated costs are over $1 billion annually. The range of cost estimates reflects estimates of the number of cases annually reported from different sources. Cancer cases due to exposure to VOCs, radon, and ETS account for the largest portion of estimated costs.

These estimates do not include the costs of many potential major illnesses and indoor air pollutants due to the limited quantisation of health impacts for these pollutants. For example, quantified national health impacts are not available for indoor exposures to pesticides, asbestos, formaldehyde, or many types of VCs, nor are estimates available for numbers of cases of emphysema or other illnesses due to ETS exposure. Except for radon, risk estimates reported in Table 38.3 have not been critically evaluated by EPA.

TABLE 38.3

Annual direct medical costs of major illness ($ 1986)

Total cost source	New cases/year	Cost/case	($ millions)
Cancer			
Radon	20,000 (lung) a	$21,285	$426
ETS	12–5000 (lung)	b	$21,285
	11,000	c	$24,938
Six VOCs	1000–5000	d	$24,938
Total cancer costs			$725–936
Non cancer			
ETS	32,000 (heart disease)	e	$9684
Total medical costs			$1015–1226

[a] EPA (1987) estimates 5000 to 20,000 deaths per year. However, 20,000 is used here based on revised estimates soon to be published by EPA.
[b] The range is based on several studies: Repace and Lowrey (1985, 1986, 1987); Arundel et al. (1987); Robins ((1986); and Wells (1988).
[c] Wells (1988).
[d] Wallace (1985).
[e] Wells (1986, 1988).

TABLE 38.4

Costs of additional emergency room visits for asthmatic children in households with smokers

Increased number of emergency room visits per year, asthmatic children in smoking vs. nonsmoking bouseholds	1.26[a]
Estimated cost of additional visits per year	$ 92[a]
Estimated percent of children in smoking households	62%[b]
Estimated percent of population that are asthmatic:	
– Total population	4.0%[c]
– Ratio of prevalence in children to prevalence in total population	1.25[d]
– Estimated percent of children that are asthmatic	5.0%
Number of children in the United States under 18 (1985)	63 M[e]
Estimated national annual cost of additional emergency room visits for asthmatic children in smoking households	$ 226M[f]

[a] Evans et al. (1987).
[b] Study by Bonham and Wilson (1981). Cited in Repace and Lowrey (1985).
[c] NCHS (1987).
[d] National Center for Health Statistics, private communication (1988).
[e] U.S. Census Bureau, Current Population Reports.
[f] $1.26 \times 92 \times 0.62 \times 0.05 \times 63$

Therefore, EPA does not necessarily agree with study designs used or with the conclusions reached by the authors of these assessment.

A recent study of 237 children from low-income families in New York City found that asthmatic children from smoking households visited emergency rooms more often on average than did asthmatic children from nonsmoking households (Evans et al., 1987).

Children from smoking households averaged 3.09 emergency room; visits per year compared with 1.83 times per year for children from nonsmoking households. Table 38.4 presents a calculation of aggregate national costs due to increased emergency room visits for asthmatic children due to ETS in the home, based on this study. The costs are estimated to total $226 million per year.

Few data exist on the number of employee sick days or on the productivity lost because of poor indoor air quality. However, some indirect evidence is available.

One survey of office workers conducted by Honeywell (Honeywell, 1985) found that 19% of respondents (115 of 600) often or sometimes had difficult doing their work because of office air quality. Of the 155, 64 (11% of all respondents) reported that a "tired/sleepy feeling" was a "very serious" or "somewhat serious" problem because of office air quality. Similarly, 52 (9% of all respondents) cited a "congested nose" 47 (8%) cited "eye irritation", and 46 (8%) cited "difficulty breathing" as being "very serious" or "somewhat serious" problems.

However, while the Honeywell survey was scientifically administered, it does not provide sufficient information for quantifying economic costs.

In addition to the Honeywell survey, a recent survey of 94 state government office buildings was conducted by a coalition of employee unions in the New England states of Maine and New Hampshire during the summer of 1987.

The survey sought information on the extent and effects of poor indoor air quality, including the losses in productivity, the increased number of sick days, and the frequency of doctors visits, attributed by respondents to poor indoor air quality.

The New England survey results report large numbers of complaints about health symptoms that respondents attribute to poor air quality: 30% of all respondents reported having headaches, 44% reported fatigue or drowsiness, 37% reported eye strain, and 69% reported some loss in productivity on a daily or weekly basis due to poor indoor air quality.

The New England survey provides the kind of information useful for estimating three elements of economic cost: direct medical cost of increased sick days doctors visits, the economic cost of lost productivity from increased sick days, and the economic cost of lower productivity while on the job. However, the New England survey was not scientifically administered and may have significant biases.

While the Honeywell survey and the New England survey are not directly comparable, the prevalence rates in the New England survey appear to be three to four times higher than those reported in the Honeywell survey. However, other data suggest that prevalence rates of the New England survey may not be far from the norm. For example, in a major British study prevalence rates of work-related symptoms were: lethargy (57%), headache (43%), stuffy nose (47%), and itchy eyes (27%) (Wilson and Hedge, 1987). In addition, 24% of the respondents thought that their work environment decreased their productivity by 20% or more, 59% thought it had little or no effect, and 16% thought it increased their productivity. Likewise, in a major study of Danish Town Halls and other buildings, approximate prevalence rates of work related symptoms were fatigue (28%), headaches (20%), eye irritation (13%), and nasal irritation (18%) (Skov and Valbjorn, 1987).

In order to obtain at least a qualitative estimate of the economic costs from medical visits, sick days lost, and productivity lost, some adjustments were made to the New England survey data, and conservative assumptions concerning those not responding were incorporated into the analysis[1] The adjustment

1 Excluded from the data were responses indicating more than six doctor visits per year, more than 12 days of sick leave, or more than 30% productivity loss due to indoor air pollution. In addition, it was assumed that no effect of indoor air pollution were experienced by those not receiving the survey, and those who received the survey but din not respond.

TABLE 38.5

Productivity costs of major illness (cost per case)

Health effect	Present value of lost earnings per case ($ 1986)
Lung cancer	99,532
All cancers	92,645
Coronary heart disease	44,896

Source: Hartunian et al. (1981) — Hartunian values in 1975 dollars up-dated to 1986 dollars using the ratio of the 1986 to the 1975 gross national product price deflators (114.1/59.3 = 1.92; U.S. Department of Commerce, Statistical Abstract of the United States, 1988, p. 252), and inflated by 37% to reflect the added value of fringe benefits (Nathan, 1987).

survey results show that an average of 0.24 doctor visits per worker per year were attributable to poor indoor air quality. If this figure were applied to the nation's 64 million white-collar force (BLS, 1988), and an average cost of $30 is assumed for a medical visit to a doctor or a medical practitioner, national medical care costs for doctors visits (other than for major medical illness) of white collar workers due to indoor air pollution would be on the order of half a billion dollars per year.

Table 38.5 shows estimates of the present value of lost lifetime work output for three types of major health effects. These estimates are taken from Hartunian (1981) and include an imputed value for home-making activities. The Hartunian values have been updated to 1986 dollars and inflated by 37% to reflect the value of fringe benefits for all private industry employers (Nathan, 1987). These costs per case are the present value of present and future lost work time due to a new case of cancer or CHD, with future years' values discounted at 6%.

Table 38.6 shows the calculation of the national annual cost of productivity losses associated with major illnesses caused by indoor air pollution.

This calculation uses the same approach as the calculation of direct medical costs in Table 38.3. The estimated cost ranges from $4.7 billion to $5.4 billion for new cases caused by indoor air pollution annually.

Productivity losses on the job due to indoor air pollution may take several forms. For example, workers may be less effective with their work because they feel fatigued, or suffer from headaches, eye irritation, or other effects. These are typical symptoms of sick building syndrome. Workers may therefore accomplish less per hour worked or may spend more time away from their work location, for example, taking breaks or walks outdoors to avoid poor air quality where they work. These effects will result in lower output per hour at work. In addition, workers may be out sick more often. The value of both

TABLE 38.6

Annual productivity costs of major illnesses ($ 1986)

Source	New cases/year	Cost/case	Total cost ($ million)
Cancer			
Radon	20,000 (lung)	$99,532	$1,991
ETS	12–5000 (lung)	$99,532	$1,2–518
	11,000 (other)	$92,645	$1,019
VOCs	1,000–5,000 (other)	$92,645	$93–463
Total cancer			$3,011–3,991
Non cancer			
ETS	32,000 (heart disease)	$44,896	$1,437
Total cancer and non-cancer			$4,448–5,428

Source: Tables 38.3 and 38.5.

reduced output while at work and increased sick leave time lost from work can be measured by multiplying average hours of productive work lost by an average hourly compensation rate. The average total compensation rate per hour worked for while collar workers is $15,56. This rate includes benefits such as paid leave, premium pay for overtime, insurance and retirement benefits, and legally required benefits such as Social Security (Nathan, 1987).

Data from the New England survey, which was adjusted as previously indicated, would attribute an average productivity loss of 3% to poor indoor air quality. This is equivalent to approximately 14 min per day in lost work time. Respondents would also attribute an average of 0.6 added sick days per year to poor indoor air quality. If these results were applied to the nation's white-collar labour force, the economic cost to the nation would in order of $60 billion annually. While this cannot be regarded as a reliable estimate, it suggests quite strongly that productivity losses may be in the order of tens of billions of dollars per year.

Many efforts to improve indoor air quality in office environments can be administered at little or no cost. These include, for example, proper storage of toxic cleaning and maintenance products, and basic maintenance of the ventilation system. Nevertheless, some buildings may require actions which increase energy or other operation or maintenance costs, and for this reason they are often resisted. But, we have seen that poor indoor air quality increases

TABLE 38.7

Productivity gains necessary to offset operating cost increases

Cost increases		Offsetting productivity gain
Energy or	Total environmental	
5%	1.67%	0.05%
15%	5.00%	0.15%
25%	8.33%	0.25%
50%	16.67%	0.50%

Base cost assumptions: Energy costs = $2 per sq. ft. per year; labour costs = $200 per sq. ft. per year; total environment costs = $6 per sq. ft. per year.

labour costs through losses in productivity, increased employee sick days, and medical costs. This suggests that measures to cut building construction and operating costs may increase the total cost to business because of a higher wage bill, and that money spent to improve indoor air quality may be profitable from a purely business profit and loss standpoint.

It is useful to ask whether costs incurred to improve indoor air quality would pay for themselves in increased productivity. Labour costs in a typical office setting will depend on salary levels and occupant densities.

Typical labour costs are on the order of $100 to $300 per ft^2/year[1] (Dorgan, 1988; Woods et al., 1987). In comparison, energy costs are on the order of only $1 or 2 per ft^2/year, while total environmental control costs (energy, operation and maintenance) are on the order of $2–10 per ft^2/year (Dorgan, 1988; Woods et al., 1987; Eto and Meyer, 1988). It is clear, therefore, that from a profit and loss standpoint, productivity, not energy consumption, is the dominant consideration for office environments.

While cost increases to improve indoor air quality could be substantial in absolute dollar terms, Table 38.7 demonstrates that such costs would be more than offset by very modest productivity increases. For example, a 5% increase in energy costs (or a 1.67% increase in total environmental control costs), would require only a 0.025 to 0.05% increase in productivity to make this a worthwhile expense. A 50% increase in energy costs (or a 16.67% increase in total environmental control cost) would require only a 0.25 to 0.50% increase in productivity. This result suggests that expenditures for improved indoor air

1 For example, the American Society of Heating, Refrigerating, and Air Conditioning Engineers standard 62-1981 assumes an occupant density amounting to about 150 ft^2 per person in office environments. With a labour cost rate of $15.56/h and a yearly work rate of 2000 h/year, labour cost for office environments would be $207/$ft^2$/year.

TABLE 38.8

Productivity gains necessary to offset capital expenditures

Capital cost ($/sq. ft)	Annualized cost ($/sq. ft)	Offsetting no change in operating cost	Productivity gains operating cost increase by 15%
1	0.13	0.07%	0.22%
5	0.66	0.33%	0.48%
10	1.31	0.66%	0.81%
15	1.97	0.99%	1.14%
25	3.29	1.64%	1.80%
50	6.57	3.29%	3.44%

[a] Base cost assumptions: Energy costs = $2 per sq. ft. per year; annualized costs are capital costs amortized at 10% over a 15-year life.

quality could generate exceedingly high returns to the business community, where labour is an important cost category.

Increasing ventilation capacity from 5 to 20 cfm per occupant is estimated to increase total construction costs less than $0.50 per ft^2 under typical circumstances (Eto and Meyer, 1988). However, for existing buildings, significant retrofit costs may be required to increase ventilation, improve air distribution, or to otherwise improve indoor air quality. It is useful to ask, therefore, whether productivity gains could be expected to offset capital expenditures either for new construction or for retrofit applications.

Productivity gains needed to offset alternative capital expenditure requirements, with and without a modest (15%) increase in energy operating costs, are displayed in Table 38.8. For example, a capital expenditure of $15 ft^2 would require about 1% increase in productivity to offset the expenditure. If, in addition, energy operating costs were increased 15%, productivity would have to increase by 1.14%. Given the dominance of labour costs, even a modest increase in productivity could justify substantial capital expenditures to improve indoor air quality.

Table 38.9 summarizes the estimates developed in this report of the economic costs attributable to indoor air pollution. As shown in the table, the estimates are available only for a limited set of potential indoor air pollution effects. The reported costs were developed by extrapolating from limited evidence. Substantial addition basic research and analysis will be required to improve on these estimates of economic costs.

Despite the limitations in the available evidence, the calculations presented in this chapter suggest that the costs of indoor air pollution are very

TABLE 38.9

Summary of annual economic costs of indoor air pollution ($ millions)

Pollutant	Direct medical cancer	Expenditures non-cancer	Lost productivity	Material and equipment damage	Total calculated costs[a]
Radon and radon daughters	$426	NC	$1991	$2417	
ETS	$274385	$516	$2457--2974	NC	$3136–3511
Biological contaminants	NC[b]	NC	NC	NC	
VOCs					
Six VOCs	$25–125	NC	$93–463	$118–588	
Other VOCs	NC	NC	NC		NC
Asbestos	NC	NC	NC		NC
Combustion gases	NC	NC	NC	NC	NC
Particulate matter	NC	NC	NC	NC	NC
Unspecified (sick building syndrome)	NC	see text	see text		see text

[a] Includes only costs associated with types of cancer and other health effects for which estimates of numbers of annual health impairments were available. The estimated costs shown here are therefore understated.
[b] NC= Costs not calculated.
Source: Tables 38.3, 38.4 and 38.6.

high. Many costs of indoor air pollution have not been calculated. Nevertheless, because of the large numbers of people and buildings potentially affected, as well as the wide range of effects for which there is an economic cost component, it is reasonable to conclude the aggregate costs of indoor air pollution amount to tens of billions of dollars per year.

Of the costs shown in Table 38.9, ETS accounts for a large portion of the costs attributed to specific sources (e.g., cancer, heart disease, and effects on asthmatic children). Not displayed in Table 38.9 because of data limitations are specific estimates of the economic costs associated with losses in productivity and increases in employee sick days from unspecified sources typified by the sick building syndrome. As indicated in the text, these costs may be on the order of tens of billions of dollars per year.

This chapter did not discuss the cost of methods that can be used to improve indoor air quality. In some cases, the costs of changes needed to correct poor indoor air quality will be high. This chapter suggests, however, that the costs imposed by continuing to live with poor indoor air conditions are also very

high, and for business establishments where labour is an important cost factor, remedial actions are likely to be cost effective, even if they require expensive retrofit. Over time, development and dissemination of information on the costs of poor indoor air may encourage building owners to upgrade the quality of indoor air to prevent decreases in the value of their buildings as tenants and clients become more knowledgeable about the effects of indoor air contamination.

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Appendix I

Assessment of Levels of Knowledge About IAQ

At the second working group in 1981, the experts attempted for the first time to consolidate levels of knowledge about IAQ in the form of three tables. The tables were not supposed to be used without consulting the accompanying text in that report. However, four additional working groups have since reviewed and updated the tables, making them a self-contained entity and annex of their respective reports.

TABLE 37.1

Current levels of knowledge about indoor population exposure factors

Pollutant	People with low exposure	People with high exposure	Sources	Distri-bution	Instrum-entation	Indoor & personal monitoring (incl. biological)
ETS	most	some	+	+)	+	+)
RSP	most	some	+)	+)	+)	+)
NO_2	most	few	+	+	+	+
CO	most	few	+	+	+	+
Radon and daughters	most	few*)	+	+	+	+
Formaldehyde	most	few	+	+	+	+
SO_2	most	few	+	+	+	+
CO_2	most	few	+	+	+	+
O_3	most	few	+	+	+	+
Asbestos	most	few	+	+	+)	+
Mineral fibres	most	few	+)	+)	+	+)
VOC	most	some	+	+	+	+
Other organics	most	few	0	0	+	+)
Aero-allergens	some	most	+	+)	0	0
Infectious agents	most	few	+	+)	+)	0

* = Varies from area to area; + = adequate; +) = less than adequate; 0 = inadequate.

During its meeting at Charleston, SC, US, in November 1989, the Seventh WHO working groups reviewed assessments of the levels of knowledge and presented three tables: about indoor population exposure factors in Table 37.1, about exposure–response relationships in Table 37.2, and consensus of public health concern in Table 37.3.

TABLE 37.2

Current levels of knowledge about exposure–response relationships

Pollutant	People at low exposure	People at high exposure	People exposed	Adverse effects at levels of concern	Exposure response relationship	Means of control
NO_2	most	few	+	air way effects systemic	+ 0	technical regulatory educational
CO	most	few	+	systemic	+	technical regulatory educational
SO_2	most	few	+	air way effects	+	technical regulatory educational
CO_2	most	few	+	systemic	+	technical
O_3	most	few	+	mucosal irrit. air way effects odour	+ + +	technical (indoors) regulatory
Radon and daughters	most	few*)	+	cancer	+	technical regulatory educational
ETS	most	some	+	odour irritation airway effects carcinogenic systemic	+ + + +	technical regulatory educational social
RSP	most	some	+	mucosal irrit. airway effects systemic	+ + +	technical regulatory educational
Asbestos	most	few	0	carcinogenic respiratory disorders	+ (+)	technical regulatory educational
Mineral fibres	most	few	0	irritation airway effects carcinogenic	+ + (+)	technical regulatory educational

(continued)

TABLE 37.2 (*continuation*)

Pollutant	People at low exposure	People at high exposure	People exposed	Adverse effects at levels of concern	Exposure response relationship	Means of control
VOC	most	some	+	odour sensory irrit. mucosal irrit. systemic airway effects cancer	(+) (+) (+) (+)[a] (+) +[a]	technical regulatory educational social
Formalde-hyde	most	few	+	odour mucosal irrit. airway effects cancer systemic	+ + + +[a] +[a]	technical regulatory educational
Other organics	most	few	0	odours mucosal irrit. airway effects cancer systemic	+ + + +[a] +[a]	technical regulatory
Aero-allergens	some	most	(+)	airway effects mucosal effects	(+) (+)	technical educational medical[b]
Infectious agents	most	few	0	respiratory other organs systemic	(+) 0 0	technical medical[b] educational regulatory

* Varies with region.
[a] For some it is inadequate.
[b] Medical measures are preventive.+ = Adequate; (+) = less than adequate; 0 = inadequate.

TABLE 37.3

Consensus of concern about selected indoor air pollutants at 1990 levels of knowledge.

Selected pollutant	Typical range of concentr. (10–90%)[b] mg/m^3	Concentration of limited or no concern[b] mg/m^3	Concentr. of concern[b] mg/m^3	Remarks (average exp. period) concentration mg/m^3
RSP (incl. tobacco)	0.01–0.15	<0.1	>0.15	0.15 (24 h) (Japanese Standard)
$NO_2$2	0.02–0.4	<0.15	>0.40	AQG[a] 0.4 (1 h)
CO	1–11	<2% COHb <10 <30	>3% COHb	AQG 10 (8 h)
Radon and daughters	3–75 Bp/m^3 EER	carcinogen	carcinogen	Swedish standard: new house 70 Bp/m^3 AQG 100 Bq/m^3 EER (1 y)
SO_2	0.01–0.08	<0.25	>0.35	AQG 0.35
CO_2	300–2000	<2000	>7000	1800
O_3	0.01–0.1	<0.1	>0.12	AQG 0.15–0.2 (1 h)
Asbestos	100–10000F*/m^3	carcinogen	carcinogen	(24 h)
Mineral fibres	100–10000F*/m^3	-[c]	-[c]	Skin irritat. (24 h)
Organics				
Formaldehyde	0.02–0.06	<0.06	>0.12	AQG <0.1 (30 min)
Benzene	0.002–0.02	carcinogen	carcinogen	
Dichloromethane	0.005–0.01	-[c]	-[c]	AQG 3.0 (24 h)
Trichloroethylene	0.001–0.02	-[c]	-[c]	AQG 1.0 (24 h)
Tetrachloroethylene	0.002–0.02	-[c]	-[c]	AQG 5.0 (24 h)
p-Dichlorobenzene	0.001–0.02	-[c]	-[c]	TLV[d] 450 (8 h)
Toluene	0.03–0.15	-[c]	-[c]	AQG 7.5 (24 h)
m,p-Xylene	0.01–0.04	-[c]	-[c]	TLV 435 (8 h)
n-Nonane	0.002–0.02	-[c]	-[c]	TLV 1050 (8 h)
n-Decane	0.03–0.05	-[c]	-[c]	
Limonene	0.002–0.07	-[c]	-[c]	TLV 560 (8 h)

* Fibre count with optical microscope.

[a] All gases were considered on their own without other contaminants.

[b] Short-term exposure averages.

[c] No meaningful number can be given because of insufficient knowledge.

[d]TLV (threshold limit values) established by the American Conference of Governmental Industrial Hygienists (1987/1988). These values are for occupational exposures and might be considered the extreme upper limit for non-industrial populations for very short term exposures.

[e]AQG values are from Air Quality Guidelines for Europe.

Appendix II

Conclusions and Recommendations of the WHO IAQ Working Groups

Each of the working groups ended their deliberations with a set of specific conclusions and recommendations, directed to WHO, national authorities or the scientific community concerned. Some of the recommendations have been followed up and eventually implemented, while others are still awaiting action.

This Appendix presents the conclusions and recommendations of the five most recent working groups (fifth–ninth) for easy reference and review.

FIFTH WHO WORKING GROUP ON IAQ: ORGANIC POLLUTANTS

Conclusions

1. The distribution of indoor concentrations of CO, NO_2 and VOC is much better known than those of very volatile, semi-volatile and particle-bound organic compounds, and reactive substances from all these groups. There is a need to collect data on the distribution of the latter groups.
2. Organic compounds may cause odours, mucosal and sensory irritation and airway effects at levels encountered indoors. Any acute toxic effects to various organs usually occur only at concentrations higher than normally encountered indoors.
3. Evaluating the effect on health of single compounds may not always be adequate, because indoor pollutants usually occur in mixtures and many sources emit mixtures of pollutants. However, with the present state of knowledge, only a compound-by-compound approach can be applied to risk estimates in most instances.
4. Exposure to organic compounds, particularly semi-volatile and particle-bound compounds found in the indoor environment, occurs by various routes (inhalation, ingestion and skin absorption). The assessment of total dose and the relative contribution of indoor air exposure requires further study.

5. The detection limits of analytical instruments used at present do not coincide with the detection limits of human sensory systems. Therefore, many strong odorants may not yet have been chemically identified.
6. An indoor concentration limit value based on the detection or recognition of an odour or sensory irritant by 50% of people (ED_{50}) will not protect the most sensitive part of the population. Also, such a limit does not protect against systemic or genotoxic effects of substances that are not odorants or irritants.
7. Methods are available to assess the total effect in terms of chronic disease of indoor organic air pollutants. These incorporate all available information on exposure distribution, health effects measured in other settings, and the background incidence of the disease in question.
8. The available information on the potency as odorants or sensory irritants of organic air pollutants commonly encountered indoors is inadequate.

Recommendations

1. To facilitate the use of data on exposure to organic compounds, the distribution of the respective concentrations should be reported as the 10th, 50th and 90th (and, if possible, the 95th and 98th) percentiles.
2. Because of the great variety of organic compounds in the indoor air and the difficulties in implementing indoor air quality standards, various approaches to source control should be studied and developed. Where appropriate, forms of social control (such as with ETS) should be developed through education, mass media campaigns and, if necessary, legal action.
3. Methods for estimating exposure distributions over time (peak exposures versus long-term averages) and over space (exposures in many different spaces) from relatively few measurements need to be developed and validated.
4. Total personal exposure estimates for the organic compounds known to cause adverse health effects within populations should be assessed. This is especially true for exposures due to emissions from complex sources and emissions from multiple sources.
5. Methods for assessing biological burden and activity, including exhaled breath analysis and assays of secretions, should be further developed for organic compounds and their metabolites.
6. Planning studies concerned with health effects, exposure characterization and mitigation of organic compounds, attention should be focused on those compounds whose estimated contribution to the disease in question is more than 2% of the total background incidence.

7. Unwanted odorous compounds should not be present in concentrations exceeding the ED_{50} detection threshold. Similarly, sensory irritants should not be present in excess of their ED_{10} detection threshold.
8. Increased emphasis should be given to research in human on the sensory effects of organic compounds in low concentrations. This is especially true for detection and recognition data, which should be collected in a way that allows the full dose-response curve to be determined, including the ED_{10} and the ED_{50} values.
9. The source strength, as well as the rate of change of emissions of organic compounds from building materials and consumer products, should be determined and evaluated in relation to actual human exposure and the associated health effects.
10. As the available information on indoor concentrations of pesticides and herbicides does not permit an adequate evaluation of the associated acute and chronic health effects and in view of the toxicity of these compounds, further data should be urgently generated.

SIXTH WHO WORKING GROUP ON IAQ: BIOLOGICAL CONTAMINANTS

Conclusions

1. A substantial portion of disease and absenteeism from work or school is associated with infections and allergic episodes caused by exposure to indoor air. Since this morbidity is often due to biological contaminants generated in buildings or to the crowding of occupants, it can be reduced significantly.
2. The increase in costs associated with improving the inadequate maintenance of ventilation systems results in greater comparative benefits in terms of better health for the occupants and reduced absenteeism.
3. Biological aerosols in buildings, including homes, are caused predominantly by persistent moisture and inadequate ventilation in spaces and building elements; proper design and construction are essential to prevent these conditions.
4. The levels of biological contaminants in indoor air vary enormously in time and space, so data bases on the distribution of the levels of contaminants in conjunction with occupants' response must be large enough to provide useful information for risk management.
5. Methods for collecting environmental samples of biological contaminants have generally not been standardized. Sampling methods for pollen, specific bacteria and viruses are close to standardization, but sampling

methods for fungi, mycotoxins and other biological materials are not.
6. Laboratory procedures for the analysis of some fungi, mycotoxins, viruses, bacteria and other biologically derived materials of potential interest in indoor environments have not yet been standardized.
7. The use of biocides in the cleaning and maintenance of heating, ventilating and cooling systems or surfaces in buildings presents risks, both directly and through the promotion of resistant microbes.

Recommendations

1. Buildings and their heating, ventilating and cooling systems should not produce biological contaminants that are then introduced into the ventilation air. If the use of biocides is unavoidable, they should be prevented from entering space that can be occupied.
2. Standards and building codes should ensure the effective maintenance of ventilation systems by specifying adequate access and regular inspection and maintenance schedules.
3. In a building in which the occupants cannot effectively control the quality of ventilation air themselves, an individual who is responsible for this task should be made known to them.
4. To reduce allergic disease in the community, total exposure to allergens should be minimized by controlling allergens and their sources in buildings.
5. For the risk assessment of allergic diseases, exposure-response curves should be established by measuring antigens in the air and specific IgE antibodies in the population.
6. Statistically designed population studies should be carried out using commonly accepted methods to obtain the concentration distributions of biological contaminants in specific geographic locations.
7. Sampling and analysis methods for aero-allergens and biological irritants should be standardized, and the effects of time, temperature and moisture should be determined.
8. The production of biological aerosols in buildings should be prevented by introducing appropriate prescriptions for design and construction practices into building codes.
9. The maintenance personnel of public and office buildings should be given adequate training in the routine inspection and maintenance of the buildings' systems.
10. Biological irritants and infectious agents cause nonspecific aggravation of respiratory and skin diseases, and they should be minimized by controlling their levels and sources in buildings.

SEVENTH WHO WORKING GROUP ON IAQ: COMBUSTION PRODUCTS

Conclusions

1. Indoor Sources

Indoor, unvented kerosene space heaters are the major source of SO_2, and an important source of NO_2, CO, RSP, and acids in the vapour and particulate phase. Unvented gas space heaters are the major source of sustained (>1 hour) high levels of NO_2, and an important source of CO. Gas cooking ranges and unvented domestic hot water heaters produce high peak concentrations of NO_2. Wood burning stoves and fireplaces contribute RSP and a wide range of organic compounds. ETS is the major source of RSP and an important source of a wide range of both vapour and particulate phase air contaminants, including VOC.

2. Health effects

Combustion products from indoor sources contribute significantly to the total human exposure to NO_2, CO and CO_2 and to increased acute and chronic disease. CO concentrations can reach values as high as in busy streets, and venting failures may leak to lethal concentrations. NO_2 concentrations may exceed health based air quality guidelines for short periods of time. ETS ingredients have irritant and genotoxic properties and, with extensive exposure, there is evidence of an increased lung cancer risk in non-smokers. ETS and NO_2 are associated with a significant attributable risk for bronchial responsiveness and acute respiratory disease.

3. Engineering controls

In cold and temperate climates, where the fact of making the envelope of buildings more airtight has resulted in increased concentrations of air pollutant, the principal indoor combustion appliances are those related to space heaters. Generally they have flues that discharge all combustion products outdoors, but faulty design or installation, blockage or adverse weather conditions can lead to leakage indoors. Current building codes and standards governing design and installation of vented appliances are not adequate for controlling their leakage.

4. Vehicles

Occupants of vehicles can be exposed to elevated concentrations of combustion products if the vehicle has a faulty exhaust system or because of the exhaust gases from other vehicles entering through the cabin air intake.

Recommendations

1. Emission rates and concentrations of certain combustion products, such as particle-bound organic compounds and acids, should be determined, also through the development of appropriate instrumentation, to assist in the evaluation of distributions of population exposure.
2. Means for water vapour control indoors should be publicized and applied.
3. Existing and future new air quality guidelines should be used to guide decisions on source control in indoor vented and unvented combustion.
4. The general public should be informed about the dangers of fireplace spillage and down-drafting, especially at the end of the burn, and be provided with instructions on ways and means to avoid this hazard.
5. Buildings should be designed and operated so as to reduce the intake of vehicular combustion products from garages and streets, and from neighbouring buildings' exhaust vents.
6. The use of modes of transportation and traffic strategies which minimize emissions should be promoted to reduce exposures in vehicles.
7. Studies should be conducted to determine the carcinogenic potential of particle-bound formaldehyde.
8. Whenever the envelope of a building is made more airtight, careful consideration should be given to the active and controlled ventilation required.
9. Educational strategies should be used to communicate the risks associated with exposure to combustion products, as well as the options each person has to avoid such exposures.
10. The frequency and severity of leakage of combustion products from vented appliances should be documented with the objective of minimizing such occurrences through better equipment design, installation, maintenance and operation.
11. The use of unvented space heating and water heating appliances should be phased out as rapidly as possible.
12. Properly designed local exhaust with adequate provision of make-up air should be used to reduce peak exposures to NO_x associated with gas cooking.
13. Tobacco smoking should not be allowed in public buildings, public trans-

portations and related buildings and areas, or office buildings, and it should be minimized in the residential environment through education campaigns and other appropriate means.

EIGHTH WHO WORKING GROUP ON IAQ: INORGANIC FIBRES AND PARTICULATE MATTER

Conclusions

1. Airborne contamination with asbestos is widespread and as a consequence asbestos fibres can be found in most human lungs.
2. The carcinogenic potential of mineral fibres increases with fibre length, and with their increasing durability.
3. All commonly used forms of asbestos have produced excess incidence of asbestosis, lung cancer and mesothelioma.
4. Elevated rates of lung cancer have been reported in rock- or slag-wool production workers. Current airborne MMMF concentrations in indoor environments are considered to represent an insignificant risk.
5. There have been special situations where environmental exposure to erionite and crocidolite have caused increased rates of mesothelioma.
6. The possibility of lung fibrosis is of concern only to people who repeatedly disturb fibrous materials, thus creating high local concentrations of airborne fibres. These people may also be at increased risk for lung cancer and mesothelioma.
7. Exposed, loose or friable thermal and acoustic insulation materials are the major sources of indoor exposure to mineral fibres.
8. The main causes of mineral-fibre release are installation, removal and damage of mineral fibre-containing materials, and demolition of building.
9. The great majority of buildings have airborne asbestos concentrations which do not represent a significant excess risk.
10. Exposure to MMMF can cause skin and eye irritation.
11. Average indoor levels of MMMF range up to 300 F/m^3 in buildings containing these materials. Average indoor levels of respirable asbestos range from 100 to 1000 F/m^3 (fibre length >5 μm) in buildings containing asbestos products. When loose, friable asbestos and MMMF materials are abraded, much higher levels are generated.

Recommendations

1. Standard methods for measuring indoor inorganic fibres should be established so that they can be determined and reported in terms of fibre length

and diameter, with speciation of fibre type. Indoor samples should be collected over a one-week period.
2. Prior to their use in building products, mineral fibres should be systematically investigated with respect to the exposure and health effects likely to result.
3. All construction and operating documents, including those listing an inventory of inorganic fibre-containing materials in the building should be kept by the building manager, and referred to in the event of maintenance and repair.
4. The use of carcinogenic mineral fibres (such as asbestos and erionite) in construction should be avoided wherever reasonably possible.
5. Exposure to airborne mineral fibres should be kept as low as possible. Particular attention should be given to the management of loose, soft, friable and accessible mineral fibre-containing products.
6. Mineral fibres should be coated or covered with other materials to prevent fibre release and to minimize exposure of the public by inhalation and skin contact. In the event of contamination, mineral-fibre dust should be removed from the affected area before normal activity is resumed.
7. Precautions should be taken to reduce airborne-fibre exposure of people who disturb mineral-containing materials maintenance and repair.

NINTH WHO WORKING GROUP ON IAQ: A RISK-BASED APPROACH TO HEALTH FOR RADON INDOORS

On risks to health:

1. Radon, a human carcinogen, is present both in mines homes and poses an important public health problem.
2. The most powerful design for epidemiological studies for precise quantification of residential radon-related risks to health is the case control approach. However, few such studies with adequate statistical power are available at present.
3. Although residential exposure levels are generally lower than occupational ones, extrapolation is not made over a large range, and cumulative residential exposures may even reach occupational ones.
4. Despite differences in population, methodology and exposures to other contaminant, the miner studies have consistently shown similar excess lung cancer risk from cumulative exposure.
5. A linear model for extrapolation of risk from miners to residential populations is, at this time, a reasonable approach.

6. At present, the "time since exposure" model, as described in the BEIR IV report, modified to incorporate adjustments for uncertainties and differences in lung dosimetry, provides a reasonable basis for risk assessment and the setting of radiation protection guidelines and standards.
7. Although there are numerous uncertainties with the risk assessment models, the uncertainties are less than those associated with many other environmental and occupational carcinogens.
8. There is no conclusive evidence at present concerning radon-induced effects on health other than lung cancer.
9. Smokers are at a higher risk due to radon exposure than non-smokers because the combined risk of smoking and radon is more than additive.

On risk management:

10. Risk management tools for dealing with the control of risk from indoor radon are seldom available, and considerable effort is needed for creating a proper basis for dealing with this task.
11. Management of the radon risk proceeds both from radiation protection and from indoor health questions. This has resulted in the fact that different types of authorities are in charge of this questions in different countries.
12. Some countries do apply an "indoor radon action level" design target for new houses, which is lower than the action level applied for existing houses.

On risk communication:

13. An effective radon communication programme involves testing alternative radon messages with appropriate audiences, such as scientists and home owners, prior to public distribution, to ensure that radon messages are clear and accurate, and achieve their aims.
14. Effective radon communication programme involves key issues, such as identification of target audiences, enlistment of sources respected by the target audiences, and making use of the existing communications channels available to these sources.

Recommendations

On risks to health:

1. Future epidemiological case control studies aimed at the precise quantifi-

cation of the effect of residential radon exposure should be conducted in regions of high and varied radon concentrations and be controlled for age, sex, occupational exposure, site of exposure and tobacco smoking.
2. Pooled analysis of indoor radon studies should take place and necessary methodology should be improved.
3. Pooled analysis of miner data should proceed and address specifically the effect of dose rate and low doses.
4. In both indoor and miner studies, the questions of risk to non-smokers and the interrelation between radon exposure and smoking history should be addressed.
5. Laboratory based investigations of the effects of alpha emitters in general, and radon in particular, should be carried out.
6. To help in the prediction of risk, especially at lower exposures not accessible to epidemiological studies, the elucidation of the mechanisms by which radon and its progenies induce lung cancer, and the construction of biologically-based models should be encouraged.

On risk management:

7. A comprehensive radon policy, with a built-in evaluation programme, should be designed in which the regulatory approach comprises a small part and into which other aspects such as risk assessment, measurement protocols, contractor training and mitigation approaches should be integrated.
8. A national policy should be taken in a step by step manner from characterization of the problem to implementation. Accordingly, different types of pilot studies should be carried out to identify potential sources of elevated radon exposure, detailed national surveys should be initiated, and a national risk management strategy should be defined.
9. Construction materials, which are a particular source of radon, should be avoided, and building codes and guidelines, which apply especially to radon affected areas, should be developed.
10. When addressing the population at risk, the risk reducing strategy should treat homes, school and any other public buildings.
11. Risk reduction strategy should also include ordinary workplaces by applying occupational standards in industry, where applicable. Where radon exposure is incidental to the work situation (e.g. normal offices), the applicable action levels and guidelines should be the same as in homes.
12. Priority should be given to risk reduction for individuals at high risk, while at the same time the reduction of the overall population risk should be addressed.

13. Indoor situation with individual risks exceeding 10^{-3} per year should always be considered severe, and action to reduce the risk should always be taken.
14. When individual risk levels are less severe than 10^{-3} per year, further risk reduction should be sought, based on procedures that include criteria such as optimization, evaluation of available control techniques and others. These procedures should not be limited to a cost-benefit analysis, and social and equity considerations as well as comparisons with radiation protection and other relevant risk management approaches should also play a role.
15. The actual setting of indoor radon limits below the region of the acceptable risk should be governed by integrated risk approach and by the conventional radiation protection approach (in the given order of preference).
16. In the future, the radon policy should be integrated into a global risk management strategy, for which the various policy tools are developed in a consistent way.

On risk communication:

17. Countries should collect baseline information on existing attitudes and perceptions about radon before initiating a communication programme.
18. The setting of communications goals should be the first step in initiating a radon communication programme.
19. Continuous evaluation and improvement of the communication programme should take place in order to achieve the overall communications goals.
20. For special target audiences, appropriate (and possibly different) radon communication messages should be prepared.
21. Radon communication efforts should be linked to key issues, such as availability of qualified radon contractors, testing services, etc.
22. Radon messages should be repeated and reinforced, as one message one time will not result in societal behaviour changes.
23. Countries should be encouraged to share information and discuss new findings in the area of radon risk communication.

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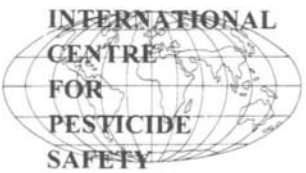

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