Nutrition and Health Series Editors: Adrianne Bendich · Connie W. Bales

James M. Rippe Editor

Nutrition in Lifestyle Medicine



NUTRITION AND HEALTH

Adrianne Bendich, Ph.D., FACN, FASN, Connie W. Bales, Ph.D., R.D., SERIES EDITORS

More information about this series at http://www.springer.com/series/7659

James M. Rippe Editor

Nutrition in Lifestyle Medicine

💥 Humana Press

Editor James M. Rippe Rippe Lifestyle Institute Shrewsbury, MA USA

Nutrition and Health ISBN 978-3-319-43025-6 ISBN 978-3-319-43027-0 (eBook) DOI 10.1007/978-3-319-43027-0

Library of Congress Control Number: 2016959599

© Springer International Publishing Switzerland 2017

This work is subject to copyright. All rights are reserved by the Publisher, whether the whole or part of the material is concerned, specifically the rights of translation, reprinting, reuse of illustrations, recitation, broadcasting, reproduction on microfilms or in any other physical way, and transmission or information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed.

The use of general descriptive names, registered names, trademarks, service marks, etc. in this publication does not imply, even in the absence of a specific statement, that such names are exempt from the relevant protective laws and regulations and therefore free for general use.

The publisher, the authors and the editors are safe to assume that the advice and information in this book are believed to be true and accurate at the date of publication. Neither the publisher nor the authors or the editors give a warranty, express or implied, with respect to the material contained herein or for any errors or omissions that may have been made.

Printed on acid-free paper

This Humana Press imprint is published by Springer Nature The registered company is Springer International Publishing AG The registered company address is Gewerbestrasse 11, 6330 Cham, Switzerland To my beautiful wife Stephanie Hart Rippe and our four exceptional daughters, Hart, Jaelin, Devon, and Jamie, who continue to support and love me and make it all worthwhile.

Preface

An overwhelming body of literature supports the concept that what each of us does on a daily basis significantly impacts both our short- and long-term health and quality of life. This influence may be either positive or negative. Hundreds, if not thousands, of studies provide evidence that maintenance of a healthy weight, regular physical activity, not smoking cigarettes, and following sound nutritional and other health-promoting practices all profoundly impact health. Nutrition, in particular, plays a prominent role in multiple aspects of positive lifestyle and good health.

Moving forward it is clear that the field of how daily habits and actions impact on health will be called "lifestyle medicine." An academic professional organization has been established, the American College of Lifestyle Medicine. ACLM has doubled in membership over each of the last 2 years. The National Meeting of the ACLM this year had over 800 attendees. The Council of the American Heart Association (AHA) which was previously named the "Council on Nutrition, Physical Activity, and Metabolism" changed its name in 2013 to the "Council on Lifestyle and Cardiometabolic Health." In addition, the AHA launched an ambitious series of essays published in *Circulation* entitled "Recent Advances in Preventive Cardiology and Lifestyle Medicine: A Themed Series." In addition, the debate over the past few years over the Affordable Care Act has drawn widespread attention to the important role that lifestyle practices play in achieving both cost containment and positive health outcomes.

Researchers at my laboratory, Rippe Lifestyle Institute (RLI), have been particularly active in studying and publishing how habits and actions impact on health. In fact, the RLI team named this field "lifestyle medicine" with the publication of my first multiauthored academic textbook *Lifestyle Medicine* (Blackwell Science, 1999). The second edition of this major academic textbook was published by CRC Press in 2013 and contains a variety of aspects of lifestyle medicine. I also edit the only peer-reviewed journal in this area, the *American Journal of Lifestyle Medicine* (SAGE Publishing), and a two-volume *Encyclopedia of Lifestyle Medicine and Health* (SAGE Publishing 2012).

Perhaps no single area is more important in health-promoting practices than nutrition. In fact, guidelines and consensus statements from virtually every major professional medical organization contain significant emphasis on nutrition principles and practices as key components of the prevention and treatment of disease.

Despite widespread emphasis and abundant knowledge concerning the interaction between lifestyle practices and health, it has been frustratingly difficult to improve nutritional practices in the American population. Witness the following:

- Over two thirds of the adult population in the United States is either overweight or obese (a staggering 40% increase over the past 20 years).
- Less than one third of the adult population consumes adequate servings of fruits and vegetables and follows simple evidence-based nutritional principles for good health.
- The prevalence of diabetes in the United States has doubled in the past 20 years.

- Over one third of the adult population in the United States has high blood pressure, yet less than 20% of individuals with high blood pressure follow the American Heart Association DASH Diet recommended as the proper nutrition framework for controlling blood pressure.
- Despite improvements over the past 20 years, cardiovascular disease (CVD) remains the leading killer of both men and women in the United States, resulting in 37 % of mortality each year. CVD has multiple nutritional factors as a component of overall lifestyle choices.

Finding practical strategies and ways of implementing these strategies to help people make proper nutritional choices in their lives is an urgent mandate in the United States and around the world. Yet, many individuals in the health-care community do not have adequate skills to provide nutritional counseling and do not incorporate this modality into the regular practice of medicine. Despite the fact that the Affordable Care Act provides some reimbursement for nutritional counseling, very few health-care practitioners or organizations are utilizing this provision.

For all of the above reasons, I felt the time had come to devote an entire academic textbook to how nutrition interacts with lifestyle. The goal of this book is to provide up-to-date, evidence-based information concerning nutrition, not only in the prevention and treatment of disease but also as a key component of an overall healthy lifestyle. In addition to providing information, this book provides practical strategies on how to incorporate healthy nutritional practices into the daily lives of children and adults.

A particular emphasis of *Nutrition in Lifestyle Medicine* is on nutritional considerations related to obesity, diabetes, and cardiovascular disease. These three metabolically based diseases are so closely aligned that it has been suggested that this field should be called "cardiodiabesity."

In addition to an emphasis on these three disease processes, *Nutrition in Lifestyle Medicine* also focuses on a variety of specialized areas such as nutrition for athletes and physically active individuals, hydration, and nutrition throughout the life cycle—spanning from children to individuals over the age of 60. In addition, chapters also include controversies in nutrition such as the health consequences of added sugars in the diet and saturated fatty acids. Specialized chapters are also included in such areas as nutrition for a healthy pregnancy, nutrition for men, nutrition and behavior change, and strategies for helping adults manage what they eat. Separate chapters are also devoted to the emerging role of genetics in nutrition, the concept of nutritional status, how to evaluate nutrition research, and public policy issues including the important issue of food safety.

In the important area of children's nutrition, an entire three-chapter section is included, containing important chapters on childhood obesity and managing cholesterol and other risk factors for heart disease in children. Since most children eat lunch at school, an evidence-based chapter on the nutritional aspects of school lunches concludes this section.

Authors for *Nutrition in Lifestyle Medicine* were drawn from recognized experts in each of these areas. The emphasis throughout the book is on how health-care professionals can incorporate information and advice into their practices to encourage individuals to adopt healthier nutritional habits in their daily lives and throughout the life cycle.

What has emerged is an academic book defining how nutrition plays a critically important role in our daily lives. We hope this book will be useful to all health-care professionals as they incorporate more nutritional counseling in the overall practice of health care. As the roles of daily habits and actions continue to emerge as key areas both in the prevention and treatment of disease, we hope that this book will serve as a useful tool for health-care professionals as they work on nutritional practices with their multiple patients and for the benefit of public health in general.

Boston, MA

James M. Rippe, MD

Series Editor Page

The great success of the Nutrition and Health Series is the result of the consistent overriding mission of providing health professionals with texts that are essential because each includes (1) a synthesis of the state of science; (2) timely, in-depth reviews by the leading researchers and clinicians in their respective fields; (3) extensive, up-to-date fully annotated reference lists; (4) a detailed index; (5) relevant tables and figures; (6) identification of paradigm shifts and the consequences; (7) virtually no overlap of information between chapters, but targeted, interchapter referrals; (8) suggestions of areas for future research; and (9) balanced, data-driven answers to patient and health professional questions which are based upon the totality of evidence rather than the findings of any single study.

The series volumes are not the outcome of a symposium. Rather, each editor has the potential to examine a chosen area with a broad perspective, both in subject matter and in the choice of chapter authors. The international perspective, especially with regard to public health initiatives, is emphasized where appropriate. The editors, whose trainings are both research and practice oriented, have the opportunity to develop a primary objective for their book, define the scope and focus, and then invite the leading authorities from around the world to be part of their initiative. The authors are encouraged to provide an overview of the field, discuss their own research, and relate the research findings to potential human health consequences. Because each book is developed de novo, the chapters are coordinated so that the resulting volume imparts greater knowledge than the sum of the information contained in the individual chapters.

Nutrition in Lifestyle Medicine edited by Dr. James M. Rippe, MD, is a very welcome addition to the Nutrition and Health Series and fully exemplifies the Series' goals. This unique volume represents the first fully referenced text to provide an integrated review of the concept of nutritional status and how this status is related to overall health as well as disease risk with particular emphasis on cardio-vascular disease, diabetes, and obesity. The volume includes balanced, data-driven discussions of the beneficial and potentially harmful effects of certain behaviors that affect both the quality and quantity of food that is consumed. The explosion of clinical research in the field of lifestyle medicine over the last two decades warrants this 23-chapter tome. The volume is designed as an important resource for nutritionists and dietitians, research and public health scientists, cardiologists, gastroenterologists and related physicians, and health-care professionals who interact with clients, patients, and/or family members. The volume provides objective, relevant information for professors and lecturers, advanced undergraduates and graduates, researchers, and clinical investigators who require extensive, up-to-date literature reviews, instructive tables and figures, and excellent references on the importance of nutrition as a keystone in the practice of lifestyle medicine.

The editor of this comprehensive volume is Dr. James Rippe, MD. He established and, for the past 25 years, has led the largest research organization in the world, exploring how daily habits and actions impact short- and long-term health and quality of life. This organization, Rippe Lifestyle Institute, has documented, in peer-reviewed publications, the scientific basis for the fields of lifestyle medicine and high-performance health. Rippe Lifestyle Institute also conducts numerous studies every year on nutrition and healthy weight management. Dr. Rippe has also edited another timely volume for the

Nutrition and Health Series entitled *Fructose*, *High Fructose Corn Syrup*, *Sucrose and Health* that was published in 2014. Dr. Rippe is a graduate of Harvard College and Harvard Medical School with postgraduate training at Massachusetts General Hospital. Dr. Rippe is professor of biomedical sciences at the University of Central Florida in Orlando, Florida; founder and director of Rippe Lifestyle Institute in Shrewsbury, Massachusetts, and Orlando, Florida; and founder and director of Rippe Health Evaluation in Orlando, Florida.

This comprehensive volume contains 23 chapters that are organized in seven parts: "Nutrition in Lifestyle Medicine: General Considerations," "Nutrition in Prevention and Treatment of Metabolic Diseases," "Nutrition in Childhood," "Nutrition in Athletes and Physically Active Adults," "Nutrition in Specialized Populations and Conditions," "Controversies in Nutrition and Lifestyle Medicine," and "Nutrition and Public Policy Issues."

Part I: Nutrition in Lifestyle Medicine: General Considerations

The first section in this volume contains six chapters, and the first chapter, written by the editor, describes in detail the goals of the Nutrition in Lifestyle Medicine volume. These include the provision of up-to-date, evidence-based information concerning nutrition and the prevention and treatment of disease in the context of an overall healthy lifestyle. In addition to providing information, this book is intended to provide practical strategies on how to incorporate healthy nutritional practices into the daily lives of children and adults. Emphasis of the volume is placed on nutritional considerations related to obesity, diabetes, and cardiovascular disease. These three metabolically based diseases are so closely related physiologically as well as pathologically that it has been suggested that these could be considered together in a global term called cardiodiabesity. In addition, the volume also focuses on a variety of specialized areas such as nutrition for athletes and physically active individuals, hydration, and nutrition throughout the life cycle spanning from children to individuals over the age of 60. Chapters are also included on controversies in nutrition such as sugars and health and saturated fats. Specialized chapters are included in such diverse areas as promoting nutrition in men's health, nutrition for a healthy pregnancy, and nutritional considerations for Hispanics. Since considerations of nutrition and health do not take place in a vacuum, a whole section is devoted to public health and public policy issues such as promoting an environment to support healthy eating and issues relating to food safety. Some practical issues related to frozen foods as well as school lunch programs are included as well as issues related to current or future research needs including nutrigenomics and metabolomics.

The second chapter contains a historical perspective of the development of dietary intake standards for large population groups over the past century and provides an overview of tools for evaluating dietary intakes of individuals. The chapter describes the concepts of dietary status, nutritional status, and the methods for their measurement and includes 12 informative tables and figures. The standards for nutrient intakes in the United States and Canada are described, and their multiple uses are discussed. The Dietary Guidelines for Americans and recommendations to help guide Americans, summarized in the pictogram entitled MyPlate (formerly MyPyramid and the Food Guide Pyramid), is described. The Healthy Eating Index, a simple scoring system for evaluating overall dietary, is also included. Healthy People 2020, a set of US national goals for promoting health and preventing disease that involve nutrition, and the new National Nutrition Research Roadmap for federal human nutrition research 2016–2021 are also discussed. The third chapter reflects upon the critical role of behavioral therapy to assist individuals in the process of improving their diet so that it includes more of the guidelines discussed in the second chapter. There is a detailed discussion of implementing SMART goals that stand for specific, measurable, action oriented, realistic, and time sensitive. The importance of aligning these goals with the person's life goals helps to assure that the goals are ones that the patient is motivated to accomplish in the short and long term. The value of accountability is also stressed using the COACH approach which is defined as the nutrition counseling process that is full of curiosity, openness, appreciation, compassion, and honesty. These methodologies are contrasted and compared with other behavioral change programs, and the chapter includes over 150 relevant references. Chapter 4, coauthored by the volume's editor, expands upon the complexities involved in the control of eating behaviors. There are comprehensive discussions of neurophysiological and genetic factors, hunger and satiety signaling systems in the brain and gut, the brain's reward system, and the role of dopamine. Also included is a review of psychological factors that affect eating behavior and food choices such as emotions and moods, disinhibition, and impulsivity. The chapter includes over 150 references and practical suggestions to help clients and patients develop more mindful and attentive eating habits.

The last two chapters in this introductory section provide unique perspectives concerning the methodologies used to study human nutrition as well as the important role of genetic predisposition in determining what we eat and how much we eat. Chapter 5 examines the major factors involved in clearly understanding the research that is published in the peer-reviewed literature. The chapter is designed to assist the readers of scientific literature in identifying some of the challenges in evaluating what exactly was studied, determining how exactly it was studied, and interpreting what can be concluded from principally human research studies. Key areas discussed include the challenges in defining exposures and outcomes; potential for oversimplification of complex concepts; differences in study designs; surrogate and hard endpoints; bias, confounding, objective data versus interpretation of data; logical fallacies; and differences between scientific conclusions and evidence-based decisions. Numerous relevant case studies and informative tables are included for the reader. Nutritional genomics is the topic of the last chapter in this section, and we learn that this relatively new field has already found a number of instances where genetic mutations that affect nutrient metabolism likewise affect specific risks of disease. Nutritional genomics is a broad term that recognizes the effects of one's genome on nutrient metabolism and how diet can alter an individual's genes and health. Nutrigenomics includes functional interactions and synergies between dietary components and the genome. The chapter provides answers to questions such as: How do genetic polymorphisms affect nutrient requirements? How does nutrition influence gene expression and metabolic pathways? How is regulation altered or disturbed in diet-related diseases? There are discussions of polygenic diseases including obesity, diabetes, cardiovascular disease, cancer, and other complex conditions. Some common genes have been identified such as the genes for apolipoprotein E and methylenetetrahydrofolate reductase. The importance of educating dietitians and other health providers about the emerging science of nutrigenomics is stressed, and guidance is provided concerning sources of reliable information.

Part II: Nutrition in Prevention and Treatment of Metabolic Diseases

Part II contains four chapters that concentrate on the prevention and treatment of the cardiodiabesity area of disease management with emphasis on nutritional aspects. Chapter 7, coauthored by the editor, examines the role of nutrition and lifestyle in both prevention and treatment of cardiovascular disease. The studies that have demonstrated that not smoking; engaging in at least 30 min of physical activity per day; consuming a diet containing more fish, whole grains, fruits, and vegetables; and maintaining a healthy weight have shown that these healthy lifestyles can reduce the risk of coronary heart disease by over 80% in both men and women. The major guidelines and references are reviewed in detail. Diabetes (both types I and II) and prediabetes are discussed in the next chapter with emphasis on nutritional intervention for prevention of the prediabetic patient becoming diabetic. Goals of nutrition therapy for prediabetes to diabetes by selection of food choices that facilitate moderate weight loss and by an increase in physical activity. Topics such as bariatric surgery, glycemic index, weight loss medications, types of fats, and targeted exercise programs are included along with seven relevant tables. Nutrition in weight management and obesity is the topic of the Chap. 9 that reviews the physiological

and environmental factors associated with the development of obesity and reminds us that at any time 50–70% of obese persons are dieting. There is an in-depth discussion of evidence-based dietary treatment of obesity using medical nutrition therapy (MNT); the chapter includes over 125 references. The evidence that MNT improves weight, waist circumference, hip circumference, fasting blood glucose, LDL cholesterol, HDL cholesterol, and blood pressure is reviewed. Weight loss studies and evidence-based programs as well as commercial programs and weight loss myths are examined.

The last chapter in Part II, Chap. 10, reviews the association between nutritional status and oral health. The authors remind us that diet and nutrition play a key role in tooth development, gingival and oral tissue integrity, bone strength, and prevention and management of diseases of the oral cavity. Caries in children and cariogenic foods are reviewed. There is also an in-depth review of the interrelationship between foods that adversely affect teeth and gums and their potential for diminishing the beneficial effects of consuming foods that can improve oral health. Poor oral health, including periodontal disease, is associated with the cardiodiabesity examined in the prior three chapters as well as autoimmune disorders, human immunodeficiency virus infection, eating disorders, and oropharyngeal cancer. Oral health is adversely affected by most of the chemotherapies for cancers and results in decreased food consumption that is often linked to loss of saliva and teeth. Dietary components that are associated with benefits to oral health are included.

Part III: Nutrition in Childhood

Part III includes three chapters that examine the increased risk of childhood obesity, cardiovascular disease, and lipid disorders and the role of the school lunch programs in child health. Chapter 11 provides a broad overview of the statistics on childhood obesity including measurement guidelines, discussion of comorbidities, and also examination of potential mechanisms to avoid the progression to adult obesity. Population-wide strategies, lifestyle interventions, and individual prevention strategies including drug therapy and weight loss surgery are reviewed. The following chapter examines the potential for development of cardiovascular disease in children, and as we learned in Chap. 11, obesity is a strong risk factor for increased blood pressure and other adverse cardiovascular effects. Studies have determined that genetics plays a minor role in the maintenance of ideal cardiovascular health. Less than 20% is due to genetics, and 80% or more is determined by lifestyle factors such as diet and physical activity. Interventions to lower cholesterol-linked risk in children are emphasized. The final chapter in this section, Chap. 13, reviews the role of school lunch programs and their impact on childhood health. Of importance was the implementation of the 2010 Healthy, Hunger-Free Kids Act that has resulted in measurable improvements in school meal intakes that have been documented by both plate-waste studies and self-reported intakes. We are reminded about the huge scope of these school meal programs. Schools in the United States serve over 14 million breakfasts and 30 million lunches every school day, as well as multiple snacks and supper meals in some locations. These meals are funded by federal legislation and regulated by the US Department of Agriculture nutrition standards, which are designed to follow the Dietary Guidelines for Americans that were outlined in Chap. 2.

Part IV: Nutrition in Athletes and Physically Active Adults

The two complementary chapters in this section examine the importance of nutrient-dense foods and proper caloric intake for the active adult and adolescent and also the importance of water intake in the actively exercising person. Chapter 14 examines the many biological, physical, psychological, and

behavioral changes that occur during adolescence. The chapter reviews the most favorable dietary intakes and habits that minimize unhealthful weight gain, maximize performance, and promote the development of positive lifelong health behaviors. Each of the macro- and essential micronutrient requirements during the three stages of adolescent growth is discussed, and there is a review of beverage choices and dietary supplements. The next chapter outlines the importance of hydration, especially for athletes and exercisers. The chapter reviews the data that shows that moderate reductions in body water result in changes in cardiovascular system function as well as altering cognitive function and mood. A significant number of elite athletes, recreational exercisers, and those with active occupations may have periods during the day when they are under-hydrated that may be worsened further by sweat losses during activity. Rehydration after the completion of physical activity is necessary. Undertaking physical activity in a lower than recommended hydrated state may increase an individual's perceived exertion which may negatively influence exercise performance and self-selected exercise intensity and may decrease the likelihood of further participation in physical activity. Chronically, the lack of hydration and an imbalance in the body's salt concentration can affect cardiovascular functions.

Part V: Nutrition in Specialized Populations and Conditions

The four chapters in the fifth section examine the importance of nutrition for a healthy pregnancy, in men's health, in Hispanics, and in older adults. Chapter 16 describes the modern woman who becomes pregnant. Many women enter pregnancy in an overweight or obese state that may also be linked to type II diabetes and hypertension. Women are becoming pregnant at older ages; older pregnant women have an increase in pregnancy-related risks. There are more women entering pregnancy with a history of chronic medical problems including type I diabetes, epilepsy, cancer, and multiple sclerosis or organ transplants such as the kidney or heart. Many women have undergone advanced fertility technologies and are at risk for pregnancy-related adverse effects. Nutrition has become key to a healthy pregnancy, and the prepregnancy window is especially important since the findings of the benefits of periconceptional multivitamins containing folic acid for the prevention of neural tube birth defects. The nutritional needs of the woman who is pregnant with multiple fetuses are reviewed as are numerous specific foods and dietary ingredients including alcohol. In Chap. 17, we examine the data indicating that there is a widening gender disparity that has resulted in men currently dying on average 5-6years earlier than women. Men, compared to age-matched women, have higher mortality rates for the majority of leading causes of death within all age ranges, including those in which diet plays a significant role such as cardiovascular disease, diabetes, obesity, and various forms of cancer. The chapter examines in depth the common masculine associations with foods such as beer and large portions of meat and the social aspects of eating in groups, food preparation, and the relative lack of nutritional knowledge compared to women. The chapter includes over 150 relevant references.

Hispanics are the fastest-growing population group in the United States and reflect many different countries of origin, food choices, and dietary habits that are reviewed in Chap. 18. The chapter provides an overview of the Hispanic populations in the United States, a detailed description of dietary behaviors and influencing factors observed in the population, and a discussion of dietary considerations and interventions. This chapter is especially relevant as Hispanics in the United States have higher rates of obesity and diabetes, lower health literacy, and lower access to health care than agematched non-Hispanics. Moreover, several health outcomes and disparities are diet-related. This comprehensive chapter contains six tables and figures including a tabulation of relevant clinical studies and over 100 important references.

Older adults represent a second population group at risk for nutritional inadequacies, and Chap. 19 reviews the bases of optimal nutrition for this population. The chapter includes a review of the way the older body handles nutrients as there is a decline in the functioning of organ systems that impacts

the utilization of specific nutrients. Many of the affected organs are involved in the metabolism of food including the stomach and small intestine, liver, heart, kidneys, skin, immune system, and oral cavity. Older adults often experience a decline in gastric hydrochloric acid secretion that can result in a decline in the bioavailability of vitamin B12. Older adults may be at a compromised status for vitamin D and, consequently, for calcium absorption. Changes in body composition (decreased lean muscle mass and increased fat mass) result in decreased basal metabolic rates, energy needs, and capacity for physical activity. Increased use of prescription and non-prescription medications, chronic drug therapy, and decreased capacity of the liver to metabolize drugs can compromise nutrient unitization. The chapter's relevant tables and figures help to describe the nutritional requirements of the older adult.

Part VI: Controversies in Nutrition and Lifestyle Medicine

Two of the most controversial areas in nutrition today involve the questions of whether added sugars and saturated fats are all bad, especially added sugar on beverages for children and saturated fats (regardless of source) for those at risk for cardiovascular/cerebrovascular diseases. Thus, this unique and valuable section provides objective reviews for both areas. Chapter 20 is coauthored by the editor, who, as mentioned previously, has edited a volume for the Series on this topic. The chapter contains 120 references and six figures/tables that provide a comprehensive review of the literature on metabolism and health effects of added sugars, emphasizing the differences between epidemiologic studies, ecological studies, and randomized, control intervention studies. The focus is on the health effects of the major fructose-containing sugars in the human diet, namely, sucrose and high fructose corn syrups, both of which contain roughly one half fructose and half glucose. Data provide inconsistent findings related to adverse effects of added sugars. However, clinical research studies in which added sugars are substituted for other carbohydrates of the same caloric value do not find adverse metabolic effects of the sugars.

As with the previous chapter, Chap. 21 examines the inconsistent findings linking saturated fats to increased risk of cardiovascular disease. The chapter reviews survey studies as well as intervention studies and suggests that the source of the saturated fat, the quantity consumed, and duration of consumption along with concurrent consumption of other foods are a few of the mitigating factors that continue to interfere with answering this question. Moreover, as with every aspect of nutrition research, the data are complex including the fact that saturated fat increases LDL cholesterol, which is positively associated with cardiovascular disease, while at the same time it increases HDL cholesterol, which is associated with reducing the risk of cardiovascular disease.

Part VII: Nutrition and Public Policy Issues

The final part of the volume contains two overviews of critically important public policy issues: guidelines for healthy eating as well as implementation strategies and a second chapter that reviews food safety issues. Chapter 22 discusses the current national healthy eating guidelines and then examines whether these are implemented at state and local levels; in schools, stores, and restaurants; in food labeling and advertising; and in other areas where nutrition knowledge could affect food choices. Chapter 23 reminds us that according to the Centers for Disease Control and Prevention, about 1 in every 6 US residents suffers a bout of foodborne illness every year. Of the estimated 48 million people

who have foodborne illness annually, 128,000 become so seriously ill they require hospitalization and 3000 die. The data reviewed indicate that fresh foods, including produce, meats, fish, and eggs, are the greatest sources of foodborne illnesses and deaths. In addition to bacteria and parasites, toxins, viruses, fungi, and prions can cause foodborne harm. The home environment remains the place of most exposure to foodborne illnesses, and the chapter reviews practical safety measures to implement in the home. The six tables and figures contain documented data on sources of contamination and ways to avoid these types of adverse events that are of particular danger to older individuals.

Conclusions

The above description of the volume's 23 chapters attests to the depth of information provided by the 45 well-recognized and respected chapter authors. Each chapter includes complete definitions of terms with the abbreviations fully defined and consistent use of terms between chapters. Key features of this comprehensive volume include over 80 detailed tables and informative figures; an extensive, detailed index; and more than 1800 up-to-date references that provide the reader with excellent sources of worthwhile information that will be of great value to the health provider as well as graduate and medical students.

In conclusion, Nutrition in Lifestyle Medicine edited by Dr. James M. Rippe, MD, provides health professionals in many areas of research and practice with the most up-to-date, well-referenced volume on the importance of nutrition as a key component of lifestyle medicine. The chapters review the role of food, nutrients, beverages, and other components of diet in maintaining the overall health of the healthy individuals as well as affecting the well-being of the patient with certain disease conditions, especially cardiovascular disease, diabetes, and obesity. The volume serves the reader as the benchmark in this complex area of interrelationships between nutrients, foods, social aspects, ethic factors, public policies, physical activity, pregnancy, men's health, older adults, adolescents, children, school food programs, controversies within the nutrition research community that impact the public, and the critical area of food safety. Moreover, the physiological, genetic, and pathological interactions between blood levels of, for instance, saturated fats and the functioning of the endothelium in the gastrointestinal tract, vascular system, adipose tissue, and brain chemistry are clearly delineated so that students as well as practitioners can better understand the complexities of these interactions. The editor, Dr. James M. Rippe, MD, is applauded for his efforts to develop the most authoritative and unique resource on the importance of nutrition in the achievement of long-term health under the guidance of lifestyle medicine, and this excellent text is a very welcome addition to the Nutrition and Health Series.

Adrianne Bendich PhD, FACN, FASN

About the Series Editors



Dr. Adrianne Bendich, PhD, FASN, FACN, has served as the "Nutrition and Health" Series editor for 20 years and has provided leadership and guidance to more than 200 editors that have developed the 70+ well-respected and highly recommended volumes in the Series.

In addition to *Nutrition in Lifestyle Medicine edited by Dr. James M. Rippe, MD*, major new editions published in 2012–2016 include:

- 1. *Mediterranean Diet: Dietary Guidelines and Impact on Health and Disease.*, edited by Donato F. Romagnolo, PhD, and Ornella Selmin, PhD, 2016.
- Nutrition Support for the Critically Ill, edited by David S. Seres, MD, and Charles W. Van Way, III, MD, 2016
- 3. *Nutrition in Cystic Fibrosis: A Guide for Clinicians*, edited by Elizabeth H. Yen, MD, and Amanda R. Leonard, MPH, RD, CDE, 2016
- 4. *Preventive Nutrition: The Comprehensive Guide for Health Professionals*, Fifth Edition, edited by Adrianne Bendich, PhD, and Richard J. Deckelbaum, MD, 2016.
- Glutamine in Clinical Nutrition, edited by Rajkumar Rajendram, Victor R. Preedy, and Vinood B. Patel, 2015
- 6. Nutrition and Bone Health, Second Edition, edited by Michael F. Holick and Jeri W. Nieves, 2015
- 7. *Branched Chain Amino Acids in Clinical Nutrition, Volume 2*, edited by Rajkumar Rajendram, Victor R. Preedy, and Vinood B. Patel, 2015
- 8. *Branched Chain Amino Acids in Clinical Nutrition, Volume 1*, edited by Rajkumar Rajendram, Victor R. Preedy, and Vinood B. Patel, 2015
- 9. Fructose, High Fructose Corn Syrup, Sucrose and Health, edited by James M. Rippe, 2014
- Handbook of Clinical Nutrition and Aging, Third Edition, edited by Connie Watkins Bales, Julie L. Locher, and Edward Saltzman, 2014
- 11. *Nutrition in Pediatric Pulmonary Disease*, edited by Dr. Youngran Chung and Dr. Robert Dumont, 2014
- 12. *Integrative Weight Management*, edited by Dr. Gerard E. Mullin, Dr. Lawrence J. Cheskin, and Dr. Laura E. Matarese, 2014
- 13. *Nutrition in Kidney Disease, Second Edition*, edited by Dr. Laura D. Byham-Gray, Dr. Jerrilynn D. Burrowes, and Dr. Glenn M. Chertow, 2014

- 14. *Handbook of Food Fortification and Health, Volume I*, edited by Dr. Victor R. Preedy, Dr. Rajaventhan Srirajaskanthan, and Dr. Vinood B. Patel, 2013
- 15. *Handbook of Food Fortification and Health, Volume II*, edited by Dr. Victor R. Preedy, Dr. Rajaventhan Srirajaskanthan, and Dr. Vinood B. Patel, 2013
- 16. *Diet Quality: An Evidence-Based Approach, Volume I*, edited by Dr. Victor R. Preedy, Dr. Lan-Ahn Hunter, and Dr. Vinood B. Patel, 2013
- 17. *Diet Quality: An Evidence-Based Approach, Volume II*, edited by Dr. Victor R. Preedy, Dr. Lan-Ahn Hunter, and Dr. Vinood B. Patel, 2013
- Handbook of Clinical Nutrition and Stroke, edited by Mandy L. Corrigan, MPH, RD; Arlene A. Escuro, MS, RD; and Donald F. Kirby, MD, FACP, FACN, FACG, 2013
- 19. *Nutrition in Infancy, Volume I*, edited by Dr. Ronald Ross Watson, Dr. George Grimble, Dr. Victor Preedy, and Dr. Sherma Zibadi, 2013
- 20. *Nutrition in Infancy, Volume II*, edited by Dr. Ronald Ross Watson, Dr. George Grimble, Dr. Victor Preedy, and Dr. Sherma Zibadi, 2013
- 21. Carotenoids and Human Health, edited by Dr. Sherry A. Tanumihardjo, 2013
- 22. *Bioactive Dietary Factors and Plant Extracts in Dermatology*, edited by Dr. Ronald Ross Watson and Dr. Sherma Zibadi, 2013
- 23. *Omega-6/3 Fatty Acids*, edited by Dr. Fabien De Meester, Dr. Ronald Ross Watson, and Dr. Sherma Zibadi, 2013
- 24. Nutrition in Pediatric Pulmonary Disease, edited by Dr. Robert Dumont and Dr. Youngran Chung, 2013
- 25. Magnesium and Health, edited by Dr. Ronald Ross Watson and Dr. Victor R. Preedy, 2012.
- 26. *Alcohol, Nutrition, and Health Consequences*, edited by Dr. Ronald Ross Watson, Dr. Victor R. Preedy, and Dr. Sherma Zibadi, 2012
- 27. *Nutritional Health: Strategies for Disease Prevention, Third Edition*, edited by Norman J. Temple, Ted Wilson, and David R. Jacobs, Jr., 2012
- 28. *Chocolate in Health and Nutrition*, edited by Dr. Ronald Ross Watson, Dr. Victor R. Preedy, and Dr. Sherma Zibadi, 2012
- 29. *Iron Physiology and Pathophysiology in Humans*, edited by Dr. Gregory J. Anderson and Dr. Gordon D. McLaren, 2012

Earlier books included Vitamin D, Second Edition edited by Dr. Michael Holick; Dietary Components and Immune Function edited by Dr. Ronald Ross Watson, Dr. Sherma Zibadi, and Dr. Victor R. Preedy; Bioactive Compounds and Cancer edited by Dr. John A. Milner and Dr. Donato F. Romagnolo; Modern Dietary Fat Intakes in Disease Promotion edited by Dr. Fabien De Meester, Dr. Sherma Zibadi, and Dr. Ronald Ross Watson; Iron Deficiency and Overload edited by Dr. Shlomo Yehuda and Dr. David Mostofsky; Nutrition Guide for Physicians edited by Dr. Edward Wilson, Dr. George A. Bray, Dr. Norman Temple, and Dr. Mary Struble; Nutrition and Metabolism edited by Dr. Christos Mantzoros; and Fluid and Electrolytes in Pediatrics edited by Leonard Feld and Dr. Frederick Kaskel. Recent volumes include: Handbook of Drug-Nutrient Interactions edited by Dr. Joseph Boullata and Dr. Vincent Armenti; Probiotics in Pediatric Medicine edited by Dr. Sonia Michail and Dr. Philip Sherman; Handbook of Nutrition and Pregnancy edited by Dr. Carol Lammi-Keefe, Dr. Sarah Couch, and Dr. Elliot Philipson; Nutrition and Rheumatic Disease edited by Dr. Laura Coleman; Nutrition in Kidney Disease edited by Dr. Laura Byham-Gray, Dr. Jerrilynn Burrowes, and Dr. Glenn Chertow; Nutrition and Health in Developing Countries edited by Dr. Richard Semba and Dr. Martin Bloem; Calcium in Human Health edited by Dr. Robert Heaney and Dr. Connie Weaver; and Nutrition and Bone Health edited by Dr. Michael Holick and Dr. Bess Dawson-Hughes.

Dr. Bendich is president of Consultants in Consumer Healthcare, LLC, and is the editor of ten books including *Preventive Nutrition: The Comprehensive Guide for Health Professionals, Fifth Edition* coedited with Dr. Richard Deckelbaum (www.springer.com/series/7659). Dr. Bendich serves on the editorial boards of the *Journal of Nutrition in Gerontology and Geriatrics* and *Antioxidants* and has served as associate editor for *Nutrition*, the international journal; served on the editorial board of the *Journal of Gender-Based Medicine*; and served on the board of directors of the American College of Nutrition.

Dr. Bendich was director of Medical Affairs at GlaxoSmithKline (GSK) Consumer Healthcare and provided medical leadership for many well-known brands including TUMS and Os-Cal. Dr. Bendich had primary responsibility for GSK's support for the Women's Health Initiative (WHI) intervention study. Prior to joining GSK, Dr. Bendich was at Roche Vitamins Inc. and was involved with the groundbreaking clinical studies showing that folic acid-containing multivitamins significantly reduced major classes of birth defects. Dr. Bendich has coauthored over 100 major clinical research studies in the area of preventive nutrition. She is recognized as a leading authority on antioxidants, nutrition and immunity and pregnancy outcomes, vitamin safety, and the cost-effectiveness of vitamin/mineral supplementation.

Dr. Bendich received the Roche Research Award, is a *Tribute to Women and Industry* awardee, and was a recipient of the Burroughs Wellcome Fund Visiting Professorship in Basic Medical Sciences. Dr. Bendich was given the Council for Responsible Nutrition (CRN) Apple Award in recognition of her many contributions to the scientific understanding of dietary supplements. In 2012, she was recognized for her contributions to the field of clinical nutrition by the American Society for Nutrition and was elected a fellow of ASN. Dr Bendich is adjunct professor at Rutgers University. She is listed in *Who's Who of American Women*.



Connie W. Bales, Ph.D., R.D., is a Professor of Medicine in the Division of Geriatrics, Department of Medicine, at the Duke School of Medicine and Senior Fellow in the Center for the Study of Aging and Human Development at Duke University Medical Center. She is also Associate Director for Education/Evaluation of the Geriatrics Research, Education, and Clinical Center at the Durham VA Medical Center. Dr. Bales is a well-recognized expert in the field of nutrition, chronic disease, function, and aging. Over the past two decades her laboratory at Duke has explored many different aspects of diet and activity as determinants of health during the latter half of the adult

life course. Her current research focuses primarily on the impact of protein enhanced meals on muscle quality, function, and other health indicators during obesity reduction in older adults with functional limitations. Dr. Bales has served on NIH and USDA grant review panels and is a member of the American Society for Nutrition's Medical Nutrition Council. Dr. Bales has edited three editions of the Handbook of Clinical Nutrition in Aging and is Editor-in-Chief of the Journal of Nutrition in Gerontology and Geriatrics.

About the Editor



Dr. Rippe, MD, is a graduate of Harvard College and Harvard Medical School with postgraduate training at Massachusetts General Hospital. He is currently the founder and director of Rippe Lifestyle Institute.

Over the past 25 years, Dr. Rippe has established and run the largest research organization in the world exploring how daily habits and actions impact short- and long-term health and quality of life. This organization, Rippe Lifestyle Institute (RLI), has published hundreds of papers that form the scientific basis for the fields of lifestyle medicine and high-performance health. Rippe Lifestyle Institute also conducts numerous studies every year on nutrition and healthy weight management. One recent research interest of the RLI team has been the metabolism and health effects of sugars.

A lifelong and avid athlete, Dr. Rippe maintains his personal fitness with a regular walk, jog, swim, and weight training program. He holds a black belt in karate and is an avid wind surfer, skier, and tennis

player. He lives outside of Boston with his wife, television news anchor Stephanie Hart, and their four children, Hart, Jaelin, Devon, and Jamie.

Acknowledgments

Book writing and editing are collaborative processes. I would, first of all, like to thank all of the coauthors who made important contributions to this book. I put in front of each of them the challenge of not only assembling a strong body of evidence-based research related to nutrition and health but also making practical linkages to help health-care professionals integrate this knowledge into their daily practices. All authors rose admirably to this challenge.

Particular praise goes to my editorial director, Beth Grady, who manages all of my book projects with great skill, attention to detail, and good humor. These books would not be possible without the excellent efforts of Beth. My executive assistant, Carol Moreau, always plays the critical role in organizing my complex schedule to allow time for book writing and editing. Our office assistant, Deb Adamonis, assists in multiple editorial and logistical details for Rippe Lifestyle Institute.

I would also like to thank my superb team of researchers at Rippe Lifestyle Institute of Florida who have continued to generate important research in many areas of lifestyle medicine, in particular my research director, Dr. Ted Angelopoulos, who helps conceptualize and frame our various research protocols; Josh Lowndes, our associate research director, who has carried on day-to-day responsibilities for most of our research projects; and Noy Supaswud, our highly capable office manager, who coordinates the schedules and other logistical aspects of a busy research organization. To all of these individuals, I owe a deep debt of gratitude.

Thanks also to our series editor Dr. Adrianne Bendich, who passionately believed in the concept of how nutrition should be linked to overall positive lifestyle and was an early champion of this book. I also appreciate the support and confidence of Rekha Udaiyar and the team at Springer Publishers/ Humana Press.

Contents

Part	t I Nutrition in Lifestyle Medicine: General Considerations	
1	Nutrition in Lifestyle Medicine: Overview	3
2	Nutrition 101: The Concept of Nutritional Status, Standards, and Guidesfor Nutrient Intakes, Eating Patterns, and NutritionJohanna T. Dwyer and Regan Lucas Bailey	13
3	Behavior Change and Nutrition Counseling Elizabeth Pegg Frates and Jonathan Bonnet	51
4	Effective Strategies to Help Adults Manage How Much They Eat	85
5	Critical Evaluation of Nutrition Research	103
6	Nutritional Genomics: The Wave of the Future for Nutrition and Dietetics	17
Part	t II Nutrition in Prevention and Treatment of Metabolic Diseases	
7	The Role of Nutrition and Lifestyle in the Prevention and Treatment of Cardiovascular Disease James M. Rippe and Theodore J. Angelopoulos	137
8	Nutrition Therapy for the Prevention and Treatment of Prediabetes and Diabetes 1 Marion J. Franz	151
9		
	Nutrition in Weight Management and Obesity1Tracey Ledoux, Tabbetha Lopez, Craig Johnston,1Elizabeth Vaughan, and John P. Foreyt1	173
10	Tracey Ledoux, Tabbetha Lopez, Craig Johnston,	
	Tracey Ledoux, Tabbetha Lopez, Craig Johnston, Elizabeth Vaughan, and John P. Foreyt Nutrition in Oral Health	

12	Nutritional Interventions to Lower Cholesterol and Risk for Heart Disease in Children Stephen R. Daniels, Jessica Hildebrandt, Laura K. Brennan, and Sarah C. Couch	229
13	School Meal Programs: Are They Nutritionally Sound? Dayle Hayes	251
Par	t IV Nutrition in Athletes and Physically Active Adults	
14	Nutritional Considerations for Young Athletes	267
15	Effects of an Active Lifestyle on Water Balance	281
Par	t V Nutrition in Specialized Populations and Conditions	
16	Nutrition for a Healthy Pregnancy Laurie Tansman	297
17	Promoting Nutrition in Men's Health Simon Rowlands and Brendan Gough	311
18	Nutritional Considerations for Hispanics Diana K. Cuy Castellanos	329
19	Optimal Nutrition for the Older Adults	355
Par	t VI Controversies in Nutrition and Lifestyle Medicine	
20	Added Sugars and Health: What Do We Really Know?	369
21	Saturated Fat: Friend or Foe?	387
Par	t VII Nutrition and Public Policy Issues	
22	Public Policy and Environmental Supports for Healthy Eating Zaida Cordero-MacIntyre, Hildemar Dos Santos, and Christy Mota	397
23	Food Safety	413

Contributors

Theodore J. Angelopoulos, PhD, MPH Health Sciences School of Health Sciences, Emory & Henry College, Emory, VA, USA

Regan Lucas Bailey, PhD, MPH, RD, CPH Office of Dietary Supplements, National Institutes of Health, Bethesda, MD, USA

Department of Nutrition Science, Purdue University, West Lafayette, IN, USA

Michelle M. Bohan Brown, PhD Food, Nutrition, and Packaging Sciences Department, Clemson University, Clemson, SC, USA

Jonathan P. Bonnet, MD Community Health and Family Medicine, University of Florida College of Medicine, Gainesville, FL, USA

Rebecca J. Boulos, MPH, PhD John Hancock Research Center on Physical Activity, Nutrition Obesity Prevention, Friedman School of Nutrition Science and Policy, Boston, MA, USA

Laura K. Brennan, BS, MS Candidate Department of Nutritional Sciences, University of Cincinnati Medical Center, Cincinnati, OH, USA

Andrew W. Brown, PhD Office of Energetics and Nutrition Obesity Research Center, University of Alabama at Birmingham, Birmingham, AL, USA

Carol Byrd-Bredbenner, PhD, RD, FADA Department of Nutritional Sciences, Rutgers University, New Brunswick, NJ, USA

Diana K. Cuy Castellanos, PhD, RD Health and Sport Science, University of Dayton, Dayton, OH, USA

Zaida Cordero-MacIntyre, PhD, PharmD, MPH, REHA, MS, RD Loma Linda University School of Public Health, Loma Linda, CA, USA

Sarah C. Couch, PhD, RD Department of Nutritional Sciences, University of Cincinnati Medical Center, Cincinnati, OH, USA

Stephen R. Daniels, MD, PhD Department of Pediatrics, Children's Hospital Colorado, Aurora, CO, USA

Anne-Marie Davee, MS, RD, LD University of New England, Portland, ME, USA

Hildemar Dos Santos, MD, DrPH, CSN, CHES, EPC Loma Linda University School of Public Health, Loma Linda, CA, USA

Johanna T. Dwyer, DSc, RD Frances Stern Nutrition Center, Tufts New England Medical Center, Boston, MA, USA

Gethin H. Evans, BSc, PhD, PgCAP School of Healthcare Science, Manchester Metropolitan University, Manchester, UK

John P. Foreyt, PhD Department of Medicine, Baylor College of Medicine, Houston, TX, USA

Marion J. Franz, MS, RD, CDE Nutrition Concepts by Franz, Inc, Minneapolis, MN, USA

Judith A. Gilbride, PhD, RDN, FAND Department of Nutrition and Food Studies, New York University, New York, NY, USA

Brendan Gough, PhD School of Social, Psychological & Communication Sciences, Leeds Metropolitan University, Leeds, UK

Dayle Hayes, MS, RD Nutrition for the Future, Inc., Billings, MT, USA

Daphne C. Hernandez, PhD, MSEd Department of Health & Human Performance, University of Houston, Houston, TX, USA

Jessica Hildebrandt, MS, RD Lifestyle Medicine Program, Children's Hospital Colorado, Aurora, CO, USA

Craig A. Johnston, PhD Department of Health & Human Performance, University of Houston, Houston, TX, USA

Wahida Karmally, PH, RD, CDE, CLS, FNLA Dean's Faculty, College of Dental Medicine, Irving Institute for Clinical and Translational Research, Columbia University, New York, NY, USA

Benoit Lamarche, PhD Institute on Nutrition and Functional Foods, Laval University, Quebec, QC, Canada

Tracey Ledoux, PhD, RD, FAND Department of Health & Human Performance, University of Houston, Houston, TX, USA

Alice H. Lichtenstein, DSc Cardiovascular Nutrition Laboratory, Tufts University, Boston, MA, USA

Tabbetha Lopez, RD Department of Health & Human Performance, University of Houston, Houston, TX, USA

Ronald J. Maughan, BSc, PhD Loughborough University, Loughborough, UK

Christy Mota, MPH Loma Linda University School of Public Health, Loma Linda, CA, USA

Elizabeth Pegg Frates, MD Stroke Research and Recovery Institute, Spaulding Rehabilitation Hospital, Boston, MA, USA

Department of Physical Medicine and Rehabilitation, Harvard Medical School, Boston, MA, USA

James M. Rippe, MD Rippe Lifestyle Institute, Shrewsbury, MA, USA

University of Central Florida, Orlando, FL, USA

Simon Rowlands, PhD Public Health/Health Promotion, Leeds Beckett University, West Yorkshire, UK

Susan M. Shirreffs, BSc, PhD Department of Medicine, St. Andrews University, St Andrews, UK

Abdullah Shuaib, BS Department of Health & Human Performance, University of Houston, Houston, TX, USA

Laurie Tansman, MS, RD, CDN Department of Clinical Nutrition, The Mount Sinai Hospital, New York, NY, USA

Department of Preventive Medicine, Icahn School of Medicine, New York, NY, USA

Elizabeth Vaughan, DO Department of Medicine, Baylor College of Medicine, Houston, TX, USA

Mary Abbott Waite, PhD Rippe Lifestyle Institute, Shrewsbury, MA, UK

Bridget L. Wardley, MS, RDN, CSP Department of Nutrition and Food Studies, New York University, New York, NY, USA

Atheer Yacoub, MS, RDN Irving Institute for Clinical and Translational Research, Columbia University Medical Center, New York, NY, USA

Part I Nutrition in Lifestyle Medicine: General Considerations

Chapter 1 Nutrition in Lifestyle Medicine: Overview

James M. Rippe

Key Points

- Nutrition plays a significant role in seven out of the ten leading causes of death worldwide.
- Sound nutritional practices play a central role in the prevention and treatment of chronic diseases in multiple evidence-based guidelines from various scientific organizations.
- Nutritional practices are a central component along with other lifestyle habits and practices that comprise the field called "Lifestyle Medicine."
- Influences on eating behaviors are complex including individual, family, community, and public policy factors.
- A key challenge remains to take the existing knowledge in sound nutrition and help individuals implement this knowledge in their daily lives.

Keywords Nutrition • Lifestyle medicine • Physical activity • Behavioral medicine

Introduction

An overwhelming body of scientific literature supports the concept that what each of us does on a daily basis significantly impacts on both our short- and long-term health and quality of life. This influence may be either positive or negative. Hundreds if not thousands of studies provide evidence that maintenance of a healthy weight, following sound nutritional practices, engaging in regular physical activity, not smoking cigarettes and other health-promoting practices all profoundly impact on health and quality of life. Nutrition, in particular, plays a prominent role in multiple aspects of positive life-style and good health.

Rippe Lifestyle Institute,

21 North Quinsigamond Avenue, Shrewsbury, MA 01545, USA e-mail: bgrady@rippelifestyle.com

J.M. Rippe, MD

In the past decade, many of these concepts have coalesced around the discipline of "lifestyle medicine." A major academic textbook of this title was published in 2013 (*Lifestyle Medicine*, CRC Press, 2013) [1], as well as an academic journal in this area (*American Journal of Lifestyle Medicine*; Sage Publishing) [2] and a two-volume *Encyclopedia of Lifestyle Medicine and Health* (Sage Publishing, 2012) [3] has also been generated. An academic professional organization has been established, the American College of Lifestyle Medicine (ACLM) [4]. This organization has doubled its membership annually for the past 4 years. A Council of the American Heart Association (AHA) that was previously named "The Council on Nutrition, Physical Activity and Metabolism," changed its name in 2013 to "The Council on Lifestyle and Cardiometabolic Health" [5]. The AHA has also launched an ambitious series of academic articles published in one of its official journals, *Circulation*, entitled, "Recent Advances in Preventive Cardiology and Lifestyle Medicine: A Themed Series [6]." In addition, a number of significant aspects of the Affordable Care Act focused on the important role that lifestyle practices play in achieving both cost containment and positive health outcomes. As evidence in this field continues to be developed it is clear that the field is going to be called "Lifestyle Medicine."

Perhaps no single area is more important to health-promoting practices than nutrition. In fact, guidelines and consensus statements from virtually every major professional medical organization contain significant emphasis on nutrition principles and practices as key components of the prevention and treatment of disease. Thus, nutrition continues to play a central role in both individual and population health considerations. This background provides the impetus for our belief that a textbook combining evidence-based nutritional information and the role that it can play in lifestyle medicine is a concept whose time has come.

The Challenge

While widespread emphasis and abundant knowledge exists concerning the interaction between sound nutrition and other lifestyle practices and health, it has been frustratingly difficult to improve nutritional practices in the American population. Consider the following:

- Over 2/3 of the adult population in the United States is either overweight or obese [7]. (This represents a staggering 40% increase over the past 20 years.)
- Less than 1/3 of the adult population in the United States consumes adequate servings of fruits and vegetables and follows simple evidence-based nutritional practices for good health [8].
- The prevalence of diabetes in the United States has doubled in the past 20 years [9].
- Over 1/3 of the adult population in the United States has high blood pressure [10, 11]. Yet less than 20% of individuals with high blood pressure follow the American Heart Association DASH diet recommended as an evidenced-based nutritional program for helping to lower blood pressure [12].
- Despite improvements over the past 20 years, cardiovascular disease (CVD) remains the leading killer of men and women in the United States, resulting in 37% of mortality each year [13]. CVD has multiple nutritional practices as a component of overall lifestyle choices.

Finding practical strategies and ways of implementing these strategies to help individuals make proper nutritional choices in their lives is an urgent mandate in the United States and around the world. Yet many individuals in the healthcare community do not have adequate skills to provide nutritional counseling and do not incorporate this into the regular practice of medicine. Despite the fact that the Affordable Care Act provides some reimbursement for nutritional counseling, very few healthcare practitioners or organizations are utilizing this provision.

The goal of *Nutrition in Lifestyle Medicine* is to provide up-to-date, evidence-based information concerning nutrition not only for the prevention and treatment of disease but also as a key component of an overall healthy lifestyle. In addition to providing information, this book is intended to provide practical strategies to incorporate healthy nutritional practices into the daily lives of children and adults.

A particular emphasis of *Nutrition in Lifestyle Medicine* will be nutritional considerations related to obesity, diabetes, and cardiovascular disease. These three metabolically based diseases are so closely aligned that it has been suggested that they could be lumped together in a condition some have called "cardiodiabesity."

In addition to an emphasis on these three disease processes, *Nutrition in Lifestyle Medicine* will also focus on a variety of specialized areas such as nutrition for athletes and physically active individuals, hydration, and nutrition throughout the life cycle spanning from children to individuals over the age of 60. Chapters are also included on controversies in nutrition such as sugars and health and saturated fats. Specialized chapters will also be included in such diverse areas as promoting nutrition in men's health, nutrition for a healthy pregnancy, and nutritional considerations for Hispanics.

Since considerations of nutrition and health do not take place in a vacuum, a whole section is devoted to public health and public policy issues such as promoting an environment to support healthy eating and issues relating to food safety. Some practical issues related to frozen foods as well as school lunch programs are included as well as issues related to current or future research needs and opportunities in nutrition such as nutrigenomics and how to evaluate nutrition research. The overarching goal in all of these chapters will be to provide evidence-based nutrition information.

The Opportunity

Improved nutritional practices can play a very significant role in decreasing risk factors for leading causes of mortality. It is important to remember that seven of the ten leading risk factors for mortality in developed in countries are related to diet and physical activity practices [14], including the following:

- High blood pressure
- Overweight and obesity
- High blood glucose levels
- · Physical inactivity
- High cholesterol levels
- Low fruit and vegetable intake
- Alcohol use

Moreover, individuals who follow a cluster of healthy lifestyle practices can substantially decrease their risk of major metabolic diseases. Consider the following:

- According to the Nurses' Health study, 74% of cardiovascular disease (CVD), 82% of coronary heart disease (CHD), and 91% of diabetes in women could be prevented by participation in five lifestyle behaviors: not smoking, engaging in regular physical activity, maintaining healthy weight, eating healthier food, and moderate alcohol intake [15, 16].
- According to the Health Professionals Follow-Up Study, which involved 50,000 men between the ages of 40 and 75 years, individuals who have 1,2,3,4, or 5 of the same healthy behaviors as noted in the Nurses' Health Study had respectively a 54, 63, 71, 78, and 87% lower risk of CVD, CHD, and diabetes, respectively, compared to men with no healthy behaviors [17].
- Unfortunately, in US women (35–54 years old) the coronary death rate has shifted from a decline of 5.4% per year in the period of 1980 through 1989 to an increase of 1.5% per year in 2000–2002 [15, 16]. Declines in men in the same age rate had been 6.2% per year 1980 through 1989 but only 0.5% in 2000–2002 [17].
- In 2010, the American Heart Association in their strategic plan for the year 2020 estimated that the prevalence of ideal cardiovascular health (a similar cluster of behaviors as listed as above) was present in only 5% of US adults [18].

Thus, there is an enormous opportunity through behaviors such as improved nutrition to reduce major cardiometabolic diseases.

Widespread Consensus

There is widespread consensus among numerous scientific organizations and their published guidelines about the important role of positive nutritional habits in either prevention or treatment of disease. The major authoritative guidelines incorporating an important role for sound nutritional practices include the following:

- Dietary Guidelines for Americans Advisory Committee Report 2015 [19]
- Dietary Guidelines for Americans 2010 [20]
- National Cholesterol Education Program [21]
- JNC VII [10] and VIII [11]
- Guidelines for Prevention and Management of Hypertension [12]
- Institute of Medicine Guidelines for the Management of Obesity [22]

In addition, virtually every scientific body that deals with metabolic disease has recommended sound nutrition as a cornerstone for prevention and treatment of various diseases. A listing of some of these guidelines is found in Table 1.1 [13, 18, 23–31]. Thus, the role of nutrition in positive lifestyle is built on a broad consensus of scientific statements and authoritative guidelines.

Translating Guidelines Into Individual Behavior

While a wide consensus has emerged about the importance of sound nutritional habits in both preventing and treating disease, a major challenge remains in how to translate these guidelines into specific ways to help individual behavior. The guidelines for multiple organizations are generally consistent with each other.

Despite the consistency of the recommendations, which go back over 30 years, Americans have been slow to change their habits and practices. For example, in 2011 over 2/3 of American adults were overweight or obese [7]. In 2010, only 34.4% of US adults aged 18 years or older engaged in self-reported leisure time physical activity [18]. Numerous studies report that only 25–30% of US adults consume the recommended servings of fruits and vegetables [8]. Thus, moving from the knowledge base that is summarized in numerous guidelines and from multiple professional organizations, the challenge remains how to translate these guidelines particularly related to nutrition into helping people implement healthier habits in their daily lives.

Table 1.1 Guidelines from	m various	scientific	organizations
-----------------------------------	-----------	------------	---------------

AHA Guidelines for the Prevention and Management of Coronary Artery Disease

AHA and AAP Guidelines for Prevention and Treatment of Metabolic Syndrome

AHA and American Cancer Society Joint Statement on prevention of heart disease and cancer

AHA Nutrition Implementation Guidelines

AHA 2020 Strategic Impact Goals

Guidelines from the American Diabetes Association for the Management of Diabetes

American Academy of Pediatrics Guidelines for Prevention and Treatment of Childhood Obesity

American Academy of Pediatrics for Heart Disease Risk Factor Reduction in Children

Strategies to help individuals change habits to healthier ones must deal with the fact that lifestyle behaviors are complex and influenced by many factors. Scientific evidence has identified domains of influence related to both nutrition and physical activity, including the following:

- Individual
- Social/family and close peer environment
- Community environments/school, workplace, restaurants, neighborhood (e.g., access to healthful foods and built environment)
- Macro public environment/public policy, corporate policy and marketing transportation, popular media/communications, and economic factors

Strategies for influencing individual behavior must take this complexity into account. The complexity of influences regarding how to effectively change individual behaviors was a central premise of an article by Giddings et al., entitled, "Implementing American Heart Association Pediatric and Adult Nutrition Guidelines" [24]. In this article, Giddings et al. provided considerable detail in each of the domains that interact and influence individual food choices.

• Individual Influences [24, 32–35]

Giddings et al. divided individual influences into seven interactive domains:

- Convenience:

Including available time, ease of preparation, cooking skills, and whether or not meals were consumed outside the home.

- Eating patterns and social factors
 These include food patterns whether or not food was eaten with each other, whether or not meals are skipped, snacking, and whether or not weekend patterns differ from weekday patterns.
- Psychological factors Including mood and distraction.
- Need:

This domain includes issues related to hunger and satiety as well as overall health awareness.

- Taste:

Children prefer sweet-tasting items and palatability and also tend to favor foods that are energy dense and salty; variety sustains interest.

- Knowledge/ignorance of helpful recommendations:

Further influences may include whether or not individuals are aware of what constitutes healthful eating. In this area, area barriers may include literacy, interest, messages from the media, and an unrealistic view of what constitutes a portion.

Cost and access:

The final individual domain relates to the reality that many individuals face that energy-dense foods cost less per 1000 kcals than nutrient-dense foods. Thus, an individual's economic status could significantly impact on their food choices. Furthermore, availability of nutrient-dense foods in local stores maybe a very significant factor as well as what is actually found in an individual's pantry or refrigerator.

• Family Food Influences [25, 36–42]

Interacting strongly with the factors that influence an individual with regard to food patterns is the effect that the family environment has on these matters. Giddings et al. divide the family environment into six major domains:

Economic factors:

The impact that family income has on food choices is significant. Moreover, the socioeconomic status of the neighborhood can also influence food choices.

- Parental modeling:

The food choices made by parents have been demonstrated to strongly influence children. Mothers and children have similar patterns of food acceptance. The child is more likely to try a strange food if it is eaten by the mother or other parent.

– Family eating patterns:

It has been demonstrated by multiple research studies that a family eating meals together is associated with better food choices and improved nutrient adequacy. Snacking and weekend eating patterns may also impact on food choices as will meal skipping, frequency of eating away from the home, and cultural food patterns.

- Family "Nutritional Gate Keeper":

The individual who typically buys and prepares food will have a great impact on food choices for the whole family. This individual's knowledge, skill, attitudes, and so on will have a large impact on the foods available at the home.

- Physical activity modeling:

The level of exercise and physical activity demonstrated by the parents has been clearly associated with the activity levels of children.

- Parental children feeding practices:

The way food is consumed within the family including whether or not there is food restriction or control, impacts on food choices. Also the type of diet that parents model when improving their own diets will significantly impact on children.

• Community Food Influence [25, 43–45]

The third domain of influencing individual food choices comes from the community. Giddings et al. list five domains within the community environment that impact on individual food choices:

Economic factors:

The cost of obtaining food from local sources strongly impacts on individual food choices as does the neighborhood socioeconomic status, which plays a major role in whether or not adequate grocery stores are available. Numerous studies have identified "food islands" where there are no large grocery stores available to provide fresh produce and other nutritious foods within certain lower socioeconomic neighborhoods.

Work Food environment:

The food that is available at work or in surrounding neighborhood impacts on food choices an individual will make. For example, if a company an individual works at supports health programs and whether or not healthy food choices are available will often impact food choices.

- School environment:

Whether or not there are school breakfast and lunch programs available as well as menu items available within the school lunch program strongly interact with children's eating habits as does the availability of energy-dense less-nutritious "competitive" foods in vending machines, nearby stores, and so on. This issue is so important that we have devoted a separate chapter to school lunch programs in this book.

Food availability:

Accessibility of supermarkets versus convenience stores is important, so is the prevalence of fast food and quick service restaurants versus full-service restaurants. It should be noted that evidence that fast food or quick service restaurants have a negative impact on nutritional choices is mixed.

- Peer modeling:

The intake patterns of peers that one eats with regularly at school or work or in other settings impacts on food choices.

- Built environment:

Whether or not the environment encourages or discourages activities such as places to walk and whether or not supermarkets versus convenient stores are available, and so on, also represent significant influences on individual food choices.

• Macro Public Environment [25, 46–48]

Overarching individual family and community food choices in the overall public policy environment related to food are important. Giddings et al. divided this domain of influence into six categories including the following:

- Public policy:

This includes agricultural policies, trade policies such as quotas, tariffs, and so on. In addition, public policy documents such as the Dietary Guidelines for Americans, Physical Activity Guidelines for Americans, and various prevention and treatment guidelines by scientific bodies already outlined in this chapter impact food choices.

- Economic factors:

Housing patterns, local food costs, and economic status of the neighborhood all impact on food choices.

- Food marketing:

Enormous amounts of money are spent every year on marketing food including advertising, instore displays, celebrity endorsements, and on-package offers and health claims, all of which can impact on nutritional choices.

Corporate policy and practice:

The design and manufacturing as well as pricing of foods impact on food choices as do multiple aspects related to food retailers such as the location of supermarkets, convenience stores, and restaurants.

- Cultural norms and values:
 Cultural factors such as body image strongly impact food choices.
- Transportation:

The cost to transport and store food significantly impact both nationally and globally on access to healthful and affordable food.

- Communication/Media:

Nonmarketing food stories (e.g., Internet blogs) and presentations can also impact on individual food choices.

Strategies Emphasizing Implementation

Given the complexity of influences on eating behaviors, it is incumbent upon healthcare professionals to develop effective strategies emphasizing how nutritional guidelines can be implemented. In our research group we call this moving from "what" to "how." In this area there are some key concepts to keep in mind.

Considerable evidence suggests that addressing patterns of eating behavior, not just one element, is essential. A key concept of both the Dietary Guidelines 2010 [20] and 2015 [19, 49] is to emphasize patterns of eating and the importance of physical activity, in addition to recommending specific types of foods and nutrients. Moreover, the prevention and treatment guidelines for chronic diseases typically recommend a cluster of interrelated behaviors, including not only diet but also physical activity [50], weight management, and smoking cessation.

Successful behavioral change models, whether they are in nutrition or other daily habits and actions, increasingly draw from behavioral medicine models and psychological theories and models that address variables and patterns of individual behavior. Such models can be used to identify and examine strategies to overcome barriers and choose appropriate interventions while accessing outcomes. While a detailed description of behavioral medicine models is beyond the scope of this chapter, it has been extensively viewed elsewhere [51–53]. The leading models that share many constructs or ideas with each other include the following:

- Health belief model
- · Theory of reason action/planned behavior
- Social cognitive theory
- Trans-theatrical model and socioecological model

Throughout this book we have challenged authors to not only provide state-of-the-art evidence but also bridge that evidence to effective models for how to implement the desired nutritional changes. These models will typically address the four domains of influence on individual feeding behaviors outlined in this chapter, namely individual, family, community, and macro public policy.

Summary/Conclusions

We are entering an era where the importance of nutrition and good health is increasingly being understood by all segments of the healthcare community. Moving forward it will be essential to not only apply state-of-the-art evidence but also develop effective strategies for helping individuals implement nutritional guidelines in their daily lives. We hope that *Nutrition in Lifestyle Medicine* will play a role in helping all healthcare professionals improve the health of their clients by applying the principles outlined throughout this book.

References

- 1. Rippe JM. Lifestyle medicine. 2nd ed. Boca Raton: CRC Press; 2013.
- 2. Rippe JM, editor. American Journal of Lifestyle Medicine (AJLM). SAGE Publications.
- 3. Rippe J. Encyclopedia of lifestyle medicine and health. Thousand Oaks: SAGE Publications; 2011.
- 4. American College of Lifestyle Medicine (ACLM). 2016. http://www.lifestylemedicine.org/accessed.
- American Heart Association. Council on lifestyle and cardiometabolic health. http://my.americanheart.org/professional/Councils/NPAM/Council-on-Lifestyle-and-Cardiometabolic-Health_UCM_322856_SubHomePage.jsp. Accessed 6 Jan 2016.
- Franklin BA, Cushman M. Recent advances in preventive cardiology and lifestyle medicine: a themed series. Circulation. 2011;123(20):2274–83.
- Flegal KM, Carroll MD, Kit BK, et al. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. JAMA. 2012;307:491–7.
- Schiller JS, Lucas JW, Ward BW, et al. Summary health statistics for U.S. adults: national health interview survey, 2010. Vital Health Stat 10. 2012;252:1–207. Epub 2012/07/28.
- 9. Shulman GI. Ectopic fat in insulin resistance, dyslipidemia, and cardiometabolic disease. N Engl J Med. 2014;371(12):1131–41.
- Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. JAMA. 2003;289(19):2560–72.
- James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). JAMA. 2014;311(5):507–20.
- Appel LJ, Brands MW, Daniels SR, et al. Dietary approaches to prevent and treat hypertension: a scientific statement from the American Heart Association. Hypertension. 2006;47(2):296–308.

- 13. Lloyd-Jones D, Adams RJ, Brown TM, et al. Executive summary: heart disease and stroke statistics--2010 update: a report from the American Heart Association. Circulation. 2010;121(7):948–54. Epub 2010/02/24.
- World Health Statistics. 2009. World Health Organization http://www.who.int/gho/publications/world_health_statistics/EN_WHS09_Full.pdf. Accessed 1 Apr 16.
- Stampfer MJ, Hu FB, Manson JE, et al. Primary prevention of coronary heart disease in women through diet and lifestyle. N Engl J Med. 2000;343(1):16–22.
- Bassuk S, Manson J. Lifestyle and risk of cardiovascular disease and type 2 diabetes in women: a review of the epidemiologic evidence. Am J Lifestyle Med. 2008;2(3):191–213.
- Chiuve SE, McCullough ML, Sacks FM, et al. Healthy lifestyle factors in the primary prevention of coronary heart disease among men: benefits among users and nonusers of lipid-lowering and antihypertensive medications. Circulation. 2006;114(2):160–7.
- Lloyd-Jones DM, Hong Y, Labarthe D, et al. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic Impact Goal through 2020 and beyond. Circulation. 2010;121(4):586–613.
- 19. USDA, Scientific Report of the 2015 Dietary Guidelines Advisory Committee, Advisory Report to the Secretary of Health and Human Services and the Secretary of Agriculture. 2015.
- Center for Nutrition Policy and Promotion. Report of the dietary guidelines advisory committee on the dietary guidelines for Americans. US Department of Agriculture, Washington DC. 2010. http://www.nutriwatch. org/05Guidelines/dga_advisory_2010.pdf.
- National Cholesterol Education Program. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA. 2001;285:2486–97.
- 22. Glickman D, Parker L, Sim L, et al. Accelerating progress in obesity prevention solving the weight of the nation. Washington, DC: Institute of Medicine; 2012.
- Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update: consensus panel guide to comprehensive risk reduction for adult patients without coronary or other atherosclerotic vascular diseases. Circulation. 2002;106(3):388–91.
- 24. Gidding SS, Lichtenstein AH, Faith MS, et al. Implementing American Heart Association pediatric and adult nutrition guidelines: a scientific statement from the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular Disease in the Young, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council for High Blood Pressure Research. Circulation. 2009;119:1161–75.
- 25. American Diabetes Association. Are you at risk? http://www.diabetes.org/are-you-at-risk/. Accessed 20 Jan 2016.
- 26. Daniels SR, Hassink SG. The role of the pediatrician in primary prevention of obesity. Pediatrics. 2015;136(1):e275–92.
- Daniels SR, Pratt CA, Hayman LL. Reduction of risk for cardiovascular disease in children and adolescents. Circulation. 2011;124(15):1673–86.
- 28. Steinberger J, Daniels SR, Eckel RH, et al. Progress and challenges in metabolic syndrome in children and adolescents: a scientific statement from the American Heart Association Atherosclerosis, Hypertension, and Obesity in the Young Committee of the Council on Cardiovascular Disease in the Young; Council on Cardiovascular Nursing; and Council on Nutrition, Physical Activity, and Metabolism. Circulation. 2009;119(4):628–47.
- Eyre H, Kahn R, Robertson R. ACS/ADA/AHA scientific statement: preventing cancer, cardiovascular disease and diabetes. Circulation. 2004;109:3244–55.
- Rosenzweig JL, Ferrannini E, Grundy SM, et al. Primary prevention of cardiovascular disease and type 2 diabetes in patients at metabolic risk: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2008;93(10):3671–89.
- Position of the American Dietetic Association. Total diet approach to communicating food and nutrition information. J Am Diet Assoc. 2007;107(7):1224–32.
- Larson N, Story M. A review of environmental influences on food choices. Ann Behav Med Publ Soc Behav Med. 2009;38 Suppl 1:S56–73.
- 33. Brian Wansink. Marketing nutrition: soy, functional foods, biotechnology and obesity. Urbana and Chicago: University of Illinois Press; 2005. p. 86–7. 2) Dr. Wansink's presentation to DGAC 2010. www.dietary.guidelines.gov.
- Henson S, Blandon J, Cranfield J, et al. Understanding the propensity of consumers to comply with dietary guidelines directed at heart health. Appetite. 2010;54:52–613.
- 35. Power TG, Bindler RC, Goetz S, et al. Obesity prevention in early adolescence: student, parent, and teacher views. J Sch Health. 2010;80:13–193.
- 36. Neumark-Sztainer D, Larson NI, Fulkerson JA, et al. Family meals and adolescents: what have we learned from Project EAT (Eating Among Teens)? Public Health Nutr. 2010;13(7):1113–21.
- Scaglioni S, Salvioni M, Galimberti C. Influence of parental attitudes in the development of children eating behaviour. Br J Nutr. 2008;99 Suppl 1:S22–5. Epub 2008/04/09.

- 38. Pearson N, Biddle SJ, Gorely T. Family correlates of fruit and vegetable consumption in children and adolescents: a systematic review. Public Health Nutr. 2009;12(2):267–83. Epub 2008/06/19.
- Burgess-Champoux TL, Larson N, Neumark-Sztainer D, et al. Are family meal patterns associated with overall diet quality during the transition from early to middle adolescence? J Nutr Educ Behav. 2009;41:79–86.
- 40. Clark HR, Goyder E, Bissell P, et al. How do parents' child-feeding behaviours influence child weight? Implications for childhood obesity policy.J. Public Health (Oxf). 2007;29:132–41.
- 41. de Lauzon-Guillain B, Musher-Eizenman D, Leporc E, et al. Parental feeding practices in the united states and in france: relationships with child's characteristics and parent's eating behavior. J Am Diet Assoc. 2009;109:1064–9.
- 42. Benton D. Role of parents in the determination of the food preferences of children and the development of obesity. Int J Obesity Relat Metab Disord J Int Assoc Study Obesity. 2004;28:858–69.
- Tillotson JE. America's obesity: conflicting public policies, industrial economic development, and unintended human consequences. Annu Rev Nutr. 2004;24:617–43.
- 44. Harris JL, Pomeranz JL, Lobstein T, et al. A crisis in the marketplace: how food marketing contributes to childhood obesity and what can be done. Annu Rev Public Health. 2009;30:211–25.
- Harris JL, Brownell KD, Bargh JA. The food marketing defense model: integrating psychological research to protect youth and inform public policy. Soc Issues Policy Rev. 2009;3:211–71.
- 46. Terry-McElrath YM, O'Malley PM, Delva J, et al. The school food environment and student body mass index and food consumption: 2004 to 2007 national data. J Adolesc Health Off Publ Soc Adolesc Med. 2009;45:S45–56.
- Bodor JN, Rose D, Farley TA, et al. Neighbourhood fruit and vegetable availability and consumption: the role of small food stores in an urban environment. Public Health Nutr. 2008;11:413–20.
- Mendoza JA, Watson K, Cullen KW. Change in dietary energy density after implementation of the Texas Public School Nutrition Policy. J Am Diet Assoc. 2010;110(3):434–40.
- 49. U.S. Department of Health and Human Services, U.S. Department of Agriculture. 2015 2020 dietary guidelines for Americans. 8th ed. 2015. Available at: http://health.gov/dietaryguidelines/2015/guidelines/. Epub Dec 2015.
- Nigg CR, Rhodes R, Amato KR. Determinants of physical activity: research to application. In: Rippe J, editor. Lifestyle medicine. 2nd ed. Boca Raton, FL: CRC Press; 2012. p. 1435.
- Linke S, Pekmezi D. Applying psychological theories to promote healthy lifestyles. In: Rippe J, editor. Lifestyle medicine. 2nd ed. Boca Raton, FL: CRC Press; 2012. p. 223.
- 52. Di Noia J, Prochaska JO. Dietary stages of change and decisional balance: a meta-analytic review. Am J Health Behav. 2010;34(5):618–32.
- 53. Jansink R, Braspenning J, van der Weijden T, et al. Nurse-led motivational interviewing to change the lifestyle of patients with type 2 diabetes (MILD-project): protocol for a cluster, randomized, controlled trial on implementing lifestyle recommendations. BMC Health Serv Res. 2009;9:19. Epub 2009/02/03.

Chapter 2 Nutrition 101: The Concept of Nutritional Status, Standards, and Guides for Nutrient Intakes, Eating Patterns, and Nutrition

Johanna T. Dwyer and Regan Lucas Bailey

Key Points

- Nutritional status is assessed by a combination of dietary intake, biochemical and anthropometric indices, and clinical observations. Dietary status, including only intake, is less definitive.
- The Dietary Reference Intakes are dietary standards for the United States and Canada
- The Dietary Guidelines for Americans 2015–2020 provide food-based recommendations for dietary patterns. The Nutrient Facts and Supplement Facts labels describe the composition of foods and supplements, and various types of health claims for food further provide information useful to consumers.
- The Healthy Eating Index is a tool for roughly evaluating how well patterns fit with the Dietary Guidelines

Keywords Dietary intake • Dietary status • Nutritional status • Dietary guidelines • Dietary reference intakes • Healthy eating index

Abbreviations

AI	Adequate intake
AMDR	Acceptable macronutrient distribution ranges
CV	Coefficient of variation

Johanna T. Dwyer DSc, RD (🖂)

Office of Dietary Supplements, National Institutes of Health, 6100 Executive Blvd, Bethesda, MD, USA dwyerj1@od.nih.gov

School of Medicine and Friedman, School of Nutrition Science and Policy, Tufts University, Boston, MA, USA

Jean Mayer, US Department of Agriculture, Human Nutrition Center on Aging, Tufts University, Boston, MA, USA

Frances Stern Nutrition Center, Tufts Medical Center, Boston, MA, USA

Regan Lucas Bailey PhD, MPH, RD, CPH Office of Dietary Supplements, National Institutes of Health, 6100 Executive Blvd, Bethesda, MD, USA

Department of Nutrition Science, Purdue University, West Lafayette, IN, USA e-mail: bailey@mail.nig.gov

DGAC	Dietary Guidelines Advisory Committee
DV	Daily value
DRI	Dietary reference intakes
EAR	Estimated average requirement
HHS	US Department of Health and Human Services
NHANES	National Health and Nutrition Examination survey
RDA	Recommended dietary allowance
SD	Standard deviation
USDA	US Department of Agriculture
USRDA	US recommended dietary allowance

Introduction

This chapter provides an overview of tools for evaluating dietary intakes of individuals and provides dietary recommendations. Such recommendations have been given by government and other groups for over 100 years (Fig. 2.1). The chapter describes the concepts of dietary status, nutritional status, and the available methods for measuring them. The Dietary Reference Intakes (DRI), the standards for nutrient intakes in the United States and Canada, are described and their multiple uses are discussed. Major federal guidance related to nutrition is outlined. The Dietary Guidelines for Americans, and recommendations to help guide Americans in altering their current intakes in more healthful directions are summarized. MyPlate (formerly MyPyramid and the Food Guide Pyramid), a guide that provides food-based recommendations based on the Dietary Guidelines for Americans and the DRI, is described. The Healthy Eating Index (HEI), a simple scoring system for of evaluating overall dietary quality including balance, variety, and adequacy of intakes based on the DRI and the Dietary Guidelines is also discussed. Healthy People 2020, a set of national goals for promoting health and

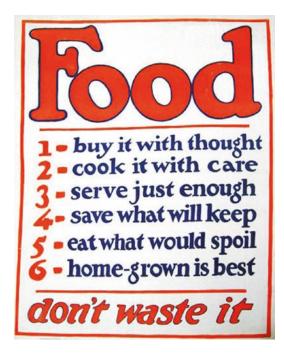


Fig. 2.1 Poster with dietary recommendations world war I

preventing disease that involve nutrition, is briefly mentioned. Finally, a new National Nutrition Research Roadmap for Federal human nutrition research 2016–2021 is briefly discussed.

The Concept of Nutritional Status and Its Measurement

Nutritional status is a component of health status that represents the bodily state resulting from the intake, absorption, utilization, and metabolism of dietary intake. It has an impact on all nutrition-related facets of health including growth, development, immunity, and risk of diet-related disease. Nutritional status is assessed by a combination of anthropometric, biochemical, clinical, and dietary measures. Dietary intake is one essential component of nutritional status. Without knowledge of an individual's intake, it is not possible to assess the causes of deficiency, imbalance, or excess, nor can appropriate interventions be crafted.

Adverse or suboptimal nutritional status is referred to as malnutrition. Malnutrition can be multifactorial, due to alterations in dietary intake, absorption, metabolism, or excretion (Table 2.1). Moreover, several types of malnutrition may exist at the same time in the same person. For example, a frail elderly person may suffer from protein calorie malnutrition and vitamin D deficiency simultaneously. Several measures of malnutrition are necessary to assess nutritional status. Nutritional status assessment usually includes anthropometric, biochemical, clinical, and dietary measures as well as functional measurements such as the activities of daily living. Because each method has its limitations, using multiple measures in conjunction with one another can best characterize the type and severity of malnutrition. Nutritional status assessment also considers the individual's metabolically relevant health characteristics, eating habits, cultural practices, and quality of life related to food.

Table 2.1 shows the various forms of malnutrition, which include deficiency disorders as well as problems with excesses and imbalances of nutrients. Table 2.2 summarizes why multiple indicators of nutritional status are needed. Nutritional assessment includes an assessment of dietary intake, and, if malnutrition is present, its form, stage of development, and severity of the problem.

There is a continuum of changes in the human body in response to malnutrition. The clinical signs of malnutrition, its effects on anthropometric measures such as weight and muscle mass, and on functions such as the activities of daily living and quality of life, are the end result of a pathological process that starts much earlier. This earlier preclinical phase of malnutrition can often be identified by the use of dietary measures, biochemical and hematological biomarkers in blood, urine, sweat, or other bodily secretions, hair, and nails, as well as by pathology in individual cells, tissues, and organ systems. If diet is the cause of the malnutrition, food intake alterations are usually apparent for days, weeks, or months before metabolism is altered and changes in biochemical measurements of nutritional status are present. Therefore, to prevent malnutrition and to do it as early as possible, dietary assessment is worthwhile, since it may provide an early warning of problems to come.

Dietary Status

This chapter focuses primarily on partially measuring nutritional status through dietary status. Dietary status involves the measurement of an individual's usual intake of nutrients, or usual consumption of foods, beverages, and nutrient-containing supplements, food groups, or food patterns. Dietary status and nutritional status are dissimilar, because food consumption is not the only factor involved in determining if dietary intakes are sufficient to maintain nutritional status and health. Therefore, it is important to remember that diet gives only a partial and incomplete but nevertheless useful glimpse at an individual's nutritional status.

Form and cause of malnutrition	Clinical terms to describe it	Comments
Dehydration: inadequate fluid intake to meet bodily needs	Dehydration	Often occurs secondary to fever, exertion, very warm dry climate, or because of diets with high solute loads or drugs that have diuretic effects
<i>Starvation</i> : virtually totally inadequate intakes of all nutrients	Marasmus, emaciation, cachexia	Occurs with prolonged fasting; withholding of fluids worsens its effects
Protein-calorie malnutrition	Kwashiorkor, protein calorie malnutrition	Often occurs secondary to disease and infection, probably via cytokine mediated responses to acute infection or trauma; examples include HIV/AIDS and Sarcopenia due to inadequate intake of protein and/or cytokine-mediated responses to insults
Vitamin, mineral, or other specific nutrient deficiencies	Pellagra (niacin/tryptophan), scurvy (ascorbic acid deficiency), rickets and osteomalacia (vitamin D deficiency in children and adults, respectively), iron deficiency anemia (iron deficiency), nutritional anemia (iron, vitamin B-6, folic acid, or vitamin B-12 deficiency), essential fatty acid deficiency	These deficiencies often occur secondarily to inadequate food intake or inadequate dietary quality. May also occur as conditioned deficiencies secondary to disease
Imbalances: increased diet-related chronic disease risk factors due to imbalances of nutrients	Excess of saturated fat, cholesterol and other atherogenic and thrombogenic dietary lipids (hyperlipidemias and perhaps altered clotting factors), excess of salt and /or sodium (blood pressure risk factors)	Imbalances or excesses of energy- yielding nutrients or related substances may give rise to metabolic aberrations and increase risks of ill health, especially in those with certain genetic profiles
Obesity: excess food energy intake and/or insufficient energy output	Excess food energy regardless of source gives rise to obesity and overweight	Physical inactivity may increase the likelihood of excess energy intakes
Alcohol excess	Alcoholism, problem drinking	At very high levels of alcohol intake, all persons develop physical signs of chronic disease; at lower levels of intake some individuals are particularly susceptible
Excess of other specific nutrients (vitamins, minerals, others)	Specific toxicities vary: hypervitaminosis A (vitamin A), hypervitaminosis D (vitamin D), fluorosis (fluoride), etc.	Intakes that exceed the upper level of the dietary reference intakes generally increase risk of compromising one or more functions. The possible functions vary from nutrient to nutrient
Toxicity: excesses of other constituents in food, drink, or supplements	Names vary depending on substance; lead poisoning, lathyrism, etc.	Many substances other than nutrients in food and supplements may cause illness
Food-borne disease	Food poisoning or food intoxication: salmonellosis, botulism, staphylococcal food poisoning, others. Parasites such as beef tapeworm may cause problems. Prions or viruses as in Bovine Spongiform Encephalopathy (BSE) may also cause illness.	Food is the carrier for a microorganism, virus, or parasite

 Table 2.1
 Forms of malnutrition and clinical terms used to describe them

Table 2.2	Why mult	ple indicators	of nutritional	status are needed	to diagnose malnutrition

Multiple forms of malnutrition exist See Table 2.1 for some examples *The causes of malnutrition vary* Some are due to deficient quality or quantity of diet alone (primary malnutrition) but most are secondary to disease, social or psychological problems, and may occur even in the face of adequate and appropriate food in the environment. Many different pieces of information are necessary to arrive at an understanding of how these causes interact

No single indicator for all the forms of malnutrition exists

The most sensitive, least costly, and most specific indicators of malnutrition vary from nutrient to nutrient

Even for a specific form of malnutrition, indicators vary with respect to how sensitive, specific, valid, and reliable they are

Severity of malnutrition varies

Milder forms require different and more sensitive indicators (e.g., measures of tissue or blood stores) than the more severe (which may be evident with anthropometric or clinical measures alone)

The rationale for measuring dietary intake and status is that food consumption is one factor affecting nutritional status that can be altered. The most important aspects of dietary intake are the amount of food, the forms in which nutrients are present in the diet (e.g., foods, beverages, nutrient-containing dietary supplements, or medications that may affect absorption or contain nutrients), and the presence of other nonnutrient bioactive constituents in food that may have beneficial or harmful effects on health.

The usual goal of assessing diet is to obtain an estimate of usual, habitual intake, either by directly observing and measuring diet or indirectly by report. Because most health effects depend on usual, habitual intake, it is important that the dietary report include multiple measurements over a sufficiently long period of time (e.g., many days or weeks) to be representative of usual intake. Usual intake is important since the effects of diet on nutritional status are most closely linked to it; minor variations from day to day or week to week are not sufficient to perturb it. For individuals, only representative intakes over relatively long periods are meaningful for assessing the effects of diet on nutritional status or the associations between diet, health, and disease. The one exception is the type of very recent intake information that is usually collected when incidents of food-borne illness or other toxicities are being traced. Then, foods and beverages eaten in the hours or days before the poisoning accident, details on food preparation, and other factors that may have caused the contamination are investigated and may provide evidence of the causative agent. For large groups of people, such as the population of the United States, it is possible to obtain reasonable estimates of the groups' usual intakes by sampling intakes of representative individuals on two or more non-successive days and then using the estimates of variability that emerge as an approximation of usual intakes.

Methods Used to Assess Dietary Intakes

A comprehensive review of assessment methods for dietary intakes is provided elsewhere [1]; so only a brief summary is provided here. Diet can be assessed on a short- or long-term basis. In the short term, instruments capture data on recent dietary intakes (e.g., a few days) whereas long-term instruments aim to capture dietary data over longer period of time. The time frame for longer-term dietary assessment typically ranges from 30 days to 1 year. Food records and 24 h dietary recalls are the most common short-term dietary assessment methods. A food record is a detailed list of all foods and beverages consumed within a specified period of time. For food records, it is best if foods and beverages can be weighed or measured by the individual who eats them since it is easy to underestimate portions

and amounts, and people tend to forget what was eaten. Multiple food records are needed to get an accurate picture of dietary intake (exposure); however, multiple days add to participant burden and can compromise the quality of the data obtained [2]. A 24 h recall assesses an individual's intake over the previous 24 h. In contrast to the food record, the recall is usually performed by an interviewer. Web-based, self-completed 24 h recalls are emerging as well. Probing questions and multiple pass methods are often used to enhance the quality of the 24 h recall and to minimize underestimation and forgetting [3]. As with food records, recalls are subject to high day-to-day variability: this influences the micronutrient content of the intake reports more than for the macronutrients, because macronutrients are consumed more consistently and are therefore more stable than those for most micronutrients, which vary more from food to food [4]. Several factors are also known to influence intake reports, including the day of the week, mode of interview (telephone or in-person), the sequence of the recall (e.g., first, second, and so on), and whether nutrient-containing dietary supplements are queried or not.

The food frequency questionnaire (FFQ) is a longer-term recall instrument and is often used in large-cohort or case-control studies. FFQs assess intake over a specified period of time and query how frequently a person consumes multiple food items that are usually aggregated into food groups with similar nutrient profiles. FFQs can be quantitative, semi-quantitative (SQFF), or qualitative [5]. FFQs offer a more cost-effective alternative to the 24 h recall. The most common tools used today are the SQFF questionnaires, which are frequently used in large studies. However, the FFQ limits the scope of foods that can be queried. The FFQ may create participant burden, and it may be difficult or confusing to complete, in part because the pattern of frequency of intakes (once or more times a day, week, month or less frequently) is an abstract concept and difficult to remember, and even people who have the same frequency of consumption of foods fill out the questionnaire differently. The reported intakes make little sense unless they are "calorie adjusted" based on usual energy intakes of individuals similar to that of the respondent. The FFQ requires literacy and physical ability to complete since it is usually self-administered. Most importantly, the accuracy of nutrient profiles determined by FFQs has been questioned. The SQFF questionnaire was designed originally to rank individuals' intakes into quartiles or quintiles, but in recent years it has been incorrectly used to provide precise quantitative estimates of intakes of individuals, which they cannot do. The use of multiple 24 h recalls has been shown to be a less biased estimator of calories and protein from foods in adults than an SQFF-based instrument [6].

Dietary supplement use can be measured using the same techniques as dietary assessment of foods: a record or diary, 24 h recalls, or a frequency-based instrument. No validation studies exist to compare the different methods for assessment of supplement use. However, since dietary supplement use can be habitual (daily) or episodic (contextual), it may be helpful to also use a frequency-based question-naire to obtain a longer time period to capture whether specific types of supplements are used or not. Ideally, both 24 h recall and a frequency questionnaire should be used together.

Dietary recommendations are intended to be met over time [7]. Daily intakes can vary and nutrients can be stored for varying lengths of time in the body; therefore, it is unnecessary to achieve nutrient recommendations every day [7]. The human body does not require specific foods; dietary recommendations for foods are designed to assist individuals to achieve recommended levels of nutrients, not because the foods themselves are essential. Therefore, there are many patterns of food intake that are acceptable from the standpoint of achieving satisfactory dietary and nutritional status.

The concept of usual nutrient intake is salient in public health where the interest is most often in capturing long-run usual intakes as opposed to intake on a given day (i.e., a 24 h recall). Many procedures have been described to adjust the dietary estimates from 24 h recalls to reduce within-person variation and to produce usual intakes [8–12]. To produce total usual intakes, it is recommended to first adjust the dietary estimates using one of these methods and then to add the daily estimates from dietary supplements to the usual dietary intakes [13].

Limitations of Dietary Assessment

Although the dietary intake methods all have their limitations, they do provide a picture of food and nutrient consumption that may be useful for purposes of assessing what has been eaten and planning intakes. The major shortcoming of most dietary assessment methods is that while they furnish a general profile of nutrient intakes, they are less accurate in providing information on the calories, nutrients, or nonnutrient bioactives that are consumed. Most dietary assessment methods depend heavily on the eater's report of what was eaten, because it is very difficult for an outsider to collect duplicates or observe everything a person eats. But eaters' reports may be inaccurate and biased. People may forget what they ate, have difficulty judging the amounts they have eaten, report inaccurately because they want to appear to conform to what the questioner wants to hear, or they may unconsciously change what they eat when they are asked to record it. Therefore, information on dietary intakes is never perfectly accurate. These errors are usually quite large, and perhaps highest among certain groups such as the very obese, or those with impaired memories, such as children, the very old, individuals with drug, alcohol, or mental health problems, and those with extremely erratic or disordered eating patterns. A common error is failure to mention dietary supplements, which, in countries such as the United States, often contribute substantially to dietary intakes [14]. Other items that the eater may judge to be socially inappropriate such as alcohol, high-fat foods, snack foods, sugar-sweetened beverages, candy or other desserts may also be omitted or underestimated. Forgetting what has been eaten is a particular problem when people are asked to remember what they have consumed over long periods, such as during the past year, especially if they are to provide estimates of the frequency and amounts they have eaten. Such estimates probably reflect more recent intake (e.g., a few weeks), and they are probably most accurate when individuals have fairly fixed intake patterns. When people are given lists of foods and asked to record their frequency of eating these foods, they often overestimate how much they ate, and results are usually adjusted to provide estimates that are more in line with plausible energy intakes. Also when people are asked to keep food records or diaries, often they unconsciously change what they eat to make the recording task simpler, or because they are surprised at how much they eat and decide to eat less to make their intakes appear more "balanced." Finally, all dietary assessment is based on food composition databases of chemical analyses of nutrients and other dietary constituents of interest. The values for constituents vary in quality and completeness, and, until recently, were not available for some nutrients, such as vitamin D and iodine. These gaps have now been remedied, but for many other bioactive constituents of interest, such as the flavonoids, isothiocyanates, and others, only limited analytical information is available.

Why Dietary Assessment Is Useful

Dietary intake assessment is used as a proxy for nutritional status because it is less expensive and easier to obtain than a full-fledged nutritional status assessment, and can often provide enough useful information to remedy problems. For example, it can identify those who are very undernourished because they are eating too little, and others who are eating too much, or imbalanced amounts of foods and nutrients. Dietary assessment also helps to clarify and strengthen assumptions about the presumed causes of changes in nutritional status when it is synchronized with information derived from anthropometric, valid biochemical, clinical, and other measures. Questioning can also reveal many environmental factors that influence dietary intakes and nutritional status that may need to be taken into account, such as climate, living conditions, exposure to pathogens, and socioeconomic conditions.

Dietary status assessment only measures what an individual eats, and not what is actually metabolized and reaches the body's cells, tissues, and organs, and so it may miss and fail to account for other causes of poor nutritional status, such as malabsorption or genetic polymorphisms that influence metabolism. Although dietary assessment theoretically is an extremely sensitive indicator of malnutrition, since it reflects a very early stage in the development of the pathology, in practice it is often not useful by itself. Dietary data are less precise and reliable than biochemical and anthropometric indices. Because dietary data are usually obtained by self-report from the patient or respondent or a proxy, they are usually more subject dependent, and vary more in validity and reliability from one person to the next. They often fail to reflect usual intakes and instead reflect only the prior few days. Intakes of nutrients from dietary supplements and oral nutritional supplements may be disregarded, causing more underestimation errors. Thus, it is difficult to determine whether suspected deficits in dietary intake are actually due to diet or to errors in reporting. Therefore, in order to fully assess nutritional status, dietary information must be combined with the other types of data.

Biomarkers

Because there are many problems involved in obtaining accurate information on food intakes, if precise estimates of intakes are necessary, as is the case in research studies, surrogate markers of dietary intake such as biomarkers are often preferable. Ideally, biomarkers are reliable and accurate biochemical or other measurements that can be objectively measured and evaluated.

Biomarkers can be indicators of intake, normal biological processes, nutritional status, pathological processes, pharmacological responses to an intervention, or health outcomes. In order to be useful, biomarkers must be analytically valid, useful, clearly show associations between the biomarker and disease, and reflect interventions with changes in both the biomarkers and clinical outcomes.

The most common sources of biomarkers are blood, urine, and other body fluids such as saliva. There are also functional biomarkers that measure processes such as immune function, cognitive function, or frailty, and more nonspecific biomarkers, such as grip strength that, together with other factors, may be used to estimate risk of disease or disability.

Some biomarkers reflect intake, others nutritional status, metabolism, risk, or health outcomes. The biochemical biomarkers in blood or urine that reflect nutrient intakes or nutritional status are used most commonly. They are useful because they are more objective than dietary intakes, which usually depend on individual reports and are subject to all the biases and errors that may entail. However, biomarkers also have their limitations. They reflect different things; some reflect dietary intake, others nutritional status, others metabolism, and still others risk or outcomes of disease. Measurements in urine and blood can be influenced by hydration status, diet, deficiencies in other nutrients, fasting, kidney function, and other factors, such as inflammation or diseases and conditions that are not diet-related. They also are dependent on correct collection, storage, and analysis using appropriate methods.

Some *intake biomarkers* are listed in (Table 2.3). Among the biomarkers reflecting *intake* are ascorbic acid and serum folate; they are specific but often only reflect intake over a short time (e.g., hours or days) rather than a longer time. Most of the biochemical or other biomarkers of intake are very specific and only reflect intakes of a single nutrient rather than the overall diet. For some nutrients, such as zinc and magnesium, there are no acceptable biomarkers that reflect long-term intake. Other biomarkers such as doubly labeled water ($D_2 O^{18}$) to estimate energy intakes are available but very expensive, require special equipment or expertise, and are difficult to use except in research studies. Biomarkers of intake must reflect it. Some biomarkers reflect diet alone over the past few hours or day, such as changes in serum folic acid levels, whereas red blood cell folic acid reflects many days or weeks of intakes. But these biomarkers may also reflect more than diet alone. Free folic acid and serum folic acid nutritional status. Clearly, both biochemical and other measures of dietary intake also have their limitations in clinical practice.

Nutrient	Biomarker	Comments
Iron	Hemoglobin	Low specificity (many different causes of anemia decrease hemoglobin, not just iron. Cutoffs vary by age, sex, and ethnicity). This is a good indicator for monitoring improvement in iron status if the individual is iron-deficient
	Serum plasma ferritin	This is an acute phase protein, and it is nonspecific; it increases independently due to acute or chronic inflammation, infection, malignancy, hyperthyroidism, liver disease, heavy alcohol use
	Serum transferrin receptor	A specific indicator of iron-deficient erythropoiesis that is not affected by inflammation
	TIBC (total iron binding capacity)	Nonspecific and varies over the day
	ZnPP erythrocyte zinc protoporphyrin	A sensitive indicator for diagnosis of the deficiency. However, the specificity is limited since it is affected by lead poisoning, the anemia of chronic disease, chronic infection, inflammation, hemoglobinopathy, and hemolytic anemias
Zinc	Serum plasma zinc	Responds to zinc supplementation, but it is easily affected by contamination, and is also affected by inflammation, fasting, estrogen use, hemolysis, and chronic illness
B12	Serum/plasma total B12	Measures total biologically active B12. Poor correlation with dietary B12. Also cutoffs are uncertain and kits for the test vary
	Serum/plasma methylmalonic acid MMA	Cutoffs are uncertain. Test is sensitive
	Serum/plasma holotranscobalamin HoloTC	
Folate	Serum/plasma folate	Varies with recent intake, and kits to test vary
	Erythrocyte folate	Kits vary. Samples are difficult to prepare and cannot be stored
	Serum/plasma total homocysteine Hcys	Depends on B2 status, B12 status, B6 status, and MTHR polymorphism state
Vitamin A		Sensitive to intake, but only if stores are low. It is not very sensitive because it is under homeostatic control

Table 2.3 Some biochemical biomarkers of intake (exposure) and/or nutritional status

Other biomarkers reflect *nutritional status*. One is 25 hydroxy vitamin D (25 OH D), which reflects both dietary intake and exposure to the sunlight's ultraviolet rays that activate the provitamin D in the skin. Some of the other nutritional status biomarkers are too nonspecific to be useful. For example, serum albumin is a marker of dietary intake, but it reflects not only protein nutritional status but inflammation and infection. Some status markers, such as serum thiamine, are specific, but they only reflect status over a short time (e.g., hours or days) rather than habitual intakes. Other status markers, such as red blood cell folate, reflect intake over a somewhat longer period of time (e.g., several weeks). 25 OH D reflects diet, but also exposure to sunlight, and thus is a good measure of nutritional status for vitamin D, but not of dietary intake alone. Another biomarker is 3-methyl-Lhistidine, which is an amino acid that is not used by the body to synthesize protein but is a catabolite of protein breakdown and therefore it is often used as an index of muscle breakdown and thus of protein nutritional status. However, its level in urine also depends on intake of muscle protein and therefore unless the subject is on a meat-free diet it is likely a poor indicator of muscle mass.

There are *functional biomarkers* or *intermediary biomarkers* that reflect metabolic processes thought to be along the way to an endpoint but which are not necessarily valid markers of the endpoint. For example, methylmalonic acid (MMA) and homocysteine (tHcy) are elevated when vitamin B12 status is low [15]. tHcy is sensitive but not specific to B12 deficiency because it can also be elevated due to reasons including a deficiency of folate, B2, and B6, as well as chronic alcohol abuse [16]. High MMA levels are almost exclusively due to a low vitamin B12 status in

folate-replete populations [17, 18] and accordingly it is a sensitive marker, but is related to renal function.

Fourth and finally, there are valid biomarkers that reflect *outcomes*, such as coronary artery disease or stroke. Often, instead of outcomes themselves that take so long to develop, valid surrogate endpoints are used such as serum cholesterol or blood pressure. These so-called surrogate endpoints are substitutes for a clinical endpoint that predicts a clinical benefit or a harm based on good evidence, and the biomarkers of them are called *surrogate biomarkers of outcome*. For example, high levels of low-density lipoprotein cholesterol (LDL-C) reflect risk of coronary heart disease, and hypertension is associated with risk of stroke. Biochemical indices such as LDL-C and high blood pressure are therefore considered good *biomarkers of risk or outcome*. Another surrogate biomarker with less predictive value is HDL cholesterol, which is not as good a predictor of risk of coronary heart disease as is LDL cholesterol.

In summary, biomarkers of intake, nutritional status, function, and risk/outcome all have their strengths and weaknesses. They are not substitutes for dietary intake measures, but they can provide useful additional or corroborating information in determining the cause of diet-related problems and in planning to alleviate them.

In the past few years, the BOND (Biomarkers of Nutrition in Development) project, an international project led by the Eunice Shriver National Institute of Child Health and Development at the National Institutes of Health has developed extensive monographs on assessing nutritional status using biomarkers for several micronutrients of public health interest: vitamins A, B12, folate, and the minerals zinc and iron [19].

Dietary Reference Intakes (DRI)

The Dietary Reference Intakes (DRI) are quantitative standards for nutrient reference intakes that are used for planning and assessing the diets of healthy people. They are based on the best evidence available for intake levels of nutrients that are compatible with good health. Prior to the development of the DRI, the 1989 Recommended Dietary Allowances (RDA) served as the standards for nutrient intakes. The DRI were developed conjointly by Health Canada and the Food and Nutrition Board, Institute of Medicine, US National Academy of Sciences in the 1990s. They are used as the basis for other materials, such as the Dietary Guidelines for Americans, that provide food-based dietary guidance, and for food labels [20, 21].

Overview of the DRI

The DRI consist of multiple reference recommendations that serve as dietary standards for various uses instead of the single RDA reference values used in the past [22–27]. They were developed because of the availability of new data on many nutrients and potential candidate nutrients. Also, new concepts on assessing dietary deficiency and excess were available by the 1990s. It was also felt that, if possible, dietary recommendations should be based on standards that would prevent obesity and reduce risks for chronic disease and excess. Finally, new-user needs, such as nutrient labeling, made a revision of existing recommendations imperative.

The DRI include the estimated average requirement (EAR), the recommended dietary allowance (RDA), or, if the RDA cannot be calculated, an adequate intake (AI). An upper reference level (UL) is also set. And acceptable macronutrient distribution ranges and energy needs are also provided based largely on epidemiological rather than experimental evidence [28]. The EAR, RDA, and AI are

defined with reference to specific criteria of nutrient adequacy. The UL, in contrast, is defined using specific indicators of excess. The reference weights and heights for the adults and children for whom the dietary recommendations are designed are based on recent population-based surveys of the US Recommendations are available for micronutrients (EAR and AI), protein, fat, carbohydrate, water (AI), and electrolytes (EAR).

Criteria on Which Nutrient Recommendations Are Based

The criteria for adequacy upon which estimates of nutrient needs are based are critical. There are often several conceivable criteria that might be chosen, and so expert judgment is involved in choosing that which is the most relevant and best supported by the available evidence. The amount of evidence and the criteria vary from nutrient to nutrient. Each EAR, RDA, and AI is described in terms of a selected criterion or criteria of adequacy and the rationale is provided with extensive documentation of the evidence available. These criteria include the best level to determine the risk of an individual's nutrient stores becoming deficient. For example, the criterion for folic acid among women in the childbearing years is based on a combination of three biochemical indicators: primarily, red blood cell folate, and, secondarily, plasma homocysteine and serum folate levels. Other criteria relate to reducing the risk of disease if they are available, although often they are not. For example, a separate recommendation for folic acid is made for women capable of becoming pregnant on a criterion related to reducing the level of neural tube defects. Prevention of chronic disease is a focus of some other nutrient adequacy criteria such as the association between fluoride and risk of dental caries. The associations between nutrient intakes and risk of most chronic degenerative diseases have proven to be more difficult to establish and remain an area of controversy and research. For example, the criterion for the amount of calcium and vitamin D recommended was based on that sufficient to provide retention of calcium during growth and minimize bone loss during adulthood. For dietary fiber, decreases in serum cholesterol were chosen. For potassium and sodium, adequate intakes were established but not on the basis of an association with chronic disease endpoints because data were judged insufficient to do so. The evidence base for a recommended level of including protein, phosphorus, magnesium, the B-vitamins, and choline was judged insufficient to define a function such as preventing chronic disease, and so the estimation of the requirement depended on other criteria, such as mineral balance. Some measures upon which EAR are established, such as nitrogen balance, are judged by some scientists to be inadequate in that they lack a close enough association with function and health to be relevant. There is currently much research devoted to developing measures of protein nutritional status that are valid, reliable, and more reflective than nitrogen balance of protein functions in the body such as maintenance of muscle mass, immune function, and the like. As new and better functional indices are developed, and these come to be associated with risk of chronic disease, estimates of nutrient requirements may also change.

Much attention lately has also been devoted to urging that "candidate nutrients" such as the flavonoids, lutein, and omega 3 fatty acids, which are thought to be associated with reduced risk for certain chronic diseases, be included as essential nutrients using the DRI process. Many of them are being consumed in relatively high doses by consumers. At present, evidence that these bioactive constituents of foods are essential nutrients is inconclusive. There is little reason to assume for some of these bioactives (that are xenobiotics) that the DRI model of a normal (Gaussian) requirement distribution of essential nutrients applies to them. It is also not clear that the DRI "risk-risk" U-shaped model of dose response with increasing signs of deficiency with decreasing levels of intake or increasing signs of toxicity with increasingly large intakes applies to them. The shape of the dose–response curves for these bioactives may be quite different. More research is needed on their roles in bringing about possibly beneficial effects on markers of chronic disease risk, and it is important to establish what are safe upper levels of intake for these constituents.

RDA

The recommended dietary allowance (RDA) is the average daily dietary intake level that is sufficient to meet the nutrient requirements of nearly all (e.g., 97–98%) of healthy persons of a specific gender at a particular stage of life or physiological condition, such as pregnancy or lactation. The only use of the RDA is to serve as a goal for individuals, and not for populations. All Dietary Reference Intake values are publicly available on the Internet [29].

To establish an RDA, agreement on the criterion to be used for the requirement is the first issue that must be resolved. This criterion is used as the metric to assess adequacy. An appropriate cutoff must then be established for each criterion that is used. The cutoff determines the difference between what is considered an adequate and an inadequate level of nutritional status. In order to set this cutoff, there must be sufficient data on individuals who are consuming levels of intake at which some reach the suggested cutoff for the criterion and at which others do not. This permits the establishment of a dose–response relationship. It is also necessary to have an understanding of the distribution of requirements among individuals in the group from which the requirement is estimated. The requirement distribution (but not the intake distribution) is usually normal, conforming to a bell-shaped Gaussian curve. When nutrient requirements are skewed for a population, such as iron in women during the childbearing years, other approaches are used to find the 97–98th centile to set the RDA.

The RDA is the value that should be used for planning individual intakes. The RDA is not appropriate for assessing the diets of groups, or for planning diets for groups. An interactive tool for calculating the appropriate RDA/AIs and energy needs from an individual's age, sex, physiological condition, weight, height, and physical activity level is available at the following web site: http://fnic. nal.usda.gov.

EAR

The estimated average requirement (EAR) is the amount of a nutrient that is estimated to meet the requirement of half of the healthy individuals in a specific life-stage and gender group. The EAR is used to assess adequacy of intakes of population groups, and along with knowledge of the distribution of requirements, to develop Recommended Dietary Allowances. The requirement is the lowest level of a nutrient eaten chronically that will maintain a defined level of nutrition in a person. Before setting an EAR, a specific criterion of adequacy must be chosen. The selection is based on a careful review of the literature. Among the criteria that may be used, reduction of disease risk may be considered, along with other health parameters. It is assumed that the distribution of requirements is normal. Ideally, the standard deviation (SD) of the requirement, that is, an estimate of its variability of requirement, is also available. If the SD is not available from empirical data, then an estimated coefficient of variation (CV) is used, usually 10%. Under these circumstances, the RDA is assumed to be $1.2 \times EAR$. Conversely, if the CV is 15%, the RDA is $1.3 \times EAR$.

Recommendations for energy intake differ from those for vitamins, minerals, and protein because individuals of the same age, sex, reference weight, and physiological condition often differ markedly in their energy needs due to differences in body composition and in physical activity levels, among other factors, and such factors as actual weight, height, and physical activity level are also needed to calculate it. Moreover, the standard to achieve is actually like the estimated average requirement to maintain weight and health, not an RDA. Two SD above the EAR for energy as a recommendation would cause many individuals to consume excess food energy, which would be stored as body fat, ultimately with adverse effects upon health.

AI

The adequate intake (AI) is a recommended daily intake level based on observed or experimentally determined estimates of nutrient intake in a group of healthy people. The main use of the AI is as a goal for the nutrient intake of individuals. It is used when an RDA cannot be determined. The AI is usually based on observed levels of intake that appear to maintain an acceptable level of health or growth. The presence of an AI is an indication that experts believe that sufficient data on requirements are not yet present for the functional criterion that has been chosen, and that more research needs to be done. The AI is set at a level that is expected to cover the needs of at least 98% of people, but it might cover far fewer, or far more, since the EAR cannot be estimated. Unfortunately, the degrees to which AIs exceed average requirements differ among nutrients and population groups. Therefore, it is meaningless to describe the proportion of the population below the AI; it is more useful to report the proportion of the population *above* the AI.

In the DRI reports, AIs rather than RDA are provided for all nutrients in infants up to a year of age, and for fluoride, potassium, pantothenic acid, biotin, and choline for persons of all ages [29]. Both the RDA and AI are appropriately used for setting goals for intakes of individuals. However, greater uncertainty surrounds the AI.

UL

Nutrients are essential for basic functions of human life and well-being. However, at excessive levels of intake they have possible adverse effects. Adverse effects include any significant alterations in the structure or function or any impairment of a physiologically important function in an individual. Also, since intakes of one nutrient may alter the health benefits conferred by another, detrimental nutrient–nutrient interactions are also considered to be adverse health effects. These values are also available at the Institute of Medicine web site (http://www.iom.edu/Activity/Nutrition/SummaryDRIs/DRI-Tables.aspx).

It is important to remember that at the levels of nutrient intakes from diets of foods, human experience is considerable. It is only in the past few decades that single concentrated sources of nutrients have become available in the form of dietary supplements and highly fortified foods; so now, the possibility of higher intakes is more likely. At present there is no evidence to suggest that nutrients consumed at current RDA and AI levels in enriched or fortified foods pose a risk of adverse effects to most persons [30]. However, with very high amounts of fortified foods, dietary supplements, and over-the-counter medications that contain nutrients all consumed together, total intakes may reach or exceed the safe levels.

The tolerable (or safe) upper intake level (UL) is the highest level of chronic and usual daily nutrient intake that is likely to pose no risks of adverse health effects to almost all individuals in the general population. The more the intake is above the UL, the greater is the risk of adverse effects. The intakes included in the UL for most nutrients are food, water, and nonfood sources such as nutrient supplements and pharmacological preparations. The word "tolerable" is used because most individuals should be able to tolerate this level of intake. However, it does not imply that this high a level of intake is desirable. The UL is *not* an intended level of a nutrient to be consumed. Moreover, the UL is not a level at which there is a beneficial effect. Rather, it describes the intake level at which there is a high probability that the dose of the nutrient can be tolerated biologically. When total intakes have been associated with toxicity, this fact is stated. In a few cases, it may be that toxicities are only associated with nutrients used in supplements or as drugs. For example, excessive beta carotene appears to be associated with adverse outcomes but only in supplements; and therefore, the UL for beta carotene is based only on supplements. Many individuals use large amounts of nutrients containing dietary supplements for preventive or curative purposes. Excessive intakes are a particular concern for nutrients that are not easily metabolized or disposed of by the body, such as fat-soluble vitamins.

Dietary Risk Assessment

Risk assessments describe the relationships between exposure to a nutrient and the likelihood that adverse health effects will occur in the exposed population. They systematically evaluate the probability of adverse health events occurring in humans from excess exposure to an environmental agent such as a nutrient or food component. Risk-risk assessments involve both the risks of taking too much of the nutrient versus the risk of not taking enough of the nutrient or supplement [31]. Such assessments are becoming increasingly common. A constant set of scientific principles involving risk assessment is used to set the UL. The process involves identifying hazards, assessing dose-responses, intake assessment, characterization, and evaluation of risk. All evaluations and judgments are explicit and evidence is provided to document the conclusions that are reached. Both qualitative and quantitative types of evidence are considered. ULs are set by first reviewing the literature to determine levels at which no observed adverse effects (NOAEL) are noted, or at the lowest level of intake associated with observed adverse effects. Then an uncertainty factor is applied to reduce the intake level from the lowest adverse effect level to insure that even the most sensitive persons would not be affected by the UL dose chosen. At present, for many nutrients there is simply not enough evidence to develop a UL. The risk is expressed as the fraction of the exposed population, if any, that has nutrient intakes in excess of the estimated ULs [23].

ULs are not always certain, fixed, and unvarying values. For many nutrients, ULs are not available because data are so sparse. Often, although intakes are available, there are no data on the adverse effects of taking large amounts of nutrients, or the data are only anecdotal in nature. In such cases, the UL cannot be established. This does not mean that there is no risk of adverse effects from high intake of the nutrient; in fact, when data about adverse effects are very limited, extra caution may be warranted. The data that exist are often scanty or drawn on studies to address other questions.

AMDR

The acceptable macronutrient distribution range (AMDR) for the energy-yielding nutrients provides a range of intakes that appear to be compatible with good health for them. The DRI and AI for water and macronutrients, and AMDR ranges for fat, carbohydrate, and protein, as well as recommended ranges for dietary cholesterol, trans fatty acids, saturated fatty acids, and added sugars are publicly available [29]. Energy needs can also be calculated from tables given in the DRI report on macronutrients and energy.

Current Status of the DRI

The most recent report on DRIs was completed in 2010 on calcium and vitamin D because of the substantial amount of new information available on both nutrients [32]. Both were given new EARs and RDA on the basis of new data that were available, and the recommendations for each were raised slightly over the previous report issued in 1997 [24]. The values for other nutrients in the 1997 report (magnesium and phosphorus) were not revised and remain the same. The report on folic acid and other B vitamins was issued in 1998 [25]. The report on "antioxidant" nutrients (ascorbic acid, vitamin E, and possibly others) was issued shortly thereafter [33], and then the macronutrients and energy [27], and a volume on electrolytes and water [26]. Volumes on the uses of the DRI for assessment [34] and planning [34] were also developed by a panel on the uses of the DRI. A composite volume is available for practitioners [35]. DRI for all nutrients, energy, and water are also available.

Gaps in data for some members of the population Increasingly more individuals in the population are very old or very young. Yet reference standards for infants less than 32 weeks gestation or elders over 80 years are derived largely by extrapolation. These gaps need to be filled with additional research.

Disease and Illness Although the DRI are designed as recommendations for healthy individuals, more than half of all Americans have one or more chronic conditions, and their number is growing as the population ages. Many of the common chronic diseases such as cardiovascular disease, diabetes mellitus, some of the cancers, and dental caries may be caused in part by diet, while others such as arthritis, asthma, and chronic obstructive lung disease have nutritional implications for their treatment. Moreover, many members of the population in countries like the United States and Canada are medicated for one or more chronic diseases. When effects of these medications are known, dietary recommendations are altered accordingly. However, little is known about whether some common medications affect some nutrient requirements. Research is needed to clarify their effects, particularly in elders with many diseases and medications. For individuals who are ill, the best recommendations for nutrient intakes at present depend on the disease itself, its effects on the absorption, metabolism, storage, and/or excretion of specific nutrients. In such cases, recommendations are best set by medical specialists for the diseases involved. Most nutrient needs are not usually affected, however, and the DRI for healthy individuals can be used for them until better data are available.

Prevention of Chronic Disease

Another major challenge is to how to assess the links of dietary constituents with chronic disease endpoints as outcomes. Since the major causes of morbidity and mortality in highly industrialized countries are chronic degenerative diseases, in order to have an impact on delaying morbidity and reducing premature mortality, it is essential to act upon them. Even small reductions in diet-related risks would have potent effects at the population level. However, it is difficult to develop the evidence that diet–chronic disease outcomes exist. Not everyone is at risk of every disease; the prevalence varies, with evidence for caries strongest, then osteoporosis, followed by coronary heart disease, kidney stones, and, lastly, cancers. Dietary intake exposures occur long before the chronic disease occurs, and experimental studies would take many years to give definitive answers. The use of observational studies to establish recommendations based on chronic disease outcomes has many problems and makes causal inference weak. Dietary exposures occur long before the chronic disease and diet is only one of many causative factors that vary in strength. There are also many problems with measures of outcomes. Morbidity measures are often imprecise and mortality takes many years. Surrogate or intermediate markers rather than these outcomes are often lacking or unvalidated, and some intermediate markers may not even be on the causal pathway to the outcome.

In the existing DRI, only five nutrients were assigned reference values based on chronic disease endpoints. They were calcium and vitamin D with osteoporosis and fractures; fluoride and dental caries; dietary fiber and coronary heart disease; and potassium and a combination of endpoints including salt sensitivity (a risk factor for hypertension), kidney stones, and blood pressure. Chronic disease endpoints do not fit well into the existing DRI paradigm since many dietary and nondietary factors contribute to chronic degenerative disease; they are multifactorial. Chronic disease risk shows only small responses to increased doses of the nutrient. By definition the EAR is the intake to achieve absolute risk reduction of 50% (i.e., the probability of getting a disease over a certain time). But this depends on the relative risk factor, and absolute risk is not 100% for everyone who has a given chronic disease. And 100% of the population is not at risk of the disease if the nonnutrient dietary component is eliminated. Moreover, adding the dietary constituent does not prevent the disease in 100% of the population. In fact, risk of the disease probably ranges from very high to very low, in a rough rank order of the association of a nutrient and risk ranging from high to low. Risk reduction is almost never 50%, as is the case when an EAR can be established. It is therefore likely that instead of focusing on single nutrients, patterns of nutrients or food intake patterns will be more useful in linking diet to chronic disease endpoints. The challenge is how best to do this.

Excessive Intakes

Although concerns about excessive energy, fat, and alcohol intakes have been of concern for many years, since the promulgation of the upper safe level of intakes, excessive intakes of micronutrients are now receiving greater attention. In part, this stems not only from estimates of the UL but from the population's widespread use of dietary supplements and fortification of the food supply, both of which are concentrated sources of nutrients. In response to these concerns, the National Health and Nutrition Examination Survey (NHANES), the nation's national population-based survey, monitors total nutrient intake from naturally occurring foods, enriched/fortified foods, and dietary supplements. It also monitors biochemical indices of nutrient status and excess, when appropriate and available.

Recommendations for Individuals Versus Populations

The DRI Committee set recommendations that apply to suggested intakes of nutrients of individuals. The EAR is intended for assessing intakes of populations or groups, whereas the RDA is an intake target for individuals. It is possible to assess intakes of populations and to make some broad statements about the adequacy of an individual's dietary intake. In contrast, it is relatively straightforward to make recommendations about and plan adequate intakes for individuals but it is more difficult to do these tasks for populations. At present, population recommendations have not been set for the United States or Canada, but only for individuals. From the practical standpoint, population recommendations for nutrient intakes are much more environmentally influenced and culture-bound than estimates of population requirements, and likely vary much more between countries. This is because

mean usual nutrient intakes differ greatly between populations of one country and another, as do nutrient intake distributions, which are very wide in some countries and very narrow in others. In setting population-level recommendations, both the issue of ensuring that everyone in the population gets enough (prevalence of inadequacy) while avoiding too much (prevalence of excess) must be addressed. This has proven to be possible to do for relatively homogeneous groups, such as recipients of some of the large USDA food programs, such as the Women, Infants, and Childrens' Supplemental Food Program and the National School Lunch and Breakfast Program. However, the US population as a whole is much more heterogeneous in their requirements. Moreover, most individuals make their own food and dietary choices rather than being given and eating a large share of their total intakes with rations designed to cover evident shortfalls, and thus population recommendations are much more difficult to develop. Recommended levels for population intakes must therefore take into account not only EARs but also the distributions of nutrient intakes within each of the major subgroups within the population with very different intakes or requirements. Strategies such as fortification, supplementation, dietary diversification, nutrition education, limitation of excessive intakes, and others that may be theoretically acceptable for bringing intake distributions with respect to requirement distributions into an optimal range in one country may not be so in another. For example, in the United States and Canada, fortification of wheat flour with folic acid was acceptable and appears to have achieved its goals of reducing the proportion of the population below the EAR while avoiding a large proportion of the population achieving intakes in excess of the UL. Vitamin D fortified milk has also largely eradicated rickets in young children. However, fortification with folic acid and vitamin D has not proven to be culturally acceptable in many European countries. Also, variations in resource and environmental constraints exist. The desired percent of the population having access to, or actually achieving, intakes that meet whatever criteria are chosen for the DRI also vary from one country to another. Economic realities, usual intakes, and historical precedents are best considered separately from biological requirements. Setting population recommendations for intakes therefore involves scientific judgment, but also political will, and cultural sensitivity.

With respect to the difficulty of establishing intakes for populations, consider, for example, the difficulties in implementing a goal of population intakes not to exceed 7% of calories from saturated fat. In order to achieve this, the population intake distribution would have to have a mean much lower than this, since no individual could exceed 7%. Such a goal may be impractical or difficult to achieve. In any event, knowledge of the distribution of population intakes as well as that of requirements is essential before such recommendations are promulgated.

Uses Outside the United States: Are the DRI Exportable?

In the past two decades, the DRI have been used in whole or in part by many other countries. While the DRI are an excellent set of standards, North America is not the world repository of all nutrition scientific wisdom and truth, and, as time goes on, they will no doubt be refined by experts in other countries as well as in the United States. It may be useful to consider which of the DRI are likely to be useful and exportable to other countries, and which are not. Many factors influencing DRI values are based primarily on biological rather than environmental influences, and these are largely the same from one country to another. Determinations of the EAR and UL, which are influenced largely by human biology, should also be useful in other countries. The evidence-based reviews of the scientific literature on experimental studies of requirements for each nutrient focus on these biological factors. As new studies become available, other experts can review the evidence, add to it, and come to their own conclusions. Other factors are not as easily exportable from one country to another. For example, the functional criteria that could be used to set the US/Canadian requirements are many, and the most appropriate ones for another country may vary with the environment, expert judgment, and health realities. Other countries may have circumstances that make different functional criteria and endpoints more appropriate.

Exportability of the AMDR is likely to be limited since they rely more on US and Canadian intake levels and not on direct experiment. Food patterns, nutrient intakes, and environmental factors vary from country to country. In other countries, population profiles, resource constraints, and expert judgment may lead to the choice of different criteria for adequacy, with the result that AMDR, EAR, RDA, AI, and UL values chosen may differ from one country to the next.

Diversity in expert judgments often helps to drive science forward and seek firmer evidence. For other countries, the DRI recommendations, which are more evidence-based than ever before, deserve examination. The DRI are useful compendia to review for efforts in other countries. However, exportability/importability judgments of existing US/Canadian values must be considered on a case-by-case basis. Over the past decade there has been growing awareness that international efforts to establish nutrient requirements make sense, and projects such as the European Union's EURECCA (European Micronutrient Recommendations Aligned) and the BOND (Biomarkers of Nutrition in Development) have developed approaches for such collaborative efforts.

Revising the DRI

Science is constantly changing and growing; therefore, there is always a need for revision as new data become available. The DRI were conceived as the first attempt, and not as the last word, leaving room for them to change and diversify. Because all scientific conclusions are subject to revision as new evidence emerges, constant review is needed [23, 36]. Similar efforts are now in progress in the European Union, led by the European Food Standard Agency (EFSA) and elsewhere. Expert groups in other countries and regions will no doubt have useful suggestions and improvements that the US/ Canadian committees can learn from and incorporate into revisions. There is growing realization that systematic evidence-based reviews tailored to nutritional data are important in evaluating the evidence, and that these must go hand in hand with expert judgment [37–41].

Next Steps for the DRI

Dietary reference standards must be revised periodically when new science become available. There is currently interest in the nutrition community for developing DRI for omega-3 fatty acids, magnesium, protein, vitamin E, and several other nonnutrient bioactives, such as flavonoids, polyphenols, and isothiocyanates. Alcohol, while not an essential nutrient, also requires more detailed consideration than it has yet received, because it appears to have both beneficial and detrimental health effects.

Eventually, it is likely that many of the processes of evidence review and other technical aspects of data collections will be conducted in one country and then used as the basis of recommendations in other countries. The World Cancer Research Foundation has adopted a systematic process for ongoing evidence reviews for dietary recommendations on cancers that may also be adaptable to reviews of nutrient requirements [42]. Complete information about the Canadian/US DRI and their applications is provided in detail elsewhere [43]. Some of the lessons learned and new challenges yet to be addressed have been summarized in a recent publication from the Institute of Medicine [36]. They include the conceptual framework for DRI development, criteria for scientific decision making on nutrient recommendations, improving guidance for users of the DRI, and what kind of process should be envisioned for DRI development in the future. Greater insights with respect to physiological,

environmental, and genetic factors are needed. Techniques for extrapolation and scaling are needed. There is also limited evidence that some bioactive nonnutrient constituents of foods such as the flavonoids, lutein, glucosinolates, and others may have effects on chronic disease, and there is a need to develop a scientific framework for intake recommendations of these substances. Preventing nutrient excess and chronic disease prevention are key public health concerns [44].

Uses of the DRI

For assessing the adequacy of individual intakes, the best criterion is to compare intake to the EAR, although only a very rough estimate of adequacy can be made (for this reason other indices of nutritional status are also necessary) [43]. The RDA is a useful target for planning individual intakes. The most appropriate ways to evaluate the intakes of groups are to use the EARs when they are available. The proportion of individuals below the 50th centile (e.g., the EAR) is estimated. These are the individuals who are at greatest risk of dietary inadequacy. Note that the percent of the population below the Recommended Dietary Allowances is *not* the population at risk of inadequacy; such a criterion will overestimate the proportion truly at risk because of the very definition of the RDA. The AI is probably close to the group mean intake in some but not all instances. If the mean intake of a group is at or above the AI for a nutrient, there is unlikely to be a problem. However, it is not possible to assess risk of inadequacy quantitatively with AIs, although qualitative statements can be made. The AI is a good target for planning group intakes.

Dietary Guidelines for Americans

The Dietary Guidelines for Americans [45] are recommendations to help guide individuals to make healthy dietary choices. They are targeted to people over the age of 2 years; other recommendations are more appropriate for small infants and toddlers who are not yet consuming family fare. The guidelines emphasize healthy eating, which involves not only getting enough food but balance, variety, and moderation in consumption patterns to decrease diet-related risks of chronic degenerative diseases.

The original Dietary Guidelines, first issued in 1980 and presented here (Table 2.4), have stayed quite constant over the years [46]. However, the messages have evolved from proscriptive suggestions that centered on foods and dietary constituents to be minimized or avoided, to more prescriptive and quantitative recommendations about positive dietary behaviors and dietary patterns. Also, as scientific evidence supporting the guidelines has increased, the emphasis and content have changed somewhat based on new information.

 Table 2.4
 Original Dietary Guidelines for Americans in 1980

Eat a variety of foods Maintain desirable weight Avoid too much fat, saturated fat, and cholesterol Eat foods with adequate starch and fiber Avoid too much sugar Avoid too much sugar Drink alcohol in moderation, if you drink

^aCalorie levels assume choices are low-fat, lean foods from the five major food groups, using foods from the fats, oils, and sweets group sparingly

Another chapter elsewhere presents an excellent, detailed overview of the 2010 Dietary Guidelines [47]. Updates and details on the 2015 Scientific Advisory Committee Report [21] are also available at http://www.dietaryguidelines.gov. The 2010 Dietary Guidelines for Americans recommend that to stay healthy, one should eat a variety of foods, maintain or improve one's weight by balancing food intake with physical activity, choose a diet that is plentiful in grain products, vegetables, and fruits; moderate in salt, sodium, and sugars; and low in fat, saturated fat, and cholesterol. For those who consume alcoholic beverages, they need to do so in moderation. Note that the guidelines are qualitative for the most part, recommending patterns of foods and food groups, rather than very specific amounts of foods or nutrients. This qualitative emphasis reflects both scientific uncertainty about exactly how much of each nutrient should be eaten, and the Dietary Guidelines' educational purpose. Some of the guidelines are more quantitative. For example, the variety goal is defined more precisely in the text that accompanies the dietary guidelines. Also, specific goals are provided for dietary fatty acids, since the evidence base on intakes of these nutrients is now very well developed.

The 2015 Dietary Guidelines Scientific Advisory Committee's (DGAC) report was issued in mid-2015; the official Dietary Guidelines for Americans (DGA) 2015–2020 were issued by the US Department of Health and Human Services and the US Department of Agriculture early in 2016 [45]. It is available on the Internet at http://health.gov/dietaryguidelines/2015/guidelines/. Many of the elements of the prior DGA are conserved with updates on the nutrition science when it had changed. Using population-based data on food consumption, modeling was done to develop healthful intake patterns. Since modeling to construct food patterns was done using data largely that did not include intake of dietary supplements, it is likely that the prevalence of nutrient deficiencies is actually lower, and the prevalence of excess of some micronutrients is likely to be higher than results would indicate; the effects on macronutrients are probably less, and those on energy nil.

In the 2015 DGA report, the emphasis on overweight, obesity, and physical activity continues. The basic structure of the recommendations, emphasizing healthful overall dietary patterns by recommending different kinds and amounts of food groups and subgroups rather than individual foods, also remains. The recommended pattern is high in vegetables, fruit, whole grains, low or nonfat dairy foods, seafoods, legumes, and nuts. A pattern lower in red and processed meat is suggested. Multiple dietary patterns including the USDA food pattern, the DASH (Dietary Approaches to Stop Hypertension) [48], a vegetarian pattern, and a "Mediterranean" pattern are all considered acceptable. More emphasis is given to sustainability than in prior reports; the DGAC stated in its report that a dietary pattern higher in plant-based foods and lower in animal-based foods is more health promoting and associated with lesser environmental impact than the average US diet, but another aspect of sustainability, waste, was not extensively addressed. Regardless of one's eating patterns, perhaps the best basic advice on the topic is that given to consumers by government nearly 100 years ago during World War I: Buy food with thought, cook it with care, save what will keep, eat what would spoil, home-grown is best, and do not waste food! (Fig. 2.1).

In the DGA 2015–2020, reductions are called for in sodium (less than 2300 mg per day), saturated fat (not more than 10% of Calories), less added sugars, and more potassium. Specific guidance on cholesterol was not given since it was not considered to be a current public health concern any longer. Little emphasis unfortunately was paid to alcohol, although this is a major source of calories for many Americans. Food-based sources rich in nutrients were emphasized rather than a reliance on dietary supplements.

It goes without saying but goes better said that providing guidelines is one thing, incorporating them into one's daily life is quite another and a much more difficult task for experts as well as laypersons. Recommendations for putting the Dietary Guidelines for Americans into action are available and downloadable in consumer-friendly format. They deserve careful reading by health professionals and consumers. Many other guidelines are also available, including many disease-specific guidelines, such as those issued by the American Heart Association, the American Cancer Society, and the American Association of Cancer Research [49]. In most respects, these other guidelines are similar to that of the Dietary Guidelines for Americans. There is no evidence that they promote better health or decrease risk of chronic disease than the Dietary Guidelines, although they may be helpful and reassuring to those who know they are at risk for particular health problems.

USDA Food Pattern

Another key piece of Federal food guidance is the USDA Food Pattern, which is designed to help people consume adequate and balanced diets that are in line with the Dietary Guidelines by providing recommendations for groups of foods that are similar [50]. Human beings do not require foods; they require nutrients. However, since people eat foods, recommendations that help the most are those that are food-based recommendations. Variants of the USDA Food Pattern are also the basis of other food-based recommendations such as the Dietary Approaches to Stop Hypertension (DASH) [48], the Harvard Alternative Healthy Eating Index [51, 52], and Harvard's Alternate Healthy Eating Pyramid [53], as well as various Web-based tools to help consumers plan menus that meet the DRI [54]. The USDA Food Pattern identifies the amounts of foods consumers are recommended to consume from five major food groups (fruits, vegetables, grains, protein foods, and dairy) and their subgroups (dark green vegetables, orange and red vegetables, starchy vegetables, other vegetables, beans and peas, whole grains, enriched/refined grains, meat/poultry/eggs, nuts, seeds, soy products, seafood). The patterns were designed to meet the Recommended Dietary Allowances (RDA) for nutrients so that nutritional adequacy would be achieved without exceeding recommended energy intakes. They were also designed to be below the 2010 Dietary Guidelines for Americans limits for sodium and saturated fat. Recommended amounts to consume from each food group differ depending on an individual's energy and nutrient needs. Patterns are provided for 12 different calorie levels and assigned based on the person's age, sex, and activity level. The patterns provide for only limited amounts of solid fats and added sugars for the calories left after the other goals have been met. This is because meeting nutrient needs within energy constraints, particularly if people are very sedentary, provides very litle room for components that provide mostly calories. The complete Food Pattern modeling report and details on how it is constructed are provided in the 2015 Dietary Guidelines Advisory Committee report. The USDA Pattern uses nutrient-dense foods because when amounts eaten of typical foods rather than the more nutrient-dense varieties of the same foods most people eat were used in modeling, the goals for moderation in intakes were not met. Energy, total fat, saturated fat, and sodium exceeded the upper limits in all of the patterns that were modeled, often by substantial margins. When modeling was done using typical food choices of consumers, calories were 15–30% (i.e., 350–450 kcal) above the target calorie levels for each pattern, although adequacy goals for nutrient deficiency were not substantially affected [50, 55]. In comparison to recommended amounts in the USDA Food Patterns, the 2015 DGAC found that the majority of the US population had low intakes of key food groups that are important sources of the shortfall nutrients, including vegetables, fruits, whole grains, and dairy. Furthermore, US population intakes were judged to be too high for refined grains and added sugars. Americans also ate fewer vegetables than experts recommend. When typical consumption patterns were examined, intakes of vegetables in general and starchy vegetables in particular differ from the recommended pattern in both the types and amounts that are eaten.

The USDA system for food modeling uses a number of subgroups and item clusters. Americans eating typical diets do not consume the most nutrient-dense forms of foods within each food group, and thus often nutrient intakes may fall short while energy intakes surpass requirements [56]. To remedy this, it has been suggested that rather than emphasizing achieving a certain number of servings from each food group more emphasis be placed on appropriate (e.g., higher nutrient density, lower in caloric

density) choices within food groups [57]. The Dietary Guidelines for Americans 2015–2020 describes the associations of food patterns with health outcomes, and several patterns are recommended.

USDA's MyPlate

The USDA Pattern is difficult for people to remember while they are eating foods each day. Therefore, over the years, there have been various efforts to develop graphics to accompany guides for healthy eating that will be easier to remember and follow. Fifty years ago the most popular guide was called the Basic Four Food Groups. It focused only on intakes of foods that were necessary to assure adequate intakes of protein, vitamins, and minerals, and assumed that people would eat additional foods to meet their energy needs. Over time, the need for a graphic representation that included entire intakes including energy became evident. The US Department of Agriculture introduced the Food Guide Pyramid in 1992 in response to these requests. Figure 2.2 presents this "first" pyramid. It provided a simple graphic tool that assisted people in choosing healthy dietary patterns that were varied, balanced, and moderate. The pyramid also suggested the number of recommended servings per day from each food group that people were likely to need to eat to meet their nutrient needs from food alone. The reason for dividing foods into groups was that each of the food groups provided some, but not all, of the nutrients people need to eat every day. The pyramid design emphasized the importance of consuming a menu that was based on plenty of breads, cereals, rice and pasta, vegetables and fruits, two to three servings from the milk group, and two to three servings from the meat group, with an emphasis on minimizing foods high in fats, sugars, and alcohol, and avoiding excessive energy intakes.

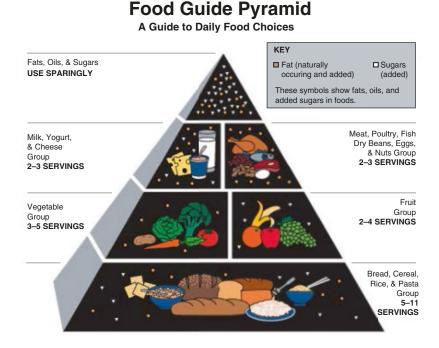


Fig. 2.2 USDA food pyramid early 1990s

The suggested serving sizes that were provided in the first pyramid were not necessarily those that people were accustomed to eating, and this led to confusion among consumers and health professionals. The amount people ate at a sitting would often be more than just one serving. For example, a usual dinner portion of spaghetti counted as two or three servings, and not a single serving of pasta. No serving sizes were given for fats, oils, and sweets, although it was noted that they should be limited if a person needed to lose weight. These calorie-dense foods, and alcohol, provide calories but relatively few vitamins or minerals.

The "second" pyramid, *MYPYRAMID*, was introduced in conjunction with the Dietary Guidelines of 2005 (Fig. 2.3). The illustration of MyPyramid shows that instead of an Egyptian style pyramid of bricks representing food groups, this pyramid appeared to many commentators to look more like a circus tent, with colored streamers representing each food group in tiers down the pyramid. The designers' hope was that the graphic better portrayed the desirability of eating nutrient-dense foods that were less calorically dense first (e.g., the base or bottom of each food group), and only later the more calorie-dense foods if energy needs had not been met (foods at the narrow top of the pyramid). However, the concept was difficult to grasp and the graphic was not successful in depicting the concept of "nutrient-dense foods first." Several other attempts to convey this concept have been made but none has gained traction with professionals or consumers [58, 59]. Another Web-based innovation in 2005 offered a calculator to tailor the pyramid to individual needs. It customized recommendations to the individual's age, sex, and weight, and presented menus that were appropriate in calories and other nutrients. The MyPyramid Planner (now supplanted by MyPlate) is available on the World Wide Web and is useful for helping people tailor their intakes of specific foods. It is available at http://www. MyPlate.gov. New tools are now being developed.

The third graphic, and the one that is now current, is the ChooseMyPlate icon, which consists of a plate (See Fig. 2.4), along with a new interactive tool designed to permit health professionals and

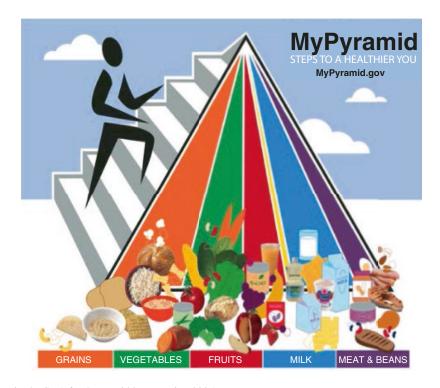


Fig. 2.3 Revised USDA food pyramid later version 2005



Fig. 2.4 ChooseMyPlate Icon 2014

consumers to develop their own personalized nutrition and physical activity plans, and to track intakes and outputs [60]. Other support on planning intakes and making healthier choices is also available there. Most of the information and educational materials that were developed for MyPyramid can also be used with ChooseMyPlate. Updates for the Dietary Guidelines 2015–2020 are now in progress.

There are many other graphics, pyramids, and other shapes produced by other organizations and designed to help consumers. Oldways, an organization dedicated to changing the ways people eat and to featuring traditional cuisines, has produced Vegetarian, Asian, Latino, and Mediterranean diet pyramids. The pyramids are available on the World Wide Web at http://www.oldwayspt.org. Special Pyramids are also available for older Americans, children, and other groups. The extent to which these "other" pyramids meet the DRI may vary however, and their construction needs to be reviewed before they are adopted for use in counseling.

Healthy Eating Index

The Healthy Eating Index (HEI) is a summary measure of people's overall diet quality. Quality is defined as variety among food groups and also avoiding too much of certain dietary constituents. In a very rough sense, the score for the population as a whole provides a "report card" on how well Americans are eating [61, 62]. The HEI-2010 is a 100-point index that aims to capture the multidimensional nature of diet quality and allows assessment of total quality as well as individual nutrients and food groups of interest [63]. This updated diet quality index, developed by the US Department of Agriculture, reflects the recent Dietary Guidelines for Americans (2010) and is in the process of being updated to accommodate changes in the Dietary Guidelines for Americans 2015–2020. The HEI-2010 has 12 components, nine of which assess adequacy (higher scores are achieved through a greater supply relative to energy or a more appropriate balance in the case of Fatty Acids) and three of which capture moderation (higher scores are achieved through a lower supply relative to energy). The adequacy components include Total Fruit, Whole Fruits, Total Vegetables, Greens and Beans, Whole Grains, Dairy, Total Protein Foods, Seafood and Plant Proteins, and Fatty Acids. The moderation components include Refined Grains, Sodium, and "Empty" Calories (i.e., calories and little else). The minimum score for all components is zero, whereas the maximum score varies between 5, 10, and 20. Additional details regarding the development and scoring of the HEI-2010 can be found elsewhere [64]. A high score on a component indicates that intakes are close to the recommended ranges or amounts, as stated in the USDA Food Guide Pyramid/ ChooseMyPlate, the 2010 Dietary Guidelines for Americans, or some other similar authoritative foodbased dietary guidance. Low scores indicate less adherence with recommended ranges or amounts of nutrients. Thus, the maximum overall score for the components combined is 100.

Definitive, comprehensive reports on nutritional status were issued periodically by the Federal government for many years in a single document on national nutrition monitoring. Each report provided detailed discussions of dietary patterns and nutrient intakes, but the process of assembling them took many years. Today, reports on specific issues are published periodically but in a more timely manner by agencies in the relevant departments.

The more recent analyses indicate that most Americans fail to meet the guidelines set forth by federal nutrition policy [56]. In general, Americans consume more sodium, energy, and fat than recommended and less fruits, vegetables, whole grains, and low-fat dairy than recommended [47, 65]. Indeed, the average HEI score for Americans increased from 48/100 in the 1970s to 55/100 in more recent national survey data [66]. Miller et al. point out in a recent publication that the food supply available to US consumers is inadequate for all Americans to achieve higher HEI scores [66], and additional concerns have been raised about economic costs to achieve health diets that continue to be of concern [67].

Nutrient Labeling

Ingredient Labeling

Another consumer education and awareness tool is the food label. Packaged and processed food products have had ingredient labeling for many years, with ingredients listed in order by weight and now many perishable foods and commodities are labeled as well. These ingredient lists are helpful for people who want to include or avoid certain food ingredients in their diets for health or cultural reasons. New information is also available on labels now make it even easier for consumers.

Nutrition Facts Label

Nutrient labeling of processed foods was first made mandatory in 1993. The "Nutrition Facts" panel now provides information about the nutrient content of processed foods in standardized portion sizes. This label information can be used as a tool to help people select healthy diets. Today, the vast majority of processed foods and commodities, such as fresh fruits and vegetables, meats, poultry and fish, also have Nutrition Facts labels. The percentage of a standard population weighted value based on nutrient needs, called the USRDA, is used, which consists of the highest values for each adult age group by gender at the time when the label was promulgated. In addition to these labels, over-the-counter vitamin and mineral supplements have Supplement Facts that use a dietary standard known as the Daily Value (DV), which is also based on the Recommended Dietary Allowances, to provide a comparison of the content with this nutrient standard. Figure 2.5 presents the current Nutrition Facts label, and the recently proposed revision of the Nutrition Facts label that the Food and Drug Administration will finalize once comments are received and additional changes incorporated in 2016 (Fig. 2.5). There are several changes to reflect new public health and scientific information. Serving size requirements listed on the label would be updated to reflect the amounts of food people are actually eating and drinking now as opposed to 20 years ago when the Nutrition Facts label was

Amount Per Serv	ing	oul 8		Serving	rvings per containe size 2/3 cup (55g		
Calories 230	Cal	laries fron	n Fat 72		t per 2/3 cup		
		% Dall	y Value*	Cal	ories 230		
Total Fat 8g			12%				
Saturated Fa	£ 1g		5%	% DV*			
Trans Fat 0g				12%	Total Fat 8g		
Cholesterol) mg		0%	5%	Saturated Fat 1g		
Sodium 160m	g		7%		Trans Fat 0g		
Total Carbol	ydrate 37	'g	12%	0%	Cholesterol Omg		
Dietary Fiber	4g		16%	7%	ġ.		
Sugars 1g					Total Carbs 37g		
Protein 3g				14%			
				1975			
Vitamin A			10%		Sugars 1g		
Vitamin C			8%		Added Sugara 0g		
Calcium			20%		Protein 3g		
Iron			45%				
"Percent Daily Values are based on a 2,000 calorie dist.			10%	Vitamin D 2 mog			
Your daily value no your calorie needs.	s be higher or	lover depen	ding on	20%	Calcium 260 mg		
,	Cale iex	2.000	2,500	45%	Iron Sma		
Total Fat Sat Fat	Less than Less than	20g	80g 25g		Potassium 225 mg		
Cholesterol Sodium Total Carbohydrate Distary Fiber	Less than 300mg 300mg Less than 2,400mg 2,400mg 300g 375g 25g 30g		9,400mg 375g		te on Daily Values (DV) and calories ce to be inserted here.		

Fig. 2.5 Previous and proposed revision of nutrition facts food label

first introduced. The format of the label is also proposed to be refreshed, with key parts of the label, especially calories, as well as serving sizes, and percent daily value so that they are more prominent.

FDA has also proposed changes to the Nutrient Facts listing as well. These include:

Revised Daily Values for certain nutrients that are either mandatory or voluntary on the label Examples include calcium, sodium, dietary fiber, and vitamin D. Some Daily Values are intended to guide consumers about maximum intake – saturated fat, for example – while others are intended to help consumer meet a nutrient requirement – iron, for example. Daily Values are used to calculate the percent daily value (%DV) on the label, which helps consumers to understand the nutrient information on the product label in the context of the total diet. The revisions in Daily Values are based on recommendations published as the Dietary Reference Intakes by the Institute of Medicine and other reports such as the Dietary Guidelines for Americans. In addition to changing some Daily Values, FDA is also changing the units used to declare vitamins A, E, and D from "international units," or "I.U." to a metric measure – milligrams or micrograms. FDA is also proposing to include the absolute amounts in milligrams or micrograms of vitamins and minerals, in addition to the %DV, on the label.

Requiring the declaration of "Added Sugars" on the label "Sugars" include both "added sugars" and sugars that are naturally occurring in food. Americans on average eat 16% of their total calories from added sugars, the major sources being soda, energy and sports drinks, grain-based desserts, sugar-sweetened fruit drinks, dairy-based desserts, and candy. Currently, "Sugars" are required to be labeled

on packages, and if the proposal is finalized the declaration of "Added Sugars" will be indented and included under "Sugars" so that both would be listed. Although added sugars are not chemically different from naturally occurring sugars, many foods and beverages that are major sources of added sugars have lower micronutrient densities compared to foods and beverages that are major sources of naturally occurring sugars.

Removing the requirement for declaring "Calories from fat." Current research shows that the total fat in the diet is less important for chronic degenerative diseases like coronary heart disease than the type of fat. In addition, FDA consumer research shows that removal of the declaration of "calories from fat" has no effect on consumers' ability to judge the healthfulness of a product. FDA would continue to require "Total Fat," "Saturated Fat," and "*Trans* Fat" on the label.

Revising the nutrients of public health significance that must be declared on the label. These are nutrients that the US population is consuming in inadequate amounts, which are associated with the risk of chronic disease. Data from the National Health and Nutrition Examination Survey (NHANES) suggested that calcium, vitamin D, potassium, and iron should be mandatory. Calcium and iron are already required; vitamin D and potassium would be newly required. Vitamin D is important for its role in bone development and general health, and intakes among some population groups are inadequate. Adequate potassium intake is beneficial in lowering blood pressure and intakes of this nutrient are also low among some population groups. Mandatory labeling would no longer be required for vitamin C or vitamin A because current data indicate that deficiencies are not common. However, these vitamins would still be allowed to be declared on labels voluntarily.

The changes will likely be finalized in 2016 and appear on labels shortly thereafter.

Nutrient Content Claims

Nutrient content claims on food labels, such as "low in fat" or "good source of dietary fiber" are specified so that the same standard adjectives are used on all foods of the same type.

Structure–Function Claims

Over the last two decades, in addition to nutrient content claims, and structure–function claims and health claims have also been permitted on foods. Structure–function claims describe the association between a nutrient or other bioactive in food and a bodily function, such as "calcium builds bones."

Health Claims

Health claims describe the relationship between a nutrient or food and a disease. These provide additional information for consumers. Until 2003, only claims for which there was "significant scientific agreement" (SSA) were allowed on food labels. After 2003, the claims that were allowed were broadened to include claims for which there was less scientific agreement of the association. The highest level of evidence, the SSA claims, were designated as unqualified or "A" claims; they did not require disclaimers or qualifications. However, it was felt that for the claims lower down in the hierarchy, additional explanation, "qualifications" or disclaimers were needed to avoid misleading consumers. The other claims, B, C, and D, are called qualified health claims and they indicate a connection that is less certain than the unqualified claims.

Thus, the language on the label for the different levels of claim varies:

- A. "There is significant scientific agreement for [the claim]."
- B. "Although there is some scientific evidence supporting [the claim], the evidence is not conclusive."
- C. "Some scientific evidence suggests [the claim]. However, the F.D.A. has determined that this evidence is limited and not conclusive."
- D. "Very limited and preliminary scientific research suggests [the claim]. The F.D.A. concludes that there is little scientific evidence supporting this claim."

Figure 2.6 presents a "report card" that graphically highlights the different types of claims (Fig. 2.6). Some of the permissible SSA health claims for food packages are presented in (Table 2.5). All health claims for foods that are currently allowed, are found at the FDA web site, and they are updated as new claims become available.

Allergen Labeling

About 2% of adults and about 5% of infants and young children in the United States suffer from food allergies. Approximately 30,000 consumers require emergency room treatment and 150 Americans die each year because of allergic reactions to food. The Food Allergen Labeling and Consumer Protection Act (FALCPA) of 2004 is an amendment to the Federal Food, Drug, and Cosmetic Act. It requires that the label of a food that contains an ingredient or contains protein from

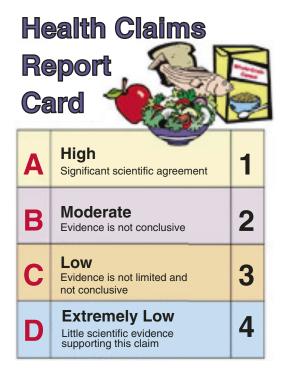


Fig. 2.6 "Report Card" for health claims on foods

Health claim	Example of some statements
Osteoporosis Calcium, vitamin D, and osteoporosis	A calcium-rich diet is linked to a reduced risk of osteoporosis, a condition in which the bones become soft or brittle
Heart Disease Dietary saturated fat and cholesterol and risk of coronary heart disease	A diet low in saturated fat and cholesterol can help reduce the risk of heart disease
Fiber-containing grain products, fruits, and vegetables, and cancer	A diet rich in high-fiber grain products, fruits, and vegetables can reduce the risk of some cancers
Soluble fiber from certain foods and risk of coronary heart disease	
Soy protein and risk of coronary heart disease	
Stanols/sterols and risk of coronary heart disease Substitution of saturated fat with unsaturated fatty acids and risk of heart disease	
Whole grain foods and risk of heart disease and certain cancers	
Nuts and heart disease ^a	
Walnuts and heart disease ^a	
Omega 3 fatty acids and coronary heart disease ^a	
Unsaturated fatty acids from canola oil and coronary heart disease ^a	
Corn oil and heart disease ^a	
B vitamins and vascular disease ^a	
Monounsaturated fatty acids from olive oil and coronary heart disease ^a	
Unsaturated fatty acids from canola oil and coronary heart disease	
Corn oil and heart disease	
<i>Dental caries</i> Fluoride and the risk of dental caries	
Dietary non-cariogenic carbohydrate sweeteners and dental caries	
High blood pressure	
Sodium and hypertension Potassium and the risk of high blood pressure and stroke	
Calcium and hypertension, pregnancy-induced hypertension, and pre-eclampsia ^a	
Cancer	A diet low in total fat is linked to a reduced risk of some
Dietary lipids (fat) and cancer	cancers
Fruits and vegetables and cancer	A diet high in fruits and vegetables and foods that are low in fat and may contain dietary fiber, vitamin A, or vitamin C is linked to lower risk of some cancers.
Whole grain foods and risk of heart disease and certain cancers	
Tomatoes and/or tomato sauce and prostate, ovarian, gastric, and pancreatic cancers ^a	
Calcium and colon/rectal cancer and calcium and recurrent colon/rectal polyps ^a	
Green tea and cancer ^a	
Selenium and cancer ^a	
Antioxidant vitamins and cancer ^a	

Table 2.5	Permissible	health claims	for fo	ood packages ^a
-----------	-------------	---------------	--------	---------------------------

Table 2.5	(continued)
-----------	-------------

Health claim	Example of some statements
Other	
Choline	
100% whey protein partially hydrolyzed infant formula and reduced risk of atopic dermatitis ^a	
Chromium picolinate and diabetes ^a	
Phosphatidylserine and cognitive ^a dysfunction and dementia ^a	
0.8 mg folic acid and neural tube birth defects ^a	
^a Claims with an asterisk are qualified health claims subj	ect to enforcement discretion. These refer to claims when there

^aClaims with an asterisk are qualified health claims subject to enforcement discretion. These refer to claims when there is emerging evidence for a relationship between a food, food component, or dietary supplement, and reduced risk of a disease or health-related condition, but the evidence is not established well enough to meet significant scientific agreement standard, and so qualifying language is needed

a "major food allergen" declare its presence. FALCPA identifies eight foods or food groups as the major food allergens that account for about 90% of food allergies: milk, eggs, fish (e.g., bass, flounder, cod), Crustacean shellfish (e.g., crab, lobster, shrimp), tree nuts (e.g., almonds, walnuts, pecans), peanuts, wheat, and soybeans. People can be allergic to other foods. In fact, more than 160 foods have been identified to cause food allergies in sensitive individuals. However, those eight account for most of the food allergies and also are the ingredients most likely to result in severe or life-threatening reactions. In addition to allergies, some people suffer from other intolerances or sensitivities from one or more ingredients, such as lactose. Some of these can be identified on the ingredient list on the label.

Front of Pack Labeling

FDA's consumer research has found that people are less likely to check the Nutrition Facts label on the back or side panel of foods with front-of-pack labeling. Therefore, it is essential that both the criteria and symbols used in front of package and shelf-labeling systems help consumers make healthy food choices. Research results are still not clear on how consumers use and understand various types of front of pack labeling and shelf-tag systems, including those that use one symbol to summarize nutritional attributes and systems that feature or rate foods as being low, medium, or high in specific nutrients, similar to the traffic light system in the United Kingdom. A system of voluntary front-of-pack labeling has been adopted by many of the large food manufacturers in the United States over the past few years. The public health benefit of such labeling, or whether the Food and Drug Administration will ultimately view it as a public health benefit remains to be determined. It avoids the "traffic light" approach, which some interpret as green (good to go), yellow (caution), and red (avoid or stop), That stigmatizes foods as "good" or "bad," "healthy" or "unhealthy"; such terms mean little without consideration of how foods fit into the individual's needs and the total dietary pattern.

Menu Labeling in Restaurants and Vending Machines

The Food and Drug Administration has recently finalized two rules requiring that calorie information be listed on menus and menu boards in chain restaurants and similar retail food establishments and vending machines, and these are now beginning to appear. The hope is that such information will drive consumer choices toward lower calorie options.

Other Labeling Terms

There are many other terms used on packaged and other foods in the United States. Some standards, such as the "certified organic" label supervised by the US Department of Agriculture, are legally enforceable. Others are not. Below is a brief summary of their current status, starting with those that have more well-defined regulatory standards.

"Organic" Labels

Organic food products are not necessarily more healthful, sanitary, or nutritious than nonorganic products, but some consumers prefer them and are willing to pay more to obtain them. It should be noted before discussing organic foods that neither genetically engineered foods, and other foods or ingredients such as spices that are processed with ionizing radiation are unsafe. Moreover, there are regulations governing their use. The USDA regulates the use of the terms organic and certified organic on food labels. Raw or processed agricultural products in the "organic" category must be produced without excluded methods (genetic engineering), ionizing radiation, or sewage sludge, and produced in line with a national list of allowed and prohibited substances. The program is overseen by an authorized certifying agent of the USDA's National Organic Program, and all of the USDA organic regulations must be followed. For products that are labeled "100% organic," all agricultural ingredients must be certified organic, except where specified on a national list of ingredients that is issued by USDA. Nonorganic ingredients allowed on the national list may be used, up to a combined total of 5% of nonorganic content (excluding salt and water). The product labels must state the name of the certifying agent on the information panel, the organic ingredients must be identified, and all ingredients must be certified organic, and processing aids must be organic. Products meeting these criteria may use the USDA organic seal and/or organic claim.

"Non-GMO" Labels

Evidence is lacking that foods made with bioengineered or genetically modified ingredients are unsafe, unsanitary, less nutritious, or less healthful in other ways. These bioengineered foods or ingredients are sometimes incorrectly called "genetically modified organisms" [GMO]. This is a misnomer since the ingredients are not microorganisms, with the only exception of, perhaps, genetically engineered microorganisms in some yoghurts. The foods are not alive, nor are they living organisms. Laws in several states require that bioengineered (genetically modified) foods be labeled on a compulsory basis. In contrast, The Food and Drug Administration (FDA) asserts that while voluntary labeling is not prohibited, it is unnecessary to mandate that foods that contain genetically engineered ingredients be labeled. FDA supports voluntary labeling, and it is expected to issue guidance on that soon. The FDA position is that mandatory labeling is appropriate and required when there is a faulty claim or misbranding, but because a food contains genetically engineered ingredients does not constitute a material change in the product, and therefore labeling is not required. That is, GMO corn is similar to other varieties and species of corn in all major constituents. This interpretation has been supported by the courts. However, voluntary statements are permissible, such as:

- "We do not use ingredients that were produced using biotechnology"
- "This oil is made from soybeans that were not genetically engineered"
- "Our tomato growers do not plant seeds developed using biotechnology"

A statement that a food was not bioengineered or does not contain bioengineered ingredients may be misleading if it implies that the labeled food is superior to foods that are not. FDA has concluded that the use or absence of use of bioengineering in the production of a food or ingredient does not, in and of itself, mean that there is a material difference in the food. Therefore, a label statement that expresses or implies that a food is superior (e.g., safer or of higher quality or superior or more nutritious) because it is not bioengineered would be misleading.

Gluten-Free Labeling

Hundreds of thousands and perhaps as many as three million Americans suffer from celiac disease, or gluten enteropathy, an autoimmune digestive condition that can be effectively managed only by eating a gluten-free diet, and millions more do not, but believe they have the disease. In 2013, the Food and Drug Administration issued regulations defining the meaning of "gluten-free" and specifying what food products could be labeled as gluten-free. In order to use the term "gluten-free" on its label, a food must contain less than 20 ppm of gluten. The rule also requires foods with the claims "no gluten," "free of gluten," and "without gluten" to meet the definition for "gluten-free."

Whole Grain Labeling

Many Amercians eat far less than half of their servings of grains from whole grains, and so many are looking to choose products high in them. The US government does not have an official standard for whole grain labeling, but it prohibits false and misleading advertising. The word "whole grain" in the name of a product should be used only if the product contains more whole grain than refined grain (i.e., 51% or more of the grain is whole grain). A full serving of whole grain contains about 16 g whole grain. According to this logic, products containing 8 or more grams of whole grain can still be labeled whole grain. The Whole Grains Council is a private group that has created an official packaging symbol called the Whole Grain Stamp that appears on labels to help consumers find whole grain products. With the stamp, three servings of whole grains consists of three foods with the 100% Stamp or six food servings of food products with any Whole Grain Stamp. The 100% Stamp assures the consumer that all the grain is whole grain, while the basic Whole Grain Stamp appears on products containing at least half a serving of whole grain per labeled serving. If there is no stamp, the label may list the grams of whole grain, or claim the product is 100% whole grain. If the ingredient list on the package label lists whole grain first, this may also indicate a product high in whole grains. The goal is to increase whole grain intake, regardless of the source. It should be noted that whole grains are not fortified with folic acid, and so variety is important to obtain all the nutrients needed per day, and some fortified grain products should be eaten as well.

"Natural" Labeling

From a food science perspective, it is difficult to define a food product that is "natural" because the food has probably been processed and is no longer the product of the earth. The FDA has not developed a definition for use of the term natural or its derivatives. However, the agency has not objected to the use of the term if the food does not contain added color, artificial flavors, or synthetic substances. Evidence is lacking that those substances are unsafe or harmful to health, although some consumers may wish to avoid such ingredients for aesthetic or other personal preferences.

Supermarket Icons and Scoring Systems

Many supermarkets and other food retailers have adopted various systems of their own for labeling foods they consider to be particularly healthful or desirable. The criteria for them vary. These are not endorsed or regulated by the Federal government either. Some are relatively straightforward and easy to discover, while others rely on proprietary formulas, some of which are of dubious validity, and other data that are not readily available to consumers to rate the products. At present, the most objective, reliable, and transparent sources of information are the Nutrition Facts label and ingredient lists.

Healthy People

Healthy People 2020 is the health promotion and disease prevention plan issued every decade by the US Department of Health and Human Services. In the early 1970s, the need for explicit, measurable, prevention objectives for government, private, and voluntary groups became increasingly necessary. The Office of the Assistant Secretary for Health in the then US Department of Health, Education, and Welfare (now the US Department of Health and Human Services) began the process of developing consensus on some national goals that could guide prevention efforts in the future. The first effort was a slim volume entitled Promoting Health, Preventing Disease: Objectives for the Nation [68]. It stated general goals and objectives in various areas of public health and preventive medicine and ways to measure progress in achieving them. In the late 1970s, US Surgeon General Julius Richmond, who was also Assistant Secretary for Health, commissioned a much larger effort, entitled Healthy People that set forth an ambitious prevention plan [69]. Using a management by objectives planning process, the US Public Health Service set out objectives focusing on improving health status, risk reduction, public and professional awareness of prevention, health services, and protective measures, surveillance, and evaluation. The objectives were organized in 15 priority areas under the general headings of preventive services, health protection, and health promotion. Targets for achieving the objectives were set; usually with a 10-year time frame. It was recognized that in order to achieve the objectives, a health system reaching all Americans and integrating personal health care and public health measures focusing on the entire population (population-based measures) would be necessary. Moreover, it was clear that most prevention did not occur solely within the health system, but in community institutions as well, including schools, workplaces, families, and neighborhoods. Therefore, these environments as well as the traditional health care system were included as a focus for the preventive efforts.

Starting in 1980, and at the beginning of every decade thereafter, HHS has convened public, private, and voluntary groups to update the plan and assess progress toward achieving it. The resulting document is called Healthy People. It is now customary to issue a "mid-course review" halfway through the decade to make midterm corrections and redirect resources if this should prove to be necessary.

The current prevention plan is entitled "Healthy People 2020." The entire report is hundreds of pages long and accessible on the Internet at: http://www.healthypeople.gov/2020/tools-and-resources/ Federal-Prevention-Initiatives.

The overall goals of Healthy People are simple: Increase the span of healthy life, reduce health disparities among Americans, and achieve access to preventive services for all Americans. For each life stage, there are specific objectives stated in terms of reducing mortality and in the various areas. Goals are also set for each priority area. Nutrition is considered as one of these areas. They deal with

health status, risk reduction, and services-related issues and set measurable objectives in each area. The nutrition-related goals for Healthy People 2020 are discussed in detail elsewhere [70].

For some objectives, the 2010 nutrition targets have been met. But for other objectives, such as obesity, little progress has been made or progress has been in the wrong direction. The 2020 goals reflect these problems. These are the areas where the nation must concentrate its efforts in the future.

Future Research

The Federal government has recently issued a Roadmap for National Nutrition Research from 2016 to 2021 [71]. It tackles three critical questions that provide a rationale and guide to future efforts: how to better understand and define eating patterns to improve and sustain health; what can be done to help Americans choose healthy eating patterns; and how to accelerate discoveries in human nutrition. It lays out a clear rationale and useful research that will speed the process of converting basic and clinical studies into science-based nutrition policy.

Conclusions

Dietary intake is only one facet of nutritional status, but it is an important one. The Dietary Guidelines for Americans provide recommendations for altering current intakes in more healthful directions. The Dietary Reference Intakes, such as the RDA and UL provide useful guides for planning nutrient intakes for individuals and groups. Other tools, such as MyPyramid and its successor, ChooseMyPlate, are helpful food-based recommendations that, if followed, ensure that the DRI are met. Food labels aid in making wise choices in line with dietary recommendations. The Healthy Eating Index provides a simple tool for evaluating the balance, variety, and adequacy of intakes based on the USDA Food Guide and Dietary Guidelines for Americans. Health professionals can use these simple tools to plan and assess their own eating patterns and those of their clients. For many practical purposes, these tools will suffice. For more elaborate planning and assessment and for research, other tools may be necessary. The Year 2020 nutritional goals for promoting health and preventing disease provide additional useful health targets to aim for as a nation.

Acknowledgment This project has been funded at least in part with Federal funds from the National Institutes of Health and also the US Department of Agriculture, Agricultural Research Service, under contract number 53/3-K06-5-10. The contents of this article do not necessarily reflect the views or policies of the US Department of Agriculture, nor do they mention trade names, commercial products, or organizations that imply endorsement by the United States government. April 1 2016.

Bibliography

- 1. van Staveren W, Ocke M. Estimation of dietary intake. In: Russell R, Bowman B, editors. Present knowledge in nutrition. 2. Washington, DC: ILSI Press; 2003. p. 795–806.
- 2. Thompson FE, Byers T. Dietary assessment resource manual. J Nutr. 1994;124(11 Suppl):2245S-317.
- 3. Campbell VA, Dodds ML. Collecting dietary information from groups of older people. J Am Diet Assoc. 1967;51(1):29–33.
- Marr JW, Heady JA. Within- and between-person variation in dietary surveys: number of days needed to classify individuals. Hum Nutr Appl Nutr. 1986;40(5):347–64.

- Heady JA. Diets of bank clerks. Deelopment of a method of classifying the diets of individuals for use in epidemiologic studies. J R Statist Soc. 1961;124:336–61.
- Subar AF, Kipnis V, Troiano RP, Midthune D, Schoeller DA, Bingham S, et al. Using intake biomarkers to evaluate the extent of dietary misreporting in a large sample of adults: the OPEN study. Am J Epidemiol. 2003; 158(1):1–13.
- 7. National Cancer Institute. The measurement error Webinar Series. http://riskfactor.cancer.gov/measurementerror/. 2011. Available from: http://riskfactor.cancer.gov/measurementerror/.
- 8. National Research Council. Nutrient adequacy. Washington, DC: National Academy Press; 1986.
- Nusser SM, Carriquiry AL, Dodd KW, Fuller WA. A semiparametric transformation approach to estimating usual daily intake distributions. J Am Stat Assoc. 1996;91:1440–9.
- Subar AF, Dodd KW, Guenther PM, Kipnis V, Midthune D, McDowell M, et al. The food propensity questionnaire: concept, development, and validation for use as a covariate in a model to estimate usual food intake. J Am Diet Assoc. 2006;106(10):1556–63.
- Tooze JA, Midthune D, Dodd KW, Freedman LS, Krebs-Smith SM, Subar AF, et al. A new statistical method for estimating the usual intake of episodically consumed foods with application to their distribution. J Am Diet Assoc. 2006;106(10):1575–87.
- Dodd KW, Guenther PM, Freedman LS, Subar AF, Kipnis V, Midthune D, et al. Statistical methods for estimating usual intake of nutrients and foods: a review of the theory. J Am Diet Assoc. 2006;106(10):1640–50.
- 13. Carriquiry AL. Estimation of usual intake distributions of nutrients and foods. J Nutr. 2003;133(2):601S-8.
- Dwyer J, Costello RB. Assessment of dietary supplement use. In: Coulston A, Boushey C, editors. Nutrition in the prevention of disease. 2nd ed. Burlington: Academic; 2008. p. 41–56.
- Lindenbaum J, Savage DG, Stabler SP, Allen RH. Diagnosis of cobalamin deficiency: II. Relative sensitivities of serum cobalamin, methylmalonic acid, and total homocysteine concentrations. Am J Hematol. 1990;34(2): 99–107.
- Carmel R, Green R, Rosenblatt DS, Watkins D. Update on cobalamin, folate, and homocysteine. Hematol Edu Prog Am Soc Hematol Am Soc Hematol. 2003;1:62–81.
- Pennypacker LC, Allen RH, Kelly JP, Matthews LM, Grigsby J, Kaye K, et al. High prevalence of cobalamin deficiency in elderly outpatients. J Am Geriatr Soc. 1992;40(12):1197–204.
- Green R, Miller JW. Vitamin B12 deficiency is the dominant nutritional cause of hyperhomocysteinemia in a folic acid-fortified population. Clin Chem Lab Med. 2005;43(10):1048–51.
- Raiten DJ, Namaste S, Brabin B, Combs Jr G, L'Abbe MR, Wasantwisut E, et al. Executive summary: biomarkers of nutrition for development: building a consensus. Am J Clin Nutr. 2011;94(2):633S–50.
- Murphy S. Dietary standards in the United States. In: Bowman B, Russell R, editors. Present knowledge in nutrition. 2. Washington, DC: ILSI Press; 2006. p. 859–75.
- 21. Dietary Guidelines Advisory Committee. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010, to the Secretary of Agriculture and the Secretary of Health and Human Services. Washington, DC: Agricultural Research Service US Department of Agriculture; 2010.
- Institute of Medicine Food Nutrition Board. How should the recommended dietary allowances be revised? Washington, DC: National Academies Press; 1994.
- Institute of Medicine Food Nutrition Board. Dietary reference intakes: a risk assessment model for establishing upper intake levels for nutrients. Washington, DC: National Academies Press; 1998.
- 24. Institute of Medicine Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. Washington, DC: National Academies Press; 1997.
- 25. Institute of Medicine Standing Committee on the Scientific Evaluation of Dietary Reference Intakes Panel on Folate Other B. Vitamins Choline. Dietary reference intakes for thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin, and choline. Washington, DC: National Academies Press; 1998.
- 26. Institute of Medicine Standing Committee on the Scientific Evaluation of Dietary Reference Intakes Panel on Dietary Reference Intakes for Electrolytes and Water. Dietary reference intakes for water, potassium, sodium, chloride, and sulfate. Washington, DC: National Academies Press; 2005.
- 27. Institute of Medicine Standing Committee on the Scientific Evaluation of Dietary Reference Intakes Panel on Macronutrients. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (Macronutrients). Washington, DC: National Academies Press; 2005.
- Dwyer J. Nutritional requirements and dietary assessment. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, et al., editors. Harrison's principles of internal medicine. 17th ed. New York: McGraw-Hill Medical; 2008. p. 449.
- 29. Food and Nutrition Board, Institute of Medicine Dietary Reference Intakes Tables and Applications. https://fnic.nal.usda.gov.
- Fulgoni VL, Keast DR, Bailey RL, Dwyer J. Foods, fortificants, and supplements: where do Americans get their nutrients? J Nutr. 2011;141(10):1847–54.

- World Health Organization. A model for establishing upper levels of intake for nutrients and related substances: a report of a joint FAO/WHO technical workshop on food nutrient risk assessment. Geneva: World Health Organization; 2006.
- 32. Institute of Medicine Committee to Review Dietary Reference Intakes for Vitamin D Calcium. Dietary reference intakes for calcium and vitamin D. In: Ross AC, Taylor CL, Yaktine AL, Del Valle HB, editors. Dietary reference intakes for calcium and vitamin D. Washington, DC: National Academies Press; 2011.
- 33. Institute of Medicine Panel on Dietary Antioxidants Related Compounds. Dietary reference intakes for vitamin C, vitamin E, selenium, and carotenoids. Washington, DC: National Academies Press; 2000.
- 34. Institute of Medicine Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Dietary reference intakes: applications in dietary planning. Washington, DC: National Academies Press; 2003.
- Institute of Medicine Food Nutrition Board. Dietary reference intakes: the essential guide to nutrient requirements. Washington, DC: National Academies Press; 2006.
- Institute of Medicine Food and Nutrition Board. The development of DRIs 1994–2004: lessons learned and new challenges. Workshop summary. Washington, DC: National Academies Press; 2008.
- Lichtenstein A, Yetley E, Lau J. Application of systematic review methodology to the field of nutrition (Prepared by the Tufts Evidence-based Practice Center under Contract No. 290-02-0022). Rockville: Agency for Healthcare Research and Quality; 2009.
- 38. Russell R, Chung M, Balk EM, Atkinson S, Giovannucci EL, Ip S, et al. AHRQ technical reviews. Issues and challenges in conducting systematic reviews to support development of nutrient reference values: workshop summary: nutrition research series, vol. 2. Rockville: Agency for Healthcare Research and Quality (US); 2009.
- 39. Chung M, Balk E, Ip S, Raman G, Yu W, Trikalinos T, et al. Reporting of systematic reviews of micronutrients and health: a critical appraisal (Prepared by the Tufts Evidence-based PracticeCenter under Contract No. 290-02-0022). Rockville: Agency for Healthcare Reserach and Quality; 2009.
- Moher D, Tricco AC. Issues related to the conduct of systematic reviews: a focus on the nutrition field. Am J Clin Nutr. 2008;88(5):1191–9.
- 41. Chung M, Balk EM, Ip S, Lee J, Terasawa T, Raman G, et al. Systematic review to support the development of nutrient reference intake values: challenges and solutions. Am J Clin Nutr. 2010;92(2):273–6.
- 42. World Cancer Research Foundation/American Institute for Cancer Research. Food, nutrition, physical activity and the prevention of cancer: a global perspective. Washington, DC: American Institute of Cancer Research; 2007.
- 43. Food and Nutrition Board. Dietary reference intakes applications in dietary assessment. Washington, DC: National Academy Press; 2000.
- 44. Food and N, Gaine PC, Balantine DA, Erdaman JW, Dwyer JT, Ellwood KC, Hu FB, Russell R. Are dietary bioactive ready for recommended intakes? Adv Nutr. 2013;4:539041. doi:10.2945/an./13.004226. Utrition Board. The Development of the Dietary Reference Intakes 1994–2004: Lessons Learned. Washington, DC: National Academy Press; 2008.
- 45. US Department of Agriculture and US Department of Health and Human Services. Dietary guidelines for Americans 2015–2020. Washington, DC: US Government Printing Office; 2016.
- 46. Davis C, Saltos E. Dietary recommendations and how they have changed over time. In: Economic Research Service, editor. America's eating habits: changes and consequences. Washington, DC: US Department of Agriculture; 2009. p. 33–50.
- Hayes Polon R, Essery Stoody E. Dietary guidelines for Americans 2010: national nutrition policy. In: Rippe J, editor. Lifestyle medicine. Boca Raton: CRC Press; 2010. p. 131–47.
- Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997;336(16):1117–24.
- Miraglia M, Dwyer J. Dietary recommendations for primary prevention: an update. Am J Lifestyle Med. 2011: 5; 144–55.
- Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Updated US Department of Agriculture Food Patterns meet goals of the 2010 dietary guidelines. J Acad Nutr Diet. 2012;112(10):1648–55.
- McCullough ML, Willett WC. Evaluating adherence to recommended diets in adults: the alternate healthy eating index. Public Health Nutr. 2006;9(1A):152–7.
- 52. Akbaraly TN, Ferrie JE, Berr C, Brunner EJ, Head J, Marmot MG, et al. Alternative healthy eating index and mortality over 18 y of follow-up: results from the Whitehall II cohort. Am J Clin Nutr. 2011;94(1):247–53.
- Fogli-Cawley JJ, Dwyer JT, Saltzman E, McCullough ML, Troy LM, Jacques PF. The 2005 Dietary Guidelines for Americans Adherence Index: development and application. J Nutr. 2006;136(11):2908015.
- 54. Reedy J, Krebs-Smith SM. A comparison of food-based recommendations and nutrient values of three food guides: USDA's MyPyramid, NHLBI's dietary approaches to stop hypertension eating plan, and Harvard's healthy eating pyramid. J Am Diet Assoc. 2008;108(3):522–8.
- Britten P, Cleveland LE, Koegel KL, Kuczynski KJ, Nickols-Richardson SM. Impact of typical rather than nutrientdense food choices in the US Department of Agriculture Food Patterns. J Acad Nutr Diet. 2012;112(10):1560–9.

- Krebs-Smith SM, Guenther PM, Subar AF, Kirkpatrick SI, Dodd KW. Americans do not meet federal dietary recommendations. J Nutr. 2010;140(10):1832–8.
- 57. Bachman JL, Reedy J, Subar AF, Krebs-Smith SM. Sources of food group intakes among the US population, 2001–2002. J Am Diet Assoc. 2008;108(5):804–14.
- Miller GD, Drewnowski A, Fulgoni V, Heaney RP, King J, Kennedy E. It is time for a positive approach to dietary guidance using nutrient density as a basic principle. J Nutr. 2009;139(6):1198–202.
- Fulgoni VL, Keast DR, Drewnowski A. Development and validation of the nutrient-rich foods index: a tool to measure nutritional quality of foods. J Nutr. 2009;139(8):1549–54.
- 60. US Department of Health and Human Services and US Department of Agriculture Choose MyPlate 2011. Available from: http://www.choosemyplate.gov/.
- Kennedy ET, Ohls J, Carlson S, Fleming K. The healthy eating index: design and applications. J Am Diet Assoc. 1995;95(10):1103–8.
- Variyam J, Blaylock J, Smallwood D, Basiotis P. USDA's healthy eating index and nutrition information. Washington, DC: Economic Research Service US Department of Agriculture; 1998. Contract No.: Technical Bulletin No 1866.
- Guenther PM, Casavale KO, Reedy J, Kirkpatrick SI, Hiza HA, Kuczynski KJ, et al. Update of the healthy eating index: HEI-2010. J Acad Nutr Diet. 2013;113(4):569–80.
- 64. Guenther PM, Kirkpatrick SI, Reedy J, Krebs-Smith SM, Buckman DW, Dodd KW, et al. The healthy eating index-2010 is a valid and reliable measure of diet quality according to the 2010 Dietary Guidelines for Americans. J Nutr. 2014;144(3):399–407.
- Guenther PM, Lyon JM, Appel LJ. Modeling dietary patterns to assess sodium recommendations for nutrient adequacy. Am J Clin Nutr. 2013;97(4):842–7.
- 66. Miller PE, Reedy J, Kirkpatrick SI, Krebs-Smith SM. The United States food supply is not consistent with dietary guidance: evidence from an evaluation using the healthy eating index-2010. J Acad Nutr Diet. 2015;115(1): 95–100.
- Rehm CD, Monsivais P, Drewnowski A. Relation between diet cost and healthy eating index 2010 scores among adults in the United States 2007–2010. Prev Med. 2015;73:70–5.
- 68. Office of the Assistant Secretary for Health. Promoting health, preventing disease: objective for the nation. Washington, DC: US Department of Health and Human Services; 1976.
- 69. US Department of Health and Human Services. Healthy people: the surgeon general's report on health promotion and disease prevention. Washington, DC: US Department of Health and Human Services; 1980.
- Starke-Reed P, McDade-Ngutter C, Hubbard V. Healthy people 2020: highlights in the nutrition and weight status focus area. In: Rippe J, editor. Encyclopedia of lifestyle medicine and health. New York: CRC Press; 2013. p. 119–30.
- Interagency Committee on Human Nutrition Research. National nutrition research roadmap 2016–20121. Advancing nutrition research to improve and sustain health. Washington, DC: Interagency Committee on Human Nutrition Research; 2016.

Chapter 3 Behavior Change and Nutrition Counseling

Elizabeth Pegg Frates and Jonathan Bonnet

Key Points

- Nutrition counseling involves more than advising a patient on what to eat and what not to eat.
- The messages must be evidence-based, clear, and consistent.
- The most effective way for the messenger to deliver these healthy eating messages is to use collaboration and negotiation to co-create a nutrition plan with the patient.
- The "COACH" approach (Curiosity, Openness, Appreciation, Compassion, and Honesty) helps cultivate a therapeutic relationship and creates an environment that promotes healthy eating patterns and empowers patients.
- Tapping into the patient's motivation is a critical part of the nutrition counseling process.
- Motivational interviewing, self-determination theory, appreciative inquiry, and the transtheoretical model of change can help guide clinicians to elicit patient motivation and to facilitate change.
- Two often-overlooked nutrition strategies to propel patients forward toward healthy eating patterns are discovering the patient's vision for himself or herself and determining the differential between where the patient is now, and where he or she wants to go.
- Creating a new program and crafting "SMART" goals to adopt or sustain healthy eating habits need to be completed with each patient.
- Nutrition counseling is a journey for both patient and provider that needs to be personalized to each patient at each visit.

Keywords Behavior • Coach • Stage of change • Motivation • Empathy • Counseling

E.P. Frates, MD ()

J. Bonnet, MD Community Health and Family Medicine, University of Florida College of Medicine, Gainesville, FL 32610, USA e-mail: jonathanbonnet@gmail.com

Department of Physical Medicine and Rehabilitation, Harvard Medical School, Boston, MA 02115, USA e-mail: Efrates1@partners.org

Abbreviations

COACH	Curiosity, openness, appreciation, compassion, honesty		
EXPERT	Examine, X-ray, plan, explain, repeat and review, tell and sell		
GROW	Goals, reality, options, way		
MI	Motivational interviewing		
MINT	Motivational interviewing network of trainers		
MOSS	Motivators, obstacles, strategies, strengths		
OARS	Open-ended, affirmations, reflections, summaries		
SDT	Self-Determination Theory		
SMART	Specific, measurable, action-oriented, realistic, time-sensitive		
VIA-IS	Values in action inventory of strengths		

Introduction

The Behavior Change Journey

The process of changing eating patterns is one that can be challenging for health providers and patients alike. However, adopting healthy habits around food provides significant benefits for individuals, communities, and health care systems. The potential rewards of effectively counseling patients about nutrition include improved hemoglobin A1c, decreased blood pressure, improved weight management, increased self-confidence, and increased personal satisfaction. Whenever healthy habits are the subject of a research project, diet is one of the main components of the investigation. For example, the Nurses' Health Study [1] examined five healthy factors: (1) maintaining a healthy BMI, (2) exercising regularly, (3) eating a healthy diet, (4) not smoking, and (5) drinking alcohol only in moderation.

Two of these factors are directly related to nutrition, and all are interrelated. Maintaining a healthy BMI requires an individual to eat healthy foods in proper proportions and incorporate movement into daily life. Exercising has been shown to decrease appetite in some individuals [2] and this can help control the intake of unhealthy foods. Drinking alcohol can lead to the intake of excess calories. Smoking is also associated with alcohol consumption. People smoke to relieve stress and to control their weight. Participating in one unhealthy habit can prompt a person to develop other harmful behaviors. It works the other way as well. Patients adopting one healthy habit are more likely to adopt another. Smokers who start exercising are more likely to quit or cut down [3]. Thus, the five healthy factors from the Nurse's study are all part of a health-promoting lifestyle. In combination, they can work synergistically to enhance health and wellness.

The healthy diet factor can be one of the most confusing and yet one of the most powerful contributors to our overall health and well-being. Typical research protocols, such as the Food Frequency Questionnaire from the Women's Health study [4], look at the number of healthy foods consumed in a week. Many clinicians use the simple question "How many vegetables do you consume in a day?" as a crude assessment tool to gauge the quality of someone's diet. To begin a nutrition counseling session, it is important to understand the patient's current dietary intake and patterns. From there, sharing nutrition information, appropriate to that particular patient, can be useful and productive.

When counseling a patient on diet changes, using collaboration and negotiation is the key to unlocking sustainable behavior change. Perspective is essential as well. Patients spend a short amount of time in a health provider's office. The majority of patients' decisions, influences, and challenges occur outside of the walls of a clinic and are largely influenced by their family, friends, work, and community. Understanding this will enable the health care provider to go beyond simply advising patients and move toward cultivating an environment that brings out each patient's intrinsic motivators for incorporating healthy nutrition choices into the fabric of their lives. Allowing the patient to have the time and space to envision a healthy eating pattern, appreciate the difference between their current situation and the one they are striving to reach, voice reasons for changing, and strategize around their obstacles to change are all important parts of a nutrition counseling session.

Crafting goals that are relevant to that particular patient's life and are in line with their values, their vision of themselves, their sense of purpose in life, and their current priorities will set the patient up for long-term success. *SMART* goals that are *specific, measurable, action-oriented, realistic, time-sensitive*, and are aligned with the person's life goals will be the goals that the patient will be motivated to accomplish in the short and long term. SMART goals set the patient up for success, and success breeds success. Finally, putting an aspect of accountability into place with the patient is essential. This accountability will ensure that the goals, which are co-created during the consultation, will be taken seriously and that the patient will take responsibility for accomplishing them. Accountability is a vital component in the behavior change process. When the nutrition counseling process is full of *Curiosity, Openness, Appreciation, Compassion, and Honesty* (the *COACH Approach*), the journey to sustainable change can be a joyful one for both provider and patient [5, 6].

Both the message and the messenger are important in counseling on nutrition in lifestyle medicine. If the behavior change process is represented as an arch, then the message and the messenger are the cornerstones, the foundational pieces, without which the whole arch will collapse. Thus, the message needs to be evidence-based, clear, understandable, easy to follow, and consistent. The messenger needs to supply this message in a supportive, collaborative COACH approach style in order for the patient to accept, digest, embrace, and embody the nutrition messages. Specific counseling skills and tools have been studied and tested in randomized controlled trials such as motivational interviewing and health coaching [6, 7]. Thus, practicing evidence-based skills and using proven tools will help the messenger promote the message.

In addition to the foundational parts of the message and the messenger, the patient's motivation is the indispensable keystone to the arch of behavior change (Fig. 3.1). Tapping into patients' internal motivators and making sure their needs for autonomy, competence, and connection are satisfied will be the difference between a patient knowing what to do and a patient actually doing what he knows is healthy for his body and mind. The self-determination theory posed by two psychologists, Richard Ryan, PhD and Edward Deci, PhD, helps to guide this important aspect of nutrition counseling. This theory demonstrates the significance of the three components for volitional motivation; autonomy, competence, and connection [8]. Researchers have studied the intention behavior gap, meaning why people intend to do certain behaviors, but they do not end up actually doing them [9]. Clearly, it is not just a matter of telling a patient what to do. It is not just about the message. The process of behavior change is more complex and intricate than that. Like creating the perfect arch that invites people to enter a beautiful cathedral or building, creating the environment that invites people to take a step toward improved diet, health, and inner peace, requires two cornerstones (the message and the



Fig. 3.1 Behavior change arch

messenger) and a keystone (the patient motivation). In this chapter, we will dive deeply into this behavior change archway.

Many researchers have acknowledged the complexity and multifactorial nature of behavior change. Psychologist BJ Fogg, promotes the idea of Tiny Steps. These steps are built on the acknowledgment of three distinct areas, and he advocates for work in these areas with patients desiring and or needing change [10]. The areas include: (1) the capacity or ability to perform the behavior, (2) the opportunity to perform the behavior, and (3) the motivation to perform the behavior. According to Dr. Fogg, all three of these conditions need to be satisfied in order for a person to complete a certain behavior such as eating a healthy lunch at work. This takes into account the individual's competence (ability), motivation, and environment around the patient (enabling or obstructing) with respect to the desired behavior. The idea of opportunity to perform a particular is behavior derived from the social ecological model of change [10]. In this model, the individual is at the center of a series of circles, each of which grows larger as it gets further from the center. The first circle, surrounding the individual is a circle including family, friends, and relationships. Another circle encompassing that one and representing a larger circle is one for community including, neighborhood, work, and school environments. The next level of influence on the individual, represented in an even larger circle is state rules, national laws, and public policy. Accepted norms in all of these different spheres impact the individual in the middle of it all [11].

Each person is influenced by his or her environment, and each person also influences his or her environment. Nicholas Christakis and James Fowler expand on this concept in their book, *Connect: The Amazing Power of Social Networks and How They Shape Our Lives* [12]. They describe how one individual's weight loss affects more than just that person's immediate circle. Healthier lifestyle habits can spread throughout social networks. A concerted effort to take into consideration more than just the individual working on adopting healthy habits is critical to create an environment where the opportunity to eat a healthy lunch at work is the norm, not the exception. This helps satisfy the opportunity piece in BJ Fogg's behavior change methodology [13]. The capacity or ability to eat that healthy lunch requires knowing the ingredients of a health lunch, shopping for the ingredients, planning, perhaps preparing the meal the night before and packing it, obtaining an appropriate container for travel, and being able to store it in the office. Finding the motivation to accumulate the knowledge and skills required to make a healthy lunch and also to ensure that there is an opportunity to consume that lunch at the workplace is another pivotal feature of a successful behavior change process and plan. Patient motivation is often forgotten in the routine clinic visit, which is generally full of sharing knowledge and advising, sometimes without even checking that the patient is hearing the information or accepting it.

Crafting a powerful message and identifying skills, techniques, and tools that enable the messengers to effectively communicate the message about healthy eating patterns are equally important. With nutrition, the message is often murky due to competing voices and statements on diets, food groups, and individual substances like sugar and salt. The messenger can influence the message in many ways. The way a provider communicates can either build roadblocks or build bridges. Thus, the provider needs to not only acquire nutrition knowledge but also acquire interviewing skills, assessment strategies, motivational tools, and a collaborative attitude.

When treated as a journey, the behavior change process becomes less scary and risky to patients. The destination, where the patient wants to go, is usually clear. How to get there is usually unclear. The process of getting from where they are to where they want to be needs to be accompanied by a plan for sustainability that includes setting up social support and a healthful environment. A best-case scenario is that the patient arrives at his or her desired destination and then decides to continue on the behavior change journey. Perhaps, feeling confident about adopting nutrition practices that allow for weight management, blood pressure control, glucose control, and satiety throughout the day, the patient will be empowered to tackle other healthy factors from the Nurses' study like increasing physical activity. The nutrition behavior change journey can be joyful. Focusing more on the process, the actions, and behaviors that lead to the destination is vital for patients to be able to experience the

joy of the journey. The reward of reaching the destination of adopting a healthy eating pattern is fulfilling for the patient. The why of starting the journey or the why of moving from here to there needs to be elicited from the patient. As Friedrich Nietzsche said, "He who has a why to live can bear almost any how." Feeling inspired to take the first steps on the journey and feeling motivated to continue the behavior change process all the way into maintenance takes internal drive, social support, and often environmental restructuring. The journey is not always an enjoyable one, and there are hardships, as well as mistakes along the way. However, with the COACH approach, the missteps are not viewed as tracks to be covered up but footprints on the journey that are to be examined for learning purposes. This approach makes the journey more pleasant and less scary.

By examining the nutrition messages, exploring the methodology the messengers use to convey the messages, recognizing the power of the patient's motivation, determining how to make a plan for behavior change with SMART goals, and finding ways to empower patients to maintain their newly adopted healthy eating patterns, this chapter will provide a framework for behavior change counseling in nutrition.

The Message

The messages about nutrition have changed over the years as scientific evidence has led to a more comprehensive understanding of how nutrients are handled by the human body, how food groups are combined in healthy proportions, and ultimately how eating patterns are the most important aspects of the diet. The US Dietary Guidelines for 2015 focus heavily on this aspect of the diet: healthy eating patterns [14]. The research on the Mediterranean Diet helped to bring this concept of eating patterns to the forefront of nutrition science [15–19]. Dan Buettner's work on the Blue Zones [20] highlighted the importance of eating patterns and lifestyles that were correlated with living to 100 years old. The eating patterns of centenarians parallel those of the Mediterranean Diet. This textbook provides the reader with the latest in nutrition science, and this source will enable the reader to counsel and provide messages that are scientifically sound. A sound message is one of the main goals for nutrition counseling. In fact, without a solid, evidence-based message, the process of behavior change counseling in nutrition cannot go forward.

The message needs to be clear and scientifically proven, but it also needs to speak to the inner emotional life of the patient and touch them personally in their hearts and minds. Two psychologists, Chip and Dan Heath, describe the process of change in detail in their book *Switch: How to Change Things When Change is Hard* [21]. After years of research and clinical practice, the Heath brothers have distilled their findings into three main components: (1) crafting a moving message that speaks to the emotional part of the patient, (2) making a cogent and scientifically sound argument that speaks to the rational side of the patient, (3) working to "shape the path" so that the person has the opportunity and ability to perform the new healthy habit.

This third piece, "shaping the path," is often missed in the clinical visit. "Shaping the path" translates to making sure the environment, the person's social connections, and their day-to-day influences are sending messages that support healthy habits. If they are not, the path needs to be reshaped so that they are. In some cases, this might involve conversations with the patient to determine if there are bike paths near work, if there is a farmer's market in their neighborhood, if there is a refrigerator at work, or if they need to purchase a thermal bag to store their lunch in the morning while at work. Are there other people or friends that are also adopting healthy eating patterns? Messages from the environment, community, family, friends, and workplace can be powerful. The messages sent from the health care provider to the patient are often targeted to the patient directly, but they also need to take into account a patient's different spheres of influence, social support, surroundings, and environment, as is emphasized in the social ecological model of change. Nutrition counseling has special challenges due to the changing landscape of nutrition science and its interpretation. This is largely influenced by the latest dietary recommendations from the media, nutrition specialists, and those who are not specialists but have strong opinions and a platform to share those opinions. This leads to mixed messages such as "Butter is back," but "Cholesterol is bad." "Bread is bad," but "Eating fiber is healthy." These messages are plastered over the front pages of popular magazines and are often reported in the news. Even experts disagree on specifics of a healthy diet such as how much fat should be part of a meal, whether dietary cholesterol is an important factor for health, and whether red meat causes medical problems. With each provocative new nutrition message, the wisdom of solid statements backed by scientific facts is pushed to the background.

The experts do agree on the big picture of nutrition. Michael Pollan's common sense advice of "Eat whole foods, mostly plants, not too much" [22] and the Harvard School of Public Health's Healthy Eating Plate that emphasizes having half the plate full of vegetables and fruit, one quarter of the plate as complex carbohydrates and the other quarter as healthy protein [23] are messages that are worth spreading. Both of these dietary approaches focus on vegetables, fruits, whole grains, seeds, nuts, and healthy proteins. These are the components of the diets of centenarians that Dan Buettner and his National Geographic crew discovered when they studied five "Blue Zones" around the world, including Loma Linda, CA, Nicoya Peninsula, Costa Rica, Icaria, Greece, Okinawa, Japan, and Santorini, Italy [20]. The diets in these areas are full of whole foods, mostly plants. For example, in Sardinia, Italy, a lunch might consist of minestrone full of fava beans, chick peas, onions, garlic, tomatoes, potatoes, couscous, olive oil, and fresh herbs and spices [20]. These are also the components of the Mediterranean diet.

One of the other confusing issues in nutrition is that there are several different popular diets, such as the Atkins diet, Paleo diet, low-carb diet, low-fat diet, high-protein diet, South Beach Diet, and a gluten-free diet, with seemingly conflicting messages. A systematic review of different popular diets revealed that most of these diets actually shared the similar theme of eating whole foods, not too much, and mostly plants [24]. There are many medically oriented diets such as the DASH diet [25, 26], the AHA (American Heart Association) diet [27], the ACS (American Cancer Society) diet [28], the ADA (American Diabetic Association) diet [29], the Mayo Clinic diet [30], the Detox diet [31], and Dean Ornish's Spectrum diet [32]. All of these diets have their positive value and healthful effects in specific populations. They also all share similar qualities and characteristics [24, 33]. The lesson is that each person is individual and a specific diet might work well for one person, but not for another. Focusing on the basics of healthy eating patterns and consuming whole, unprocessed foods in proper portions, rather than getting lost in the arguments about calories, nutrients, and food groups is the critical step to consistent messaging about eating for health and longevity. In fact, something as easy as a text message reminder that said "Eat 5 fruits today" was successful at increasing fruit consumption in one study [34]. A simple and concise message is key for finding a clear path forward.

Portion control is a real issue in the United States. Portion distortion has affected the size of plates, amount of food served in restaurants, abundance and variety of dishes at buffets, and expectations of consumers of restaurants. There is an understood code that more food is better, and restaurants that serve more food are better because they give the patrons more for their money. However, is more really better? Perhaps, if we are discussing vegetable consumption, more is better. Otherwise, portion control is more important than getting the most for your money. Buffets and all-you-can-eat cafeterias at school and at work are setups for weight gain. Research demonstrates that if people use smaller plates, they consume less food [35]. Reviewing portion sizes for a variety of foods is recommended when counseling patients on nutrition and healthy eating patterns.

There are several serving size charts available to use with patients that can help guide patients on portion control [36–39]. MedlinePlus provides a reference handout for patients, if they are interested [40]. Using a hand as a gauge to estimate portion sizes is convenient because a hand is always available to the patient. With this technique, the palm represents 3 oz. The fist represents a cup, and half of the fist is half a cup. The fingertip represents a teaspoon, and the thumb tip is a tablespoon. These are

rough estimations, but they can be used as general guidelines. Another method of internalizing portion sizes is to connect them to common household items. For example, a portion of fish is the size of a checkbook. A portion of meat is the size of a deck of cards. A portion of raw vegetables is a cup which is the size of a fist. A portion of cooked vegetables is ½ a cup or ½ a fist. An apple is a serving of fruit, about the size of a baseball. For complex carbohydrates like cooked whole wheat pasta or cooked cereal like oatmeal, one serving is ½ cup or ½ of a fist. For beans, ½ cup of cooked beans is a serving and ¼ cup of uncooked beans is a serving. The serving size for nuts varies depending on the nut, but in general an ounce is considered the equivalent of one serving for nuts. For almonds, one ounce is about 23 almonds, a handful, or ¼ cup. Providing messages about the serving sizes of foods is helpful for patients. Connecting the serving size to a specific part of the hand or another familiar object can help patients to be mindful of the quantity of food they consume no matter where they are. Knowing serving sizes helps patients to appropriately gauge portions and to appreciate how oversized most American restaurant portions are.

Consuming water as a beverage of choice for most meals is also part of the message in a healthy eating pattern. The body is 60 % water [41]. The Institute of Medicine guidelines suggest a fluid intake of 3.7 L for men and 2.7 L for women per day [42]. However, the widespread notion of drinking eight 8 oz. glasses of water is largely unsubstantiated [43]. Drinking normally with meals and when thirsty is largely sufficient for most Americans. Much of the water we consume comes from the foods that we eat, which is a fact many people forget, or have not been taught. Water from food must also be factored into the daily consumption equation. Some people eat to fill a need, which may actually arise from being thirsty as opposed to being hungry. So, an important message is to stay hydrated.

Messages about what foods to eat and how much to eat are the first ones to convey when counseling on nutrition. A clear consistent message that leads to optimal health and wellness is "wholesome foods in sensible combinations" [33].

The Messenger

Relaying the Message: The 5 A's

The 5 A's represent a methodology of counseling on behavior change, especially weight management, that has been in use for decades [44–46]. It has been updated and altered over the years. The 5 A's include assessing, advising, agreeing, assisting, and arranging.

To assess, the health care provider starts by asking questions, determining BMI, measuring blood pressure, checking lab values, especially fasting glucose levels, and then examining the patient for signs and symptoms of disease. Specifically for nutrition counseling, asking about dietary intake (breakfast, lunch, dinner, and snacks) is important. Asking patients how many vegetables they consume in a day is a useful question to gauge the quality of their diets. Also, asking how many times patients eat out versus prepare food at home is another helpful question, as most restaurants serve extra-large portions and use extra salt in their meals. In an open and nonjudgmental way, asking about nighttime eating and stress eating can be enlightening. The assessing part of the interview is the opening section and sets the stage for the rest of the session.

After assessing the patient, the provider has an idea as to what the patient needs and can then advise the patient with specific recommendations that are directed to that particular patient's situation. For example, if the patient is obese and has an elevated fasting glucose, the recommendations will be focused on prediabetes and weight loss. If it appears that the patient may be experiencing a significant amount of stress and using food as a way to cope, then further discussions and recommendations for stress management are an important part of the counseling session. After advising, the next step is agreeing. This is the step that is often forgotten or purposely passed over to save time. Ignoring this step usually results in a failure of translating the advice into action. In the agree phase of counseling, the provider gets the patient's perspective and allows the patient to choose to agree to the proposed plan or to discuss altering it. This is the step that was added most recently to the 5 A's of counseling on behavior change. Without this step, behavior change counseling is mostly advising. After assessing and asking questions to gather information, it can become a one-way street where the provider does all the talking and the patient just listens. With the added agree step, the one-way street is widened into a two-way street for information exchange, and the patient has a voice. This transforms the counseling from just telling a patient what to do and expressing the ideas of the provider, to communicating with the patient and listening to the ideas, concerns, and feelings of the patient. With the agree step, the provider gives the patient the autonomy he or she needs for volitional motivation, as described in the self-determination theory [8].

Next, the provider works to assist the patient in achieving his or her goals by either helping to identify books, cookbooks, healthy recipes better options at a local grocery store, or resources, such as the healthy plate, to give to the patient. This step might also involve recommending an appointment with a nutritionist. It could also include finding local opportunities to take cooking classes. The provider and the other team members in the office can help assist the patient to connect with local organizations, events, or other opportunities to learn about or try healthy eating strategies and to follow through with the agreed upon plan.

Finally, the consultation closes with the last A, arranging. The provider arranges follow-up with the patient, orders any additional lab tests, and sets up the appointment with a nutritionist or another provider that is part of the team. Scheduling the next appointment creates a timeline for when the patient and provider will meet again to discuss any progress made on the patient's goals. The follow-up might also be scheduled with another member of the team such as a medical assistant, nurse, or a nurse practitioner. The patient needs to understand that there is a coordination of care with the patient and that other members of the team will be communicating any progress to the physician. This is an example of how the 5 A's work in a nutrition counseling session [46–48].

That middle step, the agree step of the 5 A's is the critical step. The magic to lasting change in dietary patterns is the collaborative attitude and COACH approach to counseling. This relies on the patient not only agreeing to but also helping to co-create the proposed plan. The COACH approach, with an emphasis on connection and two-way communication, allows for the provider and patient to be equals and teammates in the food "games" that people play in order to change these "games" into healthy habits and a joyful journey. The game is often to try the latest diet for 30 days and then go off the diet due to frustration and disappointment. This leads to the vicious cycle of gaining weight, losing weight, and gaining weight again. Weight cycling or "yo-yo" dieting [49] has been proven to be hazardous to both health and happiness [50, 51]. To change the game and to become part of a healthy journey, the health care provider can look to the patient as an expert in his or her own life. No one knows the patient's life experiences, motivators, obstacles, stresses, successes, strengths, weaknesses, fears, failures, and mindsets better than the patients themselves. The provider is the expert in nutrition, dietary guidelines, health, and disease. Thus, the provider and patient create a team of two that have the knowledge and skills to tackle unhealthy habits and embrace new healthful habits.

COACH approach	EXPERT approach
C=Curiosity	E=Examine
O=Openness	X=X-ray
A=Appreciation	P=Plan
C=Compassion	E=Explain
H=Honesty	R=Repeat and review
	T=Tell and sell

With the COACH approach, the focus is on the patient and the patient's agenda. The opposite of this approach is the EXPERT approach. In the expert approach, the health care provider adheres tightly to his or her own agenda. The provider performs the intervention(s) he or she feels is best suited for the patient and bestows the knowledge that he or she feels is necessary for the patient. The provider needs to examine the patient, obtain labs, understand circumstances, occasionally order X-rays or other tests to narrow down the differential diagnosis and be able to craft a plan for treatment. After this planning, the provider explains the situation to the patient and often needs to repeat and review the treatment plan for clarity. Usually, providers feel compelled to use the "tell and sell" approach to convince the patient of the plan. This approach only uses the assess and advise steps of the 5 A's. In an acute care setting, such as in the Emergency Room when a patient arrives with chest pain, or in the Intensive Care Unit when a patient is seriously ill, this expert approach can save a patient's life. Chronic conditions are different. In the area of behavior change related to chronic conditions, the process is a long-lasting one, not an acute problem. There is not a single medication or intervention that can solve the problems created by a poor diet or a sedentary lifestyle. The lifestyle medicine prescription is complex. Nutrition counseling is more than prescribing a pill. Thus, a different approach is warranted.

In the COACH approach, the provider uses the power of connection as an intervention in itself. By connecting with the patient, the provider opens up the opportunity for developing a trusting relationship in which the patient will feel comfortable sharing his or her true feelings, actual behaviors (healthy and unhealthy), thought patterns, mindsets, fears, failures, expectations, and hopes. This trusting, healing relationship will help drive the process of change forward. To embark on the journey of behavior change with a patient is to develop a mutually respectful relationship, a therapeutic relationship in which each person brings wisdom and insight into the discovery and development of healthy behaviors that pave the path to optimal health and wellness.

The evidence base behind this approach is rooted in the medical and psychological literature. Coaching has demonstrated improved efficacy and outcomes in managing a variety of chronic conditions including asthma, cancer pain, cardiovascular disease, diabetes, osteoporosis, obesity, chronic pain, and physical inactivity [52-64]. Specifically, randomized controlled trials using a coach approach have been conducted in patients with asthma looking at rehospitalization rates, chronic obstructive pulmonary disease (COPD) and rehospitalizations, cancer pain, cardiovascular disease, diabetes, and Emotional Intelligence. Pediatric patients with asthma who received the coaching intervention, along with their parents, had decreased rehospitalization rates compared to controls [52]. Patients with COPD who underwent a comprehensive coaching intervention experienced a marked absolute risk reduction in rehospitalization that was statistically significant [53]. Cancer pain was reduced in 2 weeks through one time coaching intervention [54]. Coaching was shown to have significant effects on lowering cholesterol levels of patients with hyperlipidemia by 21 mg/dL, compared to a 7 mg/dL reduction in controls over a 6-week period [56]. In patients with diabetes, hemoglobin A1c levels decreased, dietary self-management improved, and higher satisfaction with their care were found in patients receiving coaching as compared to controls [57–59]. A study in Japan examined mothers of young children, only including mothers with median Emotional Intelligence scores or lower in the research protocol. They were randomized into a coaching intervention or no intervention. After the 3-month coaching intervention, saliva cortisol levels were lower and Emotional Intelligence scores of the mothers were higher compared to those of mothers in the control group [65]. Health and wellness coaching have been studied in a number of diseases and conditions.

These randomized controlled trials are seedling studies with a small number of patients and short follow-up periods. More work needs to be done to conduct high-quality coaching studies [66], but there is evidence that this approach is better than usual care. Review articles have confirmed the value of health and wellness coaching and demonstrate that health coaching results in significantly better weight management, increased physical activity, and improved physical and mental health status in patients with chronic disease [67]. Although there is no formal definition of coaching, the common

themes throughout the literature suggest a collaboration, self-discovery, goal setting, and accountability as key features of a health and wellness coaching [68].

Curiosity

Curiosity is defined as "the desire to learn or know more about something or someone" [69]. Starting each visit with genuine curiosity about the patient with what Dr. Jon Kabat-Zinn, the physician who developed mindfulness-based stress reduction, calls "a beginner's mind" allows full focus on the current issues plaguing the patient [70]. It often takes a moment of silence or deep breathing to gather one's self prior to beginning a new visit or consultation. This preparation to be mindful, fully present, and genuinely curious at the start of each clinic visit is a gift to both the patient and the provider. Asking questions about the patient's motivators, obstacles, strategies, and strengths will invite the patient to think deeply about his or her current situation and will likely help guide the clinic visit. As an old proverb credited to Publilius Syrus states: "A rolling stone gathers no moss." In this case, the moss can be viewed as disease growing on the stone similar to disease growing in a patient. The idea is to keep moving to keep disease-free. Thus, this mnemonic of *M.O.S.S.* can help providers remember to ask questions about (*M*)-motivators, (*O*)-obstacles, (*S*)-strategies, and (*S*)-strengths. Questions in these four areas can spark a patient to get moving on healthy habits.

In a piece in the *Annals of Internal Medicine* "On Being a Doctor" over a decade ago, Dr. Faith Fitzgerald, who was awarded the title "Master Physician" by the American College of Physicians in 1991, summarized the importance of curiosity [71]. This excerpt from the article provides great insight into the power of curiosity.

"I believe that it is curiosity that converts strangers (the objects of analysis) into people we can empathize with. To participate in the feelings and ideas of one's patients—to empathize—one must be curious enough to know the patients: their characters, cultures, spiritual and physical responses, hopes, past, and social surrounds. Truly curious people go beyond science into art, history, literature, and language as part of the practice of medicine. Both the science and the art of medicine are advanced by curiosity" [71].

Being in the moment and genuinely curious about the patient's experience, story, and struggles, strengthens the bond between provider and patient because the patient appreciates the fact that they are the center of attention for that visit, for that moment in time. Curiosity may also improve the provider's mental health and happiness [72, 73]. In the book *Curious*?: *Discover the Missing Ingredient to a Fulfilling Life* by psychologist Todd Kashdan, there is support for curiosity's role in improving health, intelligence, social relationships, happiness, and meaning in life [74, 75]. Furthermore, being a better listener creates opportunities to discover something interesting and thought provoking, which will lead to the next inquiry [73]. Questions such as "And then what happened?" or "Why did you think that?" may lead to other revelations and provide a deeper level of understanding [73]. Some have even argued that when we are not curious, we do not listen [76]. Fostering our "curiosity skills" requires effort and practice. Being present, choosing how to listen, and asking curious open-ended questions are the salient components that enable active listening and prepare providers to learn about others. This is what ultimately leads to understanding others [76].

Practical Tip A method to open yourself up to curiosity at every visit.

Placing a hand on the door frame before entering the room of the next patient is a simple way of grounding one's self prior to beginning a new consultation. This centering process helps providers dismiss their own worries, doubts, daily concerns, and focus fully on entering into a partnership with that patient. Other possible grounding techniques include taking five deep breaths prior to entering the room or stopping right before the door, closing your eyes, and taking a moment to feel the floor

beneath your feet. With a beginner's mind, the provider can open the door to the examining room and let curiosity lead the way.

Openness

Openness means acceptance, tolerance, open-heartedness, and being nonjudgmental. It implies a collaborative and cooperative approach [77]. In a study examining what makes a physician an exemplary communicator with patients, it was demonstrated that empathy, self-reflection, and nonjudgment were notable capacities that led to exemplary communication [78]. Thus, it is no surprise that a study examining what influenced patients' ratings of physicians found that patients reported being more satisfied with providers who are rated as relatively high on openness compared to other physicians [79]. When managing difficult clinical encounters, it is advised that providers use empathy and a nonjudgmental attitude to better manage challenging situations [80].

As specified in a medical journal article over 15 years ago, "The clinician should be careful not to be judgmental or scolding because this may rapidly close down communication" [81]. Openness to whatever presents itself in the visit requires the provider to embrace a nonjudgmental attitude, or what has been described as "unconditional positive regard" toward the patient [82]. Nonjudgmental acceptance has been shown to be a significant predictor of alcohol use coping motives [83], and changing drinking patterns is one of the most difficult habits to break. A nonjudgmental stance resists the "good" or "bad" titles; everything simply is as it is [84]. An overweight patient, who has now moved into the obese category, needs a provider who can speak to him or her in a nonjudgmental tone, with a curious mind. And, if a patient does not reach his or her goals, or if he or she abandons the plan, the provider needs to approach the problems with both curiosity and openness. Being open invites effective brainstorming around obstacles without the fear of harsh judgment for an idea that is untraditional or unexpected. It fosters an environment consistent with a growth mindset where mistakes are opportunities to learn and grow [85]. It also opens up possibilities for the future that instill hope and energy into the clinical encounter. When clinicians hold an open stance, patients are more likely to divulge the truth no matter how surprising or awful it might be. Openness builds trust.

Practical Tip and Patient Example The Patient behaves in a manner contradictory to his goals.

The patient decides that he will no longer frequent fast food restaurants and then finds himself eating dinner at a fast food restaurant for four nights in a row. Instead of the provider starting the dialogue about this situation by telling the patient how he must feel or how the provider feels about the situation, which would be judgmental, the provider can probe and be open to explore what the patient is thinking. Rather than use phrases like "That is pretty disappointing" or "That shows a lack of willpower and a distinct lack of planning," the provider could start by saying, "Tell me about how you were feeling when you approached the drive thru." Then, "How did you feel after you ate the food?" "How would you have felt if you were able to go home and eat a healthy meal?" Eliciting this type of information from the patient invites the patient to investigate thoughts and feelings as well as triggers and rewards in a nonjudgmental environment.

Appreciation

Appreciation is defined as the ability to understand the worth, quality, or importance of something [86]. This is the third component of the COACH approach. It is an important way of building a patient's self-efficacy. Self-efficacy is defined as a person's belief that they can do a particular task,

such as dance or ride a bicycle [87]. The goal is to appreciate the positive events, behaviors, and attitudes as well as the strengths that the provider witnesses and perceives in that patient. "What we appreciate, appreciates." In this way, the patient starts to view his own gifts and talents as assets to be utilized in the behavior change process. Appreciation of character strengths has been demonstrated to increase self-efficacy and self-esteem in challenging populations such as adolescents with psychiatric illness [88].

When people have suffered setbacks in life, work, or health, they rely on resiliency to get back on track. Many people who receive counseling on healthy eating have either heart disease, cancer, stroke, overweight/obesity, or diabetes. Being able to identify talents, personal gifts, and character strengths unique to that individual helps them build resiliency and grow stronger from the adversity or health setback so that they can thrive after the event. Some diabetes educators are using a resiliency model of training in addition to their standard education in order to help patients improve their diabetes-related stress, their self-management, eat healthier and exercise more [89, 90]. With the challenging adolescent population, a focus on "promotive factors," such as assets like self-esteem and self-efficacy, helps troubled youth to find the strength and the ability to change their lives for the better [91].

Martin Seligman, considered the "father" of Positive Psychology, developed an online questionnaire that is free, requires little time (10–15 min), and is easily available that can help patients identify their own strengths called the Values in Action Inventory of Strengths (VIA-IS) [92]. Research on this questionnaire has shown that it can be used in the workplace to enhance positive work outcomes [93]. Using a strengths-based approach has been reported in the medical literature in the area of teaching nursing students. It emphasizes the use of affirming and enhancing the capabilities, interests, goals, and knowledge of the nursing students to help empower them [94]. With the COACH approach, patients are viewed as students and researchers of their own health, and empowering them to embrace self-management skills is the ultimate goal of the counseling session. Thus, appreciating the strengths of the patient, focusing on what is going well, and identifying the positive in the patient's story is of paramount importance.

Many times the patient is surprised that the provider is focusing on the positive event and not honing in on the negative or the problems. This surprise works to the benefit of the provider and the patient. Reflecting upon and reliving the experience of eating the salad that one ate for lunch, will allow for visualization of the experience. This can reinforce the behavior in the brain. Using visualization of the healthy behavior in reviewing a week's events, or when planning the week ahead, can be an effective strategy for priming the brain to perform that healthy behavior again, or for the first time. There is evidence that inviting patients to visualize themselves going to the grocery story, selecting the fruits, putting them in their carts, and then eating the fruits, will lead to increased fruit consumptions [95].

Practical Tip and Patient Example A patient meets some but not all of her healthy eating goals.

A patient reports that she ate salads for lunch one out of the 3 days that she had committed to eating salads, and then harps on the 2 days that did not work out as planned. The provider can ask an openended question or make a request that draws out the positive such as "Tell me about the day when you did eat the salad. What happened that day? How did you feel? How did you prepare? What did you feel after you ate the salad? Tell me about how you think choosing and eating the salad affected the rest of the day. What can you do to increase the odds that you will eat a salad for lunch in the future? What happened on the days that you did not eat a salad for lunch? What did you learn from that situation?"

Practical Tip and Patient Example A patient is gaining weight after losing weight.

The patient who had lost 20 lb, has now gained weight and has fallen off his healthy eating plan. He feels lousy. Appreciating the positive would involve mentioning the patient's strengths that allowed him to lose weight in the first place. This points out that he has been able to follow a plan that works for him in the past. The provider could discuss how his strengths could be used now to get him back on track. He might be a creative artist, which means that he could use his creativity to problem solve

around barriers to healthy eating. Another possibility is that he is a workaholic businessman dedicated to his clients. He could consider transferring some of that dedication and perseverance to his own selfcare. It will likely take connecting his self-care to his productivity at work and his ability to help clients for many years to come, for him to take care of his own health. Asking him what he thinks of the quote by Ralph Waldo Emerson, "The first wealth is health," could provide some stimulating conversation around self-care. With a curious, open, and appreciative mind, the provider can discover information about the patient that can be used to help inspire the patient on his behavior change journey.

Compassion

The second C in the COACH approach model stands for compassion. Compassion is defined as the ability to understand a person's suffering and to feel compelled to alleviate it [96]. It is one of the cornerstone pieces of the first principle in the American Medical Association's list of Principles of Medical Ethics: "A physician shall be dedicated to providing competent medical care, with *compassion* and respect for human dignity and rights" [97]. Compassion is a cardinal feature of the COACH approach, and is in itself considered an intervention. Similar to self-care, self-compassion is also important for health care providers and it relates to patient care. Research suggests that trainees who are more skilled at mindfulness and self-compassion demonstrate positive associations in providing calm, compassionate care [98].

Empathy can be considered a precursor to compassion. Empathy is the feeling that ignites people to compassionate actions. Empathy is the ability to walk in someone's shoes, really understand their situation, and feel their feelings. There is research on empathy that demonstrates the correlation between provider empathy levels and patient levels of hemoglobin A1c and LDL cholesterol [99]. Providers with higher levels of empathy, as scored on the Jefferson Scale of Empathy, had patient panels with lower hemoglobin A1c and LDL cholesterol levels compared to providers with lower empathy levels [99]. Empathy is a factor for patient satisfaction as well. A study in hand surgeons found that patient satisfaction was primarily linked to surgeon empathy, not visit duration or previsit expectation of visit length [100]. Importantly, empathy can be fostered. A study of medical students yielded improved empathy in standardized patient interactions after providing empathy-specific feedback in a virtual interaction [101].

The ability to "walk in someone else's shoes" is important when a provider is counseling patients on behavior change, especially diet. A compassionate approach is fostered by the first three elements of the coach approach (curiosity, openness, and appreciation). With a mindful style of interviewing, the provider is ripe for fully understanding the patient's situation, thoughts, feelings, needs, and desires.

Feeling empathy and expressing empathy are two different things. It is possible to feel empathy, but not express it. This is like giving a gift to someone but not allowing them to unwrap it. It is there, but it has little impact. For the full impact of the gift to be realized, the gift needs to be unwrapped. For the empathy to be felt by the patient, the provider needs to express it. How does one express empathy?

Expression is 8% verbal, 15% tone of voice, and 77% body language [102]. Thus, being mindful of one's demeanor, facial expressions, posture, hand movements, volume and velocity of speech are all important during the clinical encounter. In order to express empathy, the provider needs to look the patient in the eye. In fact, the way an office space is set up can affect how a provider expresses empathy and delivers compassionate care.

Practical Tip Setting your office space for optimal expression of empathy.

Eye contact can be encouraged by ensuring that the provider and the patient are at the same level, sitting and facing one another. Having a computer between the provider and patient can be distracting and can serve as a physical barrier to creating a high-quality connection during the visit. It can be

helpful to allow for some silence and thoughtful reflection after profound statements are expressed by the patient. This allows the patient time to reflect on his or her thoughts and also gives the provider an opportunity to ponder the implications of the patient's words. Responding in a soft tone of voice when addressing serious material is one way to demonstrate empathy. Speaking more slowly during these conversations also reveals to the patient that you grasp the severity of the situation. In this way, the provider follows the lead of the patient. Leaning into the conversation by physically leaning forward is also a sign to the speaker that the listener is interested and invested in the conversation. Maintaining eye contact shows that the provider is focused on the patient. Checking one's phone, using the computer, flipping through papers, examining lab results, sitting with arms crossed leaning away from the patient, or staring off into the corner of the room gives the impression that the listener is distracted and disinterested. Suggesting a disengaged attitude to the patient can significantly hinder the expression of empathy and the delivery of compassionate care.

Honesty

Honesty is defined as the quality of being fair and truthful, and being honest is marked by free, forthright, and sincere expression [103, 104]. For a connection to develop and to grow, it must be built on trust. Honesty is one of the most important building blocks of trust. For the provider, this means that all information shared with a patient is accurate and reliable. If a patient has a BMI in the obese category, but the provider feels uncomfortable talking about weight for fear that he or she might offend the patient, then that provider cannot be honest with the patient. Some providers put the BMI on the lab results that are sent to the patient and add a sentence stating that the patient is obese by BMI and needs to lose weight. If this happens, the patient will wonder why the provider did not bring up the BMI during the consultation when the patient could ask questions and discuss these issues. Being upfront and honest during the consultation will increase the patient's trust and belief in the provider. A provider must learn to be comfortable with the truth, even if the truth is uncomfortable. As an example, the most important themes expressed by patients with interstitial lung disease regarding disease education, was the importance of knowing what the future might bring and the need for honesty from clinicians [105]. If the provider is at ease discussing difficult topics, then these topics become less challenging and scary to the patient.

Being honest also means being apologetic to patients when a medical error occurs. Honesty and apologies go a long way, and actually lead to less litigation against physicians [106–109]. Patients who are treated with respect and honesty are more likely to treat themselves the same way.

Practical Tip and Patient Example Confronting a patient that might be binge eating.

Part of honesty is sharing your discomfort and your fears with the patient. This means that if you are genuinely worried and scared about a patient's binge eating patterns, it is helpful to express these thoughts and concerns, in a compassionate, open-minded manner. A simple statement such as "It sounds to me like you might be consuming many more calories than you are planning to consume in a sitting, and then you are feeling out of control when you are eating this way. Am I understanding this correctly?" By adding the last question, the provider allows the patient the opportunity to explore the topic further, correct the statement, and clarify his or her own feelings about the subject being discussed. Avoiding any potential conflict or discomfort does not foster an environment of honesty and compassion. In fact, doing this can inhibit a patient from addressing the real issues during the consultation. If it sounds like depression is driving the initial overeating, and the patient seems to be expressing sadness without stating it directly, then asking about feeling sad could be helpful. By naming the emotion that seems to be surfacing during an interview, like sadness or fear, a provider is being honest and normalizing the negative feelings which is also useful. If negative emotions surface, it is better to acknowledge them. Reflect what you hear. "You sound sad" or "You sound really

emotional." This builds trust. It allows the patient to experience the emotion and share it with another person. With honest expression from the patient and provider, the patient will be able to get the help he or she needs, whether it is from a lifestyle medicine practitioner, psychiatrist, or nutrition specialist. In order to truly help a patient, a positive therapeutic relationship [110] and solid connection need to be created that allows both patient and provider to be honest with one another, even when it is uncomfortable.

Motivation

Motivational Interviewing (MI) is a powerful skill set for counseling patients in nutrition and other areas of behavior change. Motivational Interviewing was developed by William Miller, PhD and Stephen Rollnick, PhD and was built on their many years of working with patients with substance use disorders, which are behaviors known to be resistant to change [7]. Through their research, they developed counseling techniques to deal with ambivalence to change and resistance to change. MI has been studied in several randomized controlled trials. In a meta-analysis, Motivational Interviewing has demonstrated positive intervention effects on total cholesterol, systolic blood pressure, and weight loss [111–113].

In their book, *Motivational Interviewing: Helping People Change*, Miller and Rollnick define motivational interviewing as a "collaborative conversation style for strengthening a person's own motivation and commitment to change" [114]. They expand on this definition and write that MI is a "collaborative, goal-oriented style of communication with particular attention to the language of change. It is designed to strengthen personal motivation for and commitment to a specific goal by eliciting and exploring the person's own reasons for change within an atmosphere of acceptance and compassion" [114].

The spirit of MI matches closely with the COACH approach. The spirit of MI involves four main components: collaboration, acceptance, evocation, and compassion. There is a continuum of communication styles with MI, ranging from directing to guiding to following. Primarily with MI, the provider is guiding the patient along through directed questions and carefully selected reflections.

Four of the main skills in MI are reflected in the mnemonic "OARS." The 'O' stands for openended questions, the 'A' for affirmations, the 'R' for reflections, and the 'S' for summaries.

These skills work synergistically with the COACH approach. Open-ended questions enhance the health care providers ability to be curious and open. Open-ended questions by definition cannot be answered with a one-word response such as yes or no. They invite the patient to discuss and expand on a point of interest. These questions are meant to make the patient think deeply about his or her current situation.

Affirmations are represented by the "A" in the Motivational Interviewing OARS framework. In the COACH approach, positive affirmations fit in the appreciation part of the framework. By using affirmations such as "It is wonderful that on Monday you were able to follow through with your plan of eating a salad for lunch," the provider guides the patient to appreciate the positive in the week, and thus the patient can experience a positive emotion in that moment of the conversation. This positivity opens the door for creativity [115], which is a critical skill to use during the behavior change journey. Affirming what is going well builds self-efficacy. These affirmations aid in developing the necessary patient–counselor relationship described by Carl Rogers, PhD [116]. Rogers work brought attention to the importance of the human potential and actualization, self-esteem, and ensuring that the counselor really understood the patient's unique perspectives and views of the world [116].

The "R" in the Motivational Interviewing framework stands for reflections. Reflections on what went well can also increase self-efficacy and serve an important role in the appreciation aspect of the COACH approach. By using reflections, the provider can point out the good things that happened and

uncover them to allow for their acknowledgment and celebration. Reflections can be simple but powerful [7]. A reflection of what you heard a patient say is a clear way of demonstrating you were listening to the patient's words. By repeating the words back directly, as a simple reflection, or in a paraphrased manner, it shows that you are actively listening to what the patient is saying. Simply stating, "I understand how you feel," might not be as effective as repeating back what you heard in an open, curious, considerate way, "I heard you say that you are so busy with work and family that you feel like you have no time for yourself and your eating patterns reflect this rushing around and lack of mindfulness." With this statement, a provider is letting the patient know that he or she not only heard the words of the patient but also he or she understands the meaning behind those words. As a followup to this, a provider can ask an open-ended question such as "How could we work together today to help you feel a greater sense of control over your life and perhaps find a way to include some mindfulness exercises into your day? Do you think that might be useful?" When making these statements and asking the open-ended follow-up questions, the provider looks directly at the patient, speaks in a concerned, kindhearted tone, and leans toward the patient. This delivers the full effect of compassion, embracing the MI spirit and following the COACH approach.

Summaries are longer reflections that are usually created toward the end of a clinical encounter, as they serve to pull together the main points of the session into a coherent paragraph of a few sentences. They can also be used at transition points in the session when closing one topic and moving to another. Reflections can be used several times throughout the interview, but summaries are used more judiciously, and only once or twice. The provider needs to be paying careful attention throughout the entire session to carefully craft an effective and powerful summary. Practicing summaries is a worthwhile endeavor.

The evocation part of the MI spirit involves evoking change talk from the patient. Instead of the expert explaining why the patient should change, the provider practicing MI encourages and guides the patient to do his or her own change talk. Asking open-ended questions such as "How would your life be different if you ate healthy meals each day?" allows the patient the opportunity to describe the benefits of a healthy eating pattern to the provider. When a provider asks patients how confident they are in achieving their goals on a scale of 1–10 and patients respond with a number below seven such as a five, the provider can ask why they selected a five instead of a three. In this way, the patients provide all the reasons that they are confident and can convince themselves of their own reasons to be confident. The same is true of an importance scale. If they report a low number of importance for a goal, then the practitioner asks why they did not select a lower number, which again allows the patient to describe why the goal is actually important.

Using a double-sided reflection can also bring out change talk. If a patient mentions at one point that he wants to stop going to fast foods restaurants because they make him sick, but then later he says, he cannot stop going because his colleagues always go to fast food for lunch. Then, the practitioner has the opportunity to point out this discrepancy and say, "I heard you say that you feel sick whenever you go to fast food restaurants, but it sounds like you can't stop going because your colleagues dine at fast food restaurants." This gives the patient the opportunity to weigh whether feeling sick is more painful than perhaps suggesting another restaurant to colleagues or bringing in food from home for lunch. These techniques bring out the change talk, and change talk is the way patients convince themselves of the need to change.

Along with evoking change talk, MI invites providers to teach and educate. MI utilizes the elicitprovide-elicit model. In this model, the provider asks if the patient wants to learn more about simple carbohydrates and blood glucose levels or if they are interested in hearing about how to use the glycemic index. Depending on how the patient responds, the provider can provide the information that the patient desires. The first step is to elicit a topic of interest to the patient that is related to their health and wellness. The second step is to provide the information that they request. Last is the elicit step, which then evokes from the patient the new information that they learned. A question that will generate this information is "What did you take away from my discussion on the glycemic index? I am just trying to make sure I explained it in a way that is understandable." This is another powerful MI tool: elicit-provide-elicit.

These are just a few of the main skills and tools of MI. The process of motivational interviewing is one that takes time to learn and many hours of training to become proficient in it. There are courses available through the Motivational Interviewing Network of Trainers (MINT) online and in person. Knowing some of the basics and practicing them is helpful for behavior change counseling in nutrition.

Using the lessons in the self-determination theory helps a provider to uncover the patient's intrinsic motivation. Richard Ryan, PhD and Edward Deci, PhD are the psychologists who have created, researched, and written about the self-determination theory (SDT) [8]. In this meta-theory, Dr. Ryan and Dr. Deci describe the ingredients for motivation: autonomy, relatedness, and competence. According to Dr. Ryan and Dr. Deci, in order to achieve the most volitional and high-quality motivation that will lead to increased creativity, performance, and perseverance, patients must experience a sense of autonomy, relatedness, and competence [8]. Recent research demonstrated that using the SDT framework can be beneficial when counseling patients on healthy eating patterns [117]. This research proposes that fulfillment of the need for autonomy, competence, and relatedness, or the obstruction of that need, might help explain the etiology of body image concerns and disordered eating as well as to understand how to regulate eating behavior going forward. Working with the SDT principles and fostering an environment where the patients make choices, have some control, have the opportunity for competence, and can experience social support may play a significant role in creating the motivational processes behind eating regulation and be a target for behavior change interventions [117]. MI and SDT are closely related. It has been suggested that SDT may serve as the theoretical backing supporting MI and that MI may provide SDT with specific direction and clinical techniques to use in practice [118]. One additional dimension to consider is the qualitative aspect of motivation (e.g., perceived level of autonomy), as autonomous regulation has been shown to be one of the key predictors of successful weight control outcomes in overweight/obese patients [119].

The COACH approach fosters an environment that supports all three of the ingredients necessary for motivation. For example, the collaborative style of the process allows for patients to decide what goals they will pursue, what solutions they will choose to get around barriers, and how they will be held accountable to those goals. The relatedness piece is built into the relationship with the provider, but can also include family, close friends, and other supporters. As for competence, this involves setting SMART goals that are challenging enough, yet achievable, which will be discussed in detail later in the chapter. Keeping this theory in mind while counseling patients on healthy eating patterns will allow patients to experience powerful motivation for change.

Intrinsic motivation to change a behavior is the most powerful type of motivation. This type of motivation is internally driven. For example, eating apples helps a person's digestion. The fact that their bowels are regular when they eat apples is a reinforcing outcome. Thus, they will be more motivated to eat apples. When some people eat more green leafy vegetables and less sweets or processed foods, they may notice that their skin clears or glows. If this is viewed as a beneficial effect, they might continue to eat the healthy foods to which they attribute this effect. Other people might notice that they feel full for a longer period of time when they eat a complex carbohydrate versus a simple carbohydrate. This reward of feeling full might drive people to consume complex carbohydrates more often.

Extrinsic motivators are those received from the outside world, like a prize from mom for eating all the vegetables on the plate at dinner. Other examples might include signing up for a weight loss competition to win a prize, such as a trip to Florida or a cruise. These are all external motivators. They can work, and they may help people get started on the development of a healthy habit. Often, people start to feel the internal rewards and notice how the behaviors enhance their lives from the inside out. In this way, an external motivator can lead to an internal motivator.

Appreciative Inquiry (AI) is another methodology to spark motivation and engage the patient in the change process. Appreciative Inquiry is an interviewing process developed by David Cooperrider, PhD [120, 121]. It has been used for decades in the business world and is utilized in many health and wellness coach training programs. There is a 5D cycle in this model, which has five steps: (1) Define, (2) Discover, (3) Dream, (4) Design, (5) Destiny.

In appreciative inquiry, the first step is to *define* the focus. Ask the question, "What do we want more of?" Then, discovering the best of the current situation and circumstance. With this *discovery* step, a guiding question is, "What is working well?" After that comes dreaming about possibilities for the future. A helpful question for envisioning the future, for this *dream* step, is, "What might be possible?" After that comes the *design step*, which works on cocreating what could be and is realistically possible in the present. Finally, the *destiny* step involves learning how to empower, grow, and expand from the plan as it unfolds. Then, the process starts again with the five steps: define, discover, dream, design, and destiny. AI is compatible with a strengths-based approach and positive psychology interventions.

The AI model, is readily adaptable to the patient encounter with definition of the problem, discovery of what works well for the patient, dreaming about what could be possible in the patient's life with healthy habits, designing a plan for action to adopt a new healthy habit, and then destiny, learning from what happens on the behavior change journey as it happens. By appreciating what is going well and building on the positive core in each individual, the provider is setting the stage for the patient to sustain behavior change. There is usually at least one positive aspect of an experience that can be highlighted and discussed. If not, then there is a strength or positive characteristic inside the patient that can be drawn out during the clinical encounter. This makes the encounter more pleasant for the patient and the provider. Recent studies have explored using Appreciative Inquiry with dementia patients and found positive results [122]. Other researchers have conducted a methodological review of all the studies on the use of appreciative inquiry in the health care setting and found promising results with dementia patients, elderly patients at the time of discharge from a hospital, and in medical practice teams [123]. They utilized a 4D cycle, leaving out the initial define step. They noted that the steps were more like guidelines, rather than strict blueprints, that could be adapted to the participants and the specific settings. The authors of the review concluded that, "Appreciative inquiry provides a positive way forward shifting from problems to solutions offering a new way of practicing in health care and health research."

The Messenger Tailors the Message to the Individual

The Transtheoretical Model of Change helps the provider meet the patient's needs as a learner and as a person with unhealthy habits [124, 125]. Each person has a unique story. Giving the patient the opportunity to express his or her story is therapeutic in itself. It also allows the provider the chance to assess the patient's stage of change. There are five stages of change: precontemplation, contemplation, preparation, action, and maintenance. James Prochaska, PhD, John Norcross, PhD, and Carlo Diclemente, PhD wrote a book titled *Changing for Good* [126, 127], which delineates the different stages of change, as well as stage-specific processes that are effective. After having worked with patients struggling with addiction for over 20 years, these psychologists know a great deal about changing when change is hard. Their experience creating this "stages of change" model has been useful not only in patients with addiction but also in patients striving to change their exercise and nutrition habits. The Stages by Principles and Processes of Change (Fig. 3.2) [128] provide recommendations as to how to meet the patient where they are and supply them with what they need to move forward.

The first stage is precontemplation. Precontemplators are not even considering change. Often, they are in a state of denial or are simply unaware of the problem. They say that they "can't" and "won't"

Stages by Principles and Processes of Change

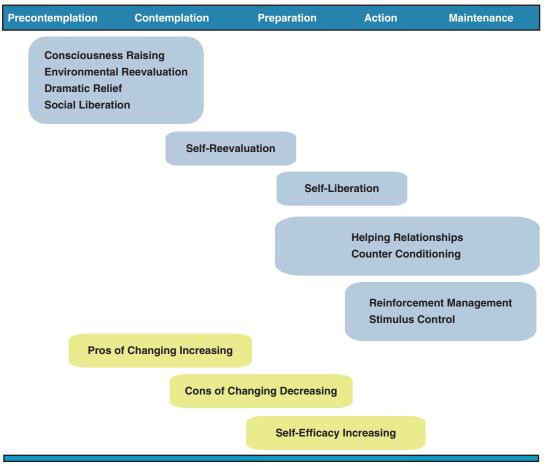


Fig. 3.2 Stages by principles and processes of change [128] (© 2010–2016 Pro-Change Behavior Systems, Inc. Reproduced from www.prochange.com with permission from Pro-Change Behavior Systems, Inc)

change. With these patients, sending the message loud and clear with a megaphone will not likely have the effect of fostering change. This is because these patients are not ready or willing to hear a message, no matter how loud or how valid the message is. For patients in precontemplation, building a connection is the top priority. The connection will create fertile soil for the seeds of change to be planted. The provider's compassion and use of reflections with these patients can be the most powerful interventions. At the same time, it is important to provide the facts and state that being obese has health risks associated with it, such as diabetes and high blood pressure, which could lead to heart attacks and strokes. Making statements that show your support as well as reveal your concern is the goal. For example, a provider might say, "I understand that you are not ready to tackle your weight right now due to your busy family and work life, but being obese comes with serious health risks. So, when you are ready, I would really like to help you work on adopting healthy habits that will help you to minimize these risks. I am here for you." This type of communication will keep the door open and allow a connection to be built.

Some of the processes that are recommended for the precontemplative stage include: consciousness raising, environmental reevaluation, dramatic relief, and social liberation. For consciousness raising, the provider can provide the facts and statistics related to the diseases and behaviors relevant to that particular patient at that time. How food with a high percentage of sugar and a high glycemic index spikes blood glucose, which increases insulin, which promotes fat storage, would be useful to mention to a person with prediabetes or diabetes. Or how high blood sugars damage endothelial cells, which can lead to plaque buildup in the arteries, clots, heart attacks, and strokes would be a powerful message to send to a person with diabetes who has a family history of heart attacks. Environmental reevaluation invites the patient to consider the effect of his or her behavior on the health and wellbeing of others around him or her, such as considering the effects of second-hand smoke on children or grandchildren. Dramatic relief purposefully brings emotion into the encounter by reporting on feararousing lab results that produce significant threats and challenges to the body. Also, a provider can conjure up emotions by telling success stories of patients who struggled initially, but then ended up making changes that brought them health and happiness. Lastly, social liberation brings the environment into the equation by asking the patient to note laws or guidelines in the workplace or common places such as the FDA ban on trans fats. Towns that provide community share gardening opportunities are sending a message of healthy eating and making eating fruits and vegetables part of the norm, rather than the exception. Doctor's offices can offer water as a beverage through a bubbler, which sends the message that water is a healthy option and hydration is important. Clinicians can literally "walk the walk" with the patient by scheduling walking consultation meetings [129].

After precontemplation comes contemplation. Contemplators are stuck in ambivalence, part of them wanting to change and another part of them wanting to stay the same. Some people stay in this stage for months or even years, called chronic contemplation. People in this stage of change often say "I may" or "I might" change. I am "thinking about changing." This contemplation stage is an important one with regard to counseling patients about nutrition. Many people are in a pattern of eating out or eating at drive throughs on the way home or while traveling at lunch. Altering this easy, comfortable, and cheap option might seem daunting and might not appear or feel worth the effort. Change comes with pros and cons. There are pros to the new healthy behavior, and there are also pros to the current behavior patterns. Just as there are cons to the new healthy behavior and cons to the current behavior patterns. Delineating these pros and cons and weighing them can be helpful with these patients. For example, with the situation of eating at fast food chains and drive throughs on the road, the pros might be cost-efficient, time-efficient, and taste, while the cons might be eating too many calories without feeling satiated, craving that specific food later, ordering more food because it is inexpensive, eating quickly while driving, and becoming used to processed, hyper-palatable food. The pros to changing this dining habit would be increasing the consumption of fresh, whole foods such as vegetables and fruits that have water, fiber, phytonutrients, vitamins, and minerals that can be health promoting. Another pro is being able to sit mindfully and enjoy a meal for the full 20 min that it takes to feel satiated. By planning meals and eating at home, a person is able to control portions and share the meal with family and friends, which adds the joy of connection to the joy of eating. The cons of changing this dining habit are that it may take time to learn new recipes, take time to prepare the meals, and there is a risk of overcooking the meal or the meal being less than delicious when completed. By listing this out in a chart, the patient can objectively evaluate the current nutrition pattern. The provider could help the patient to create a written chart of these pros and cons or could talk through the pros and cons (mostly listening). Helping the patient to identify powerful internal motivators may sway the balance of pros and cons toward the pros. Motivators that come from the patient and are connected to his or her strengths, priorities, and overall purpose are the most potent motivators for driving the process of change. Asking open-ended questions about the patient's gifts, talents, and unique characteristics as well as asking about what is most important to them in their life right at that moment will help to clarify the current state of mind of the patient and help provide a road map for a way forward that is consistent with that particular patient's needs and desires.

The processes for contemplation are similar to precontemplation. One additional process that enters the model at this point is self-reevaluation. With this process, patients are encouraged to think of themselves as having adopted the healthy habit already and image what that would be like. The provider could guide the patient by stating, "Imagine that you are eating healthy meals for breakfast, lunch and dinner for a week. What would that be like?"

When patients are finished contemplating the change, they enter the stage of preparation. Patients in preparation are getting ready for change and are planning to take action in the next 30 days. They have usually weighed the pros and cons and are convinced that the pros of changing outweigh the cons. In preparation, patients are often looking for a solid plan A, as well as a plan B. Many people are afraid of failure, so actually plunging into a plan is scary. Considering obstacles and brainstorming solutions around those obstacles enables patients to build self-efficacy. Increasing self-efficacy means bolstering their belief that they can complete the change and perform the behavior that they are seeking to adopt.

Patients often enjoy brainstorming sessions at this point. A brainstorming session involves working synergistically with the patient. The patient and provider identify an obstacle and then one of them (usually the patient) provides a potential solution. After this, the other one (usually the provider) provides a different potential solution. This can go in rounds where each person could identify possibilities. At the end, with six to eight possibilities available, the patient determines which one is best suited to the situation.

The processes that accompany the preparation stage of change include self-liberation, helping relationships, and counterconditioning. With self-liberation, the patient is liberating himself or herself by making a commitment. Patients might tell a friend, post their commitment on a social media platform, make a verbal contract with someone, or even craft a written contract. This type of declaration of their intention to change gives them more power to fulfill their goals and brings them closer to adopting the healthy habit. By telling loved ones or colleagues, the patient can start to use helping relationships to empower his or her change process. The helping relationship is only helping if it is with someone who is caring, trusting, supportive, open, and accepting. If someone is using the COACH approach they can be part of this helping relationship. This type of social support can help people move from preparation to action.

Counterconditioning is another way of saying finding substitutes for old, unhealthy habits. For example, instead of having a candy bar from the vending machine at 3 pm, can the client pack a granola bar that is whole grain with very little sugar? Could the patient eat an apple with almond butter for dessert instead of apple pie? Could the patient eat a salad before the main course instead of nachos? These are all examples of counterconditioning also known as finding substitutions.

The next stage after preparation is action. Patients in action are already doing the healthy activity. They might be actively engaged in the targeted behavior for up to 6 months. Inquiring about how the patient feels and what aspects of the activity he or she is enjoying is a good place to start. Noting any changes on biometrics that might be related to the dietary intake such as blood glucose, blood pressure, weight, or BMI could act as reinforcers for the behavior change. Concentrating on how the new eating pattern is affecting life in a positive way will be beneficial. Asking open-ended questions and listening to the patient describe his or her new routine and how it feels to the patient will allow the patient to share the story and increase awareness about how the new behavior is affecting his or her life. At this stage, reassessing goals and committing to new goals will help with focus and motivation. Involving family and friends to act as support systems for the patient to help sustain the new routine will be beneficial to the process of change at this stage.

Self-liberation, helping relationships, and counterconditioning can all be used in the action stage. The processes that enter the behavior change journey at the action stage are reinforcement management and stimulus control. With reinforcement management, the patient receives rewards from the simple statements to self, "Great job. You achieved that goal," to the acknowledgment of a spouse of close friend saying, "You ate dinner at home six times this week which means you met your goal. Congratulations!" Planning rewards such as watching a favorite movie with a friend if the patient packs a lunch 5 days 1 week, is another way to manage the reinforcements. Food rewards should generally be avoided. For example, a patient might want to justify splurging on dessert as a reward for

eating healthy options all week. In theory, this seems okay; however, this can create a reward/punishment mindset and foster an unhealthy relationship surrounding food for the patient. A better reward would be getting to purchase a new gym outfit or song to be played during workouts. These are all external rewards that will hopefully lead to acknowledging, experiencing, and enjoying intrinsic rewards like more energy, a sense of calm, a feeling of satiety, and perhaps regular bowel movements, if extra fiber from vegetables was added.

Stimulus control brings in the environment. There are cues and triggers throughout the home, car, and workplace that can act as detractors or promoters of the healthy goal behaviors. For example, throwing out all the candy in the house would be one example of stimulus control. Filling the refrigerator with fresh vegetables and fruits would be another. Buying portion control containers and a new thermal lunch bag would also serve as stimulus control techniques that would nudge the patient in the direction of bringing lunch to work and making sure it was appropriate serving sizes.

After action comes maintenance. Patients in the maintenance stage have been following the healthy pattern for over 6 months. This routine is becoming more like a habit now. Yet, the patient still needs stage-appropriate counseling. Ignoring the patient at this point and assuming that the healthy habit will stick for a lifetime is unrealistic and detrimental to the patient. Reviewing potential obstacles in the future such as a holiday celebration or a vacation that will take the patient outside his or her usual circumstances will be important to discuss during a nutrition counseling session. Brainstorming ways around the upcoming challenging situation will be useful to the patient at this stage of change. Tapping into the patient's original motivation to embark on this behavior change journey will allow the patient to reconnect with his or her initial goals and intent. The current motivation might be different than the previous one. In maintenance, many patients are ready to act as mentors and help people that are just beginning their journey of behavior change which can be a new motivator. Exploring how the patient is feeling and asking the patient to identify some new long-term goals as well as short-term goals will keep him or her focused and can enhance motivation.

Effective processes for maintenance include the same ones for action: helping relationships, counterconditioning, stimulus control, and reinforcement management. It is important to continue to reward the patient for the healthy behaviors by noting the change in body weight, hemoglobin A1C, or blood pressure when appropriate. In addition, continuing to create triggers for healthy eating patterns is essential. Making sure the old cues for unhealthy eating are removed and stay out of the way of the patient will ensure continued adherence to the new plan. When people are in maintenance, they still need attention, brainstorming, and reinforcements.

It is important to remember that the Transtheoretical Model of Change is not a smooth process that follows step-by-step. It is more of a spiral staircase in which patients can fall from action into contemplation within a few days. These slips tend to happen around stressful events and big life changes such as marriage, divorce, moving residences, getting a new job, a change to the family structure with the birth of a baby, an aging parent moving in, or children moving away to college [130]. Asking questions and determining the patient's stage of change at each visit is essential to providing the nutrition counseling the patient needs at that particular visit.

Moving Toward A Plan

Knowing where the patients are on their journeys to optimal health and wellness as well as understanding where they want to go will enable the provider to act as a coach, as in stagecoach, helping the patient find their way from point A (their current location) to point B (their goal destination). Asking the patient to imagine how he or she will look, walk, talk, act, move, and feel will allow the patient to create a vision of his or her best self. The more detailed and the more the patient can incorporate and elaborate on the different senses in the vision the better. This allows the patient to use the left and right sides of their brains to express themselves. They will be using the left side of their brain as they describe it to the provider and the right side as they visualize themselves in the future.

This discovery process of identifying where the patient is now, where he or she wants to go, what options there are to move forward and which one of those options will make the most sense at a particular time, are basic parts of the coaching process. The *GROW* model (*G-Goals, R-Reality, O-Options, W-Way forward*) was originally described in the 1980s by Sir John Whitmore, Alexander Graham, and colleagues and serves as a framework toward goal setting and problem solving. Sir John Whitmore's book, *Coaching for Performance* [131], explains the model in depth and is used by coaches today [132]. With GROW, the patient states the goals, determines where they are currently, identifies options that will get him or her closer to the goal, and then commits to a way forward.

Once the vision is set, the next step is to craft behavioral steps to get there. In 1960s, Edwin Locke and Gary Latham wrote about goal setting theory for businesses [133]. He discussed how goals need five basic elements: (1) Clarity, (2) Challenge, (3) Complexity, (4) Commitment, (5) Feedback. Goals that are specific and clear are the most effective. In addition, it is important to select a goal that is difficult enough to keep the patient engaged and not frustrated or anxious. Yet, the goal cannot be too easy that the patient becomes bored and disengaged. Mihaly Csikszentmihalyi, PhD describes the optimal level of stress (challenge and complexity) for an activity called eustress [134]. He coined the term "flow" meaning that a person is experiencing so much joy during the activity that he or she loses track of time. In this case, the patient's skills meet the challenge level, and there is no anxiety or boredom. The task is just right for fulfillment and flow. The provider and patient co-create a goal that enables a person to reach this state of flow.

Practical Tip Crafting a goal that provides the right amount of challenge and complexity.

One way to ensure that a goal is at the right level is to ask the patient, "How confident are you that you can achieve this goal on a scale of 1–10. One is not at all confident and ten is very confident." If the patient says that they are at a five on the confidence scale, then the provider can help the patient to readjust the goal so that the patient feels more confident. "What would it take to move the goal up on that scale?" Or "How you can adjust it to feel more confident that you can complete it?" This helps the patient to set a goal and achieve it, which increases self-efficacy.

Another factor in goal setting is commitment. How committed is the patient to the goal? If the goal is relevant and connected to the patient's priorities, then the patient is more likely to be motivated to work toward the goal. This comes from the Adult Learning theory described by Malcolm Knowles, PhD, which states that adult learners are autonomous, self-directed, practical, goal-oriented, and relevancy-oriented [135].

When someone commits to a goal by writing it down or committing to it verbally, they are raising the level of importance of that goal. The provider can ask a question to check on the level of importance of the goal, "How important is the goal to you using a scale of 1–10. One is not important and ten is very important." If the goal is rated at a five, then the provider can ask the patient how to adjust the goal to increase the level of importance.

Directly linking a goal to something relevant in the patient's life will help to demonstrate the importance. For example, if the patient is trying to change her diet to work on weight management, explore why this might be important to her. Perhaps the weight loss will help fulfill her vision of playing with her grandchildren at the park or enable her to go on an active vacation with her family. One can point out the fact that fruits and vegetables have a significant amount of water, which helps to create the feeling of satiety [136]. Eating fruits and vegetables will also nourish the body with vitamins, minerals, complex carbohydrates, phytonutrients, and antioxidants with relatively few calories (i.e., nutrient-dense foods), this will allow the body to function at its highest capacity. This link between the behavior (eating nutrient-dense foods) and the outcome (improved functionality, weight loss, etc.) is critical to highlight, as this is what encourages patients to buy into the process. Research in patients with heart failure demonstrates that these patients are more likely to complete their health goals if they are connected to their life goals [137].

After setting an appropriate goal and committing to it, the patient needs to be held accountable for that goal in order to keep the patient engaged in the process of change and to stay focused. If goals are set, and no one follows up, then the patient is likely to lose interest in the goals. Identifying a family member or friend to help check in with the patient can be an effective source of accountability. Having the office send a postcard, email, or text message checking in on the goal could be a useful technique. It could be as simple as, "Hello from Dr. Smith's office. We are just checking in on your goals." Some offices have Population Health Coordinators who are helping to remind patients of their visits and check in on home monitoring of blood pressures and glucose levels. If there is someone available to make calls to check in on dietary goals, this could enhance the level of accountability [138]. Medical assistants trained in health coaching have also been shown to significantly improve the quality of care in patients with poorly controlled chronic diseases [139, 140]. From research on reminding adolescent patients about immunizations, it has been demonstrated that a postcard, an email, or a text could all serve to remind the patients of appointments with the postcard being the favored form of reminder, but text messages and emails being the most effective [141]. More research on accountability reminders for dietary goals needs to be completed to determine the best options in the nutrition setting.

When counseling patients on nutrition, there is a mnemonic to guide the process of goal setting. SMART goals are goals that are specific, measureable, action-oriented, realistic, and time-sensitive. Depending on the patient's stage of change, the action part of the goal might be physically oriented or it could be cognitively oriented. For example, if the patient is in a contemplative stage of change, then searching for recipes on line that include whole food ingredients with half the plate fruits and vegetables, a quarter of the meal complex carbohydrates, and a quarter of the plate a healthy protein might be an appropriate SMART goal for that week. It is specific in that it states a particular task, researching recipes online. It is measurable because the patient can report back that they did it or not. If the goal included printing out the recipes that the patient thinks looked the most delicious and then the patient brought the recipes to the next visit, it would be measurable. It is action-oriented because there is something concrete that the patient is going to do. Action-oriented does not mean that the person needs to start eating a particular diet right away. The act of searching for recipes is action-oriented. It is more mental than physical action. The goal is realistic if the patient states that they are comfortable and confident that they can achieve the goal. The only way to determine this is to ask the patient. Using a confidence scale from 1 to 10 is a good method of checking this. Lastly, the goal needs to be time-sensitive, meaning that there is an endpoint and an expectation that the goal will be completed within a certain time frame, usually 1 week. In this case, specifically stating the day that the recipe search will take place is a critical part of making the goal time-sensitive. For example, the goal might be to research recipes on Wednesday after work, and if that does not work out, a backup plan of another day such as Saturday morning is helpful.

SMART goals	SMART goals checklist
S=Specific	Is this a goal that is detailed and explicitly stated?
M=Measurable	How will I measure the outcome of the goal?
A=Action-oriented	Will I be doing something in particular to reach this goal?
R=Realistic	How likely is it that I can attain this goal in the stated time frame?
T=Time-sensitive	What is the deadline for meeting this goal?

Long-Term and Short-Term Goals

Patients can create a vision of themselves 10 years in the future or even 20 or 30 years, depending on their age. Crafting 1-year goals can help a patient to reach these long-term visions. The 1-year goal is created using the *SMART* mnemonic. However, in this case the time frame is 1 year. Behavioral goals

are the ones to focus on instead of number goals such as losing 20 lb. For example, if a patient was eating dinner at home 1 day out of the week and eating out at fast food restaurants for dinner 6 days out of the week, a long-term goal might be eating dinner at home 6 out of 7 days a week by the end of 1 year. A 6-month goal to go along with this 1-year goal might be to eat dinner at home 3 days out of the week, and a 3-month goal might be to eat dinner at home 2 days out of the week on a consistent basis. An appropriate 1-month goal would be to eat dinner at home 1 day out of the week for 2 weeks straight. An initial 1-week goal for working toward these longer-term goals might be to go shopping for healthy food at the grocery store 1 day during the current week and specifying a day, such as Friday.

Short-term goals that might accompany the long-term goal of eating dinner at home 6 days out of the week include:

- 1. Identify recipes for healthy dinners
- 2. Make a list of food to buy at the grocery store to stock up on healthy options
- 3. Go shopping once a week (on specified days) to make sure to have healthy options at home
- Consider batch cooking on Sundays or another free day so that leftovers can serve as dinner on days when there is less time to prepare a meal
- 5. Plan menus on Sundays or another day that is flexible and free
- 6. Try to go to the grocery store one day and prepare a meal with the ingredients right away
- If cooking the meal is too time-consuming, then identify prepared meals that are healthy choices, and ideally made daily at a local supermarket
- 8. When eating out, identify meals that suit the healthy plate and include at least half the plate of fruits and vegetables, one quarter complex carbohydrates, and one quarter healthy protein
- 9. Consider taking cooking classes at a local recreation center or partner with a friend that enjoys cooking to learn from him or her
- 10. Purchase freezer-safe portion-controlled containers to store food after batch cooking
- Find healthy frozen options with low salt and preservatives that can be used when there are no easy options

Depending on the stage of change of the patient and the desires of the patient, the short-term goals will vary. Checking that they are SMART goals is important. Collaborating with the patient and making sure to fully understand the patient's needs and aspirations are the most critical steps in the process of cocreating goals with patients using the COACH approach.

Tools and Tips for Nutrition Counseling

Nutrition counseling requires the provider to be knowledgeable about healthy foods, food patterns, national guidelines, and specific diets. Also, the provider needs to focus on creating a connection with collaboration and the COACH approach. In addition to a clear, consistent message and striving to be a messenger that uses the COACH approach, the provider can use specific tools that enhance the nutrition counseling session. Figures such as the myplate.gov [142] provide examples of a healthy plate and demonstrate appropriate proportions of vegetables/fruits, complex carbohydrates, and protein. For portion control of specific foods, there are plastic replicas to demonstrate to patients. There are also charts and guidelines for portions of specific foods available on the web that are patient-friendly [40]. Using a hand as a portion tool can be effective and memorable. The patient will always have the portion tool handy. There are portion charts that can be created or downloaded and then hung on the walls of the examining rooms. For patients who are working to closely monitor their intake and calories, there are a number of apps and web sites to help (e.g., Im2Calories, LoseIt, MyFitnessPal, SparkPeople). These web sites and technology devices help count calories and percent of fat, protein, and carbohydrates consumed in a meal. Some apps only need a photo to make calculations and provide feedback. This can be useful for certain patients.

Logs, diaries, and books are tools that work for many patients. A nutrition diary created with paper and pencil, on the computer, or through a web site is easy to use. The 1-day dietary recall is a simple test to determine all the foods that a patient ate in 1 day including meals (breakfast, lunch, and dinner) as well as snacks and beverages. The daily dietary log involves being aware and conscious of eating patterns as they are happening. The recall requires patients to think about the choices they made the previous day. However, if the patient knows they will be using a dietary recall or a food diary, then they are often more careful with the choices they make the day they plan to log their intake. Using a log as a tracking device for specific foods is another way to focus on the positive goal behaviors, such as consuming more vegetables and fruit each day. Asking patients to note which vegetables they consumed, what color the vegetables were, how they tasted, how much they enjoyed them, and when they ate them is useful information and a good way to keep the focus on including whole foods at every meal. The act of reporting on their food choices invites patients to be more mindful of their choices and when combined with other self-monitoring like step count, water consumption, and weight can lead to sustained weight loss [143, 144].

Cookbooks and other health books including *The Mayo Clinic Diet* [30], *The Spectrum Diet* [32], *The DASH Diet* [25, 26], *The Full Plate Diet* [145], and the *New American Heart Association Cookbook* [146] are useful tools for patients interested in reading and who are searching for new, healthy recipes.

For patients trying to lose weight, tracking calories consumed and calories burned can be helpful for some numbers-oriented people. There are wearable devices like the Nike Fuelband and the FitBit that can be used for this function. Measuring weekly weights, taken at the same time of the day and on the same scale, is a method of tracking that can serve to keep people on target with their weight management goals. Noting BMI at the start of a dietary intervention and then 4 weeks later is another way to track progress. Beyond the scale, there are many other modalities to check progress. Waist circumference and waist-to-hip ratio are measurements that provide more information about the location of fat in the body. Abdominal adiposity is associated with a greater risk of disease [147]. With the jeans test, the patient uses a favorite pair of jeans or pants to track his or her progress with the weight loss regimen. If the jeans fit well or loose, then they know they are making progress. The belt buckle serves a similar function by monitoring which belt buckle hole a patient uses. As a patient loses weight or changes their body composition, they will use a hole that is further from the end of the belt. Many people notice that their skin clears and they experience fewer cavities when they are eating a diet high in whole foods and low in processed foods. Tracking breakouts associated with certain food consumption or number of cavities since adhering to a healthy diet are options for people who are not seeking to lose weight but are seeking a more healthy eating pattern. Everyone who is planning to eat a healthier diet has his or her own rationale for the change, and it is not always weight-related. It is best to ask an open-ended question after patients are following a healthy eating pattern for 3-6 months and inquire about what types of health benefits they have noticed. The message of "Whole foods, mostly plants, not too much," as Michael Pollan recommends has multiple benefits [22, 24].

Healthy habits are interconnected. A person's level of physical activity, sleep, stress, alcohol consumption, and other daily habits influence dietary habits. Thus, a good tip is to ask about other lifestyle habits while counseling on nutrition. Sometimes a patient might be in the stage of preparation for increasing physical activity and contemplation for starting a new pattern of healthy eating. In this case, focusing on physical activity first would be recommended. If the patient is struggling with sleep, then this might be an area to tackle, as sleep deprivation is linked to lower levels of leptin [148]. It is possible to work on different habits at one time [126]. The important part of nutrition counseling is to meet the needs of the patient. The provider might identify areas of concern in dietary patterns and can ask open-ended questions to further explore the behavior patterns around food. Through motivational interviewing, the provider can direct the patient with questions that evoke change talk. In this way, a provider can create an environment conducive to progressing through the stages of change. Another tip is to take the environment into account and "Shaping the Path," as the Heath brothers call it. This concept was mentioned in the introduction of the chapter. Specifically crafting the environment for sustained behavior change is an important piece of nutritional counseling, and it is often overlooked. The social ecological model of change emphasizes the importance of a person's social network and surroundings [11]. Patients are individuals connected to other people and influenced by their cultural norms. Thus, exploring the patient's environment at home and at work is a necessary step in successful nutrition counseling aimed at sustainable change. A useful tip is to ask a patient how close the nearest supermarket is to his or her house. Recent research reveals that the closer a patient lives to a supermarket, the more fruit and vegetable consumption increases, and the greater the improvements in weight management in obesity interventions [149]. Although patients might not be able to move closer to a supermarket, they might be able to identify one close to work or on their way to work or near a location that they frequent daily.

"Shaping the Path" in the home and work environment helps. If the provider starts asking openended questions about what is stored in the cabinets and refrigerator, then the patient receives a strong message that what is available is what is eaten. Keeping healthy options in view and easily accessible will increase the likelihood that those options will be consumed. Clearing out processed foods and "junk" foods will also help. Patients can always go to a convenience store and purchase cookies, cakes, chips, and other nutrient-poor, calorie-rich options. However, making a trip to the store is a deterrent, an obstacle to consuming these unhealthy options. In addition to examining the food in the house, asking about family and friends that live in the house will help too. Some people are supporters, and they empower the patient on their way to behavior change toward health and wellness [150]. On the other hand, other people act as saboteurs and create barriers to change, such as bringing into the home the foods that the person is actively trying to avoid, encouraging the patient to go out to eat at restaurants, serving extra-large portions, or indulging in rich desserts. Beyond the home, the patient might have a job and a work environment that is unhealthy. In this case, discussing the possibility of bringing lunch into work as well as some snacks will be a practical approach to strategize around the obstacle of a junk-food-laden cafeteria. Another tip is to teach patients about the social ecological model of change. In fact, hanging up a model of the social ecological model of change is a tool that can remind the provider and the patient about the importance of surroundings and "shaping the patient's path."

One influential connection for each patient is the provider. A health care provider often serves as an expert opinion in health but also can serve as a role model for patients. Studies show that patients view physicians who disclose some of their own personal efforts at maintaining healthy habits as more believable and more motivating [151] than those that just advise patients on healthy habits. In a study by Erika Frank, MD, MPH, the researchers created a counseling video of a physician that utilized nonverbal, healthy cues that included placing an apple on the physician's desk and having the physician display a bike helmet along with verbal cues including having the physician making a couple of simple statements indicating that he was fitting healthy habits into the daily routine by having an apple for a snack and biking to work [151]. In addition, research from over a decade ago demonstrated that physicians preach what they practice. If physicians do aerobic exercise, they counsel on it, and if they do strength training, they counsel on it. If a physician does not do strength training or aerobic exercise, they will not counsel on it [152]. Furthermore, medical students who have healthy personal practices are more likely to report counseling patients on preventive interventions [153]. Research on the number of physicians who exercise and the number of physicians who eat fruits and vegetables show that 58 % of physicians exercise 3 or more days per week and 60 % eat at least five servings of fruits and vegetables during 4 or more days per week [154]. Hippocrates said, "Physician heal thyself." The goal is "progress not perfection." Health care workers who are working on their own health and wellness are motivating to their patients. When patients see physicians, nurses, and nutritionists working on their own healthy eating patterns, they realize that diet must be important because all of these busy, knowledgeable people are striving to eat healthy options and prioritize their health. As Gandhi said, "Be the change you wish to see in the world."

Conclusion

This chapter on nutritional counseling provides the blueprint for creating a behavior change arch for each patient, which leads them to a journey of self-management, exploration, and healthy eating patterns. Both conveying powerful and personally relevant nutrition messages to the patient and using effective messenger techniques that empower patients and draw out the patient's intrinsic motivation for adopting healthy eating patterns are vital for effective counseling in nutrition. The message and the messenger need to work synergistically in order to create the foundation of the behavior change arch (message and messenger=cornerstones of the arch in Fig. 3.1). Eliciting and evoking the patient's own internal motivators for change is key (motivation=keystone of arch in Fig. 3.1). The arch serves as the entry way into the behavior change journey and facilitates lasting and sustainable patient transformation.

Adopting healthy eating patterns has the potential to prevent, treat, and reverse disease. Most people know they should eat fruits and vegetables. They have heard this basic message before. However, they might not know the reasons why or understand how fruits and vegetables work to reduce their blood pressure, control their weight, and maintain a stable blood sugar level. Personalizing the message and making it relevant to the patient, as well as giving it some emotional charge by relating healthy eating to their personal goals and priorities, will help convey the message in a way that impacts their behavior. Carefully conveying the message with curiosity, openness, appreciation, compassion, and honesty with the COACH approach enables the provider to connect with the patient and build a therapeutic relationship. Using self-determination theory basics of allowing autonomy, increasing confidence, and fostering relatedness works to draw out the patient's intrinsic motivation. Motivational interviewing, appreciative inquiry, and the transtheoretical model of change all help to propel the patient forward on the behavior change journey.

Jim Loehr and Tony Schwartz argue that life is about managing energy, in their book *The Power of Full Engagement*. It requires mindfulness and self-awareness to manage one's own energy level. This energy is necessary in order to stay up-to-date with the literature (the message), to serve as the charismatic adult from whom patients and others gather strength [155, 156] (the messenger), and to ultimately empower patients to discover their own intrinsic motivation. Providers can increase their own positive energy by increasing their use of the COACH approach for behavior change in nutrition counseling. According to Loehr and Schwartz, the key muscles fueling positive emotional energy are self-confidence, self-control, interpersonal effectiveness, and empathy [157]. The COACH approach allows providers to practice interpersonal effectiveness and empathy that will fuel both the provider and the patient. A provider gains self-confidence by reading textbooks like this one, increasing knowledge, developing new skills, practicing them, and discovering new tools, like the ones presented in this chapter. In this way, this chapter and this textbook can serve not only as a source of behavior change and nutrition information but also as a source of fuel and energy for the practice of lifestyle medicine.

References

- 1. Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, et al. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med. 2001;345(11):790–7.
- 2. Schubert MM, Sabapathy S, Leveritt M, Desbrow B. Acute exercise and hormones related to appetite regulation: a meta-analysis. Sports Med. 2014;44(3):387–403.
- Shipe M. Exercising with coronary heart disease. ACSM [Internet]. 19 Jan 2012 [Cited 19 Mar 2016]. Available from: https://www.acsm.org/public-information/articles/2012/01/19/exercising-with-coronary-heart-disease.
- Patterson RE, Kristal AR, Tinker LF, Carter RA, Bolton MP, Agurs-Collins T. Measurement characteristics of the Women's Health Initiative food frequency questionnaire. Ann Epidemiol. 1999;9(3):178–87.

- Frates EP, Moore MA, Lopez CN, McMahon GT. Coaching for behavior change in physiatry. Am J Phys Med Rehabil. 2011;90(12):1074–82.
- Frates EP, Moore M. Health and wellness coaching skills. In: Rippe JM, editor. Lifestyle medicine. 3rd ed. Boca Raton: CRC Press; 2013. p. 343–62.
- 7. Rollnick S, Miller WR. What is motivational interviewing? Behav Cogn Psychother. 1995;23(4):325–34.
- Deci EL, Ryan RM. Motivation, personality, and development within embedded social contexts: an overview of self-determination theory. In: Ryan RM, editor. Oxford handbook of human motivation. Oxford: Oxford University Press; 2012. p. 85–107.
- 9. Rhodes RE, Bruijn GJ. What predicts intention-behavior discordance? A review of the action control framework. Exer Sport Sci Rev. 2013;41(4):201–7.
- 10. Fogg Method [Internet]. c2013 [Cited 15 Feb 2015]. Available from: http://www.foggmethod.com/.
- Dahlberg LL, Krug EG. Violence-a global public health problem. In: Krug E, Dahlberg LL, Mercy JA, et al., editors. World report on violence and health. Geneva: World Health Organization; 2002. p. 1–56.
- Christakis NA, Fowler JH. Connected: the surprising power of our social networks and how they shape our lives. New York: Little, Brown and Company; 2009.
- Fogg BJ. The behavior grid: 35 ways behavior can change. In: Proceedings of the 4th international conference on Persuasive Technology. Association for Computing Machinery. New York; 2009. p. 42–6.
- 14. U.S. Department of Health and Human Services and U.S. Department of Agriculture [Internet]. 2015–2020 Dietary guidelines for Americans. 8th ed. c2015 [Cited 15 Feb 2015]. Available from: http://health.gov/ dietaryguidelines/2015/guidelines/.
- Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. N Engl J Med. 2003;348:2599–608.
- Rees K, Harley L, Flowers N, Clarke A, Hopper L, Thorogood M, et al. 'Mediterranean' dietary pattern for the primary prevention of cardiovascular disease. Cochrane Database Syst Rev. 2013;(8):CD009825.
- Estruch R, Ros E, Salas-Salvadó J, Covas MI, Corella D, Arós F, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med. 2013;368:1279–90.
- Nordmann AJ, Suter-Zimmermann K, Bucher HC, Shai I, Tuttle KR, Estruch R, et al. Meta-analysis comparing Mediterranean to low-fat diets for modification of cardiovascular risk factors. Am J Med. 2011;124:841–51.
- Serra-Majem L, Roman B, Estruch R. Scientific evidence of interventions using the Mediterranean diet: a systematic review. Nutr Rev. 2006;64:S27–47.
- Buettner D. The blue zones solution: eating and living like the world's healthiest people. Washington, DC: National Geographic Society; 2015.
- 21. Heath C, Heath D. Switch: how to change things when change is hard. New York: Broadway Books; 2010.
- 22. Pollan M. In defense of food: an eater's manifesto. New York: Penguin Group; 2008.
- Harvard TH. Chan School of Public Health [Internet]. Healthy eating plate and healthy pyramid. c2013 [Cited 16 Feb 2016]. Available from: http://www.hsph.harvard.edu/nutritionsource/healthy-eating-plate/.
- 24. Katz DL, Meller S. Can we say what diet is best for health? Annu Rev Public Health. 2014;35:83–103.
- U.S. Department of Health & Human Services. National Heart, Lung, and Blood Institute [Internet]. Description of the DASH eating plan. c2015. [Cited 20 Feb 2016]. Available from: http://www.nhlbi.nih.gov/health/healthtopics/topics/dash.
- 26. Heller M. The DASH diet weight loss solution: 2 weeks to drop pounds, boost metabolism, and get healthy (a DASH diet book). New York: Grand Central Life & Style; 2012.
- American Heart Association [Internet]. The American Heart Association's diet and lifestyle recommendations. c2015 [Cited 20 Feb 2016]. Available from: http://www.heart.org/HEARTORG/HealthyLiving/HealthyEating/ Nutrition/The-American-Heart-Associations-Diet-and-Lifestyle-Recommendations_UCM_305855_Article.jsp#. VskBrZMrKV4.
- American Cancer Society [Internet]. ACS guidelines on nutrition and physical activity center for cancer prevention. c2016 [Cited 20 Feb 2016]. Available from: http://www.cancer.org/healthy/eathealthygetactive/ acsguidelinesonnutritionphysicalactivityforcancerprevention/.
- American Diabetes Association [Internet]. Diabetes meal plans and a healthy diet. c2016 [Cited 20 Feb 2016]. Available from: http://www.diabetes.org/food-and-fitness/food/planning-meals/diabetes-meal-plans-and-a-healthydiet.html.
- 30. The Mayo Clinic diet. Intercourse: Da Capo Lifelong Books; 2010.
- Hyman M. The blood sugar solution 10-day detox diet: activate your body's natural ability to burn fat and lose weight fast. New York: Little, Brown and Company; 2014.
- 32. Ornish D. The spectrum: how to customize a way of eating and living just right for you and your family. New York: Ballantine Books; 2007.
- 33. Katz DL, Hu FB. Knowing what to eat, refusing to swallow it. The Huffington Post [Internet]. 2 July 2014 [Cited 16 Feb 2016]. Available from: http://www.huffingtonpost.com/david-katz-md/knowing-what-to-eat-refus_b_5552467 .html/.

- 34. Chow CK, Redfern J, Hillis GS, Thakkar J, Santo K, Hackett ML. Effect of lifestyle-focused text messaging on risk factor modification in patients with coronary heart disease: a randomized clinical trial. JAMA. 2015;314(12):1255–63.
- 35. Wasnick B. From mindless eating to mindlessly eating better. Physiol Behav. 2010;100(5):454-63.
- Healthy Children.org [Internet] Portions and Serving Sizes. c2016 [Cited 20 Feb 2016]. Available from: https:// www.healthychildren.org/English/healthy-living/nutrition/Pages/Portions-and-Serving-Sizes.aspx.
- 37. Dairy Council of California [Internet] Serving-Size Comparison Chart. c2012 [Cited 20 Feb 2016]. Available from: http://www.healthyeating.org/Portals/0/Documents/Schools/Parent%20Ed/Portion_Sizes_Serving_Chart.pdf.
- WebMD [Internet] Serving Size. c1995–2015. [Cited 8 Mar 2016]. Available from: http://www.webmd.com/diet/ serving-size.
- American Heart Association [Internet] Suggested Servings from Each Food Group. c2016 [Cited 8 Mar 2016]. Availablefrom:http://www.heart.org/HEARTORG/HealthyLiving/HealthyEating/Nutrition/Suggested-Servings-from-Each-Food-Group_.
- 40. National Institute of Health MedlinePlus [Internet] Portion Size. c2016. [Cited 20 Feb 2016]. Available from: https://www.nlm.nih.gov/medlineplus/ency/patientinstructions/000337.htm.
- 41. U.S. Geological Survey [Internet]. The USGS Water Science School. The Water in you. c2015 [Cited 20 Feb 2016]. Available from: http://water.usgs.gov/edu/propertyyou.html.
- 42. Institute of Medicine [Internet]. Dietary reference intakes: water, potassium, sodium, chloride, and sulfate. c2004. [Cited 20 Feb 2016]. Available from: https://iom.nationalacademies.org/~/media/Files/Activity%20Files/ Nutrition/DRIs/DRI_Electrolytes_Water.pdf.
- 43. Valtin H. "Drink at least eight glasses of water a day". Really? Is there scientific evidence for "8×8"? Am J Physiol Integr Comp Physiol. 2002;283(5):R993–1004.
- 44. Whitlock EP, Orleans T, Pender N, Allan J. Evaluating primary care behavioral counseling interventions: an evidence-based approach. Am J Prev Med. 2002;22(4):267–84.
- 45. Glasgow RE, Seth E, Miller DC. Assessing delivery of the five 'As' for patient-centered counseling. Health Promot Int. 2006;21(3):245–55.
- 46. Alexander SC, Cox ME, Boling-Turer CL, Lyna P, Ostbye T, Tulsky JA. Do the five A's work when physicians counsel about weight loss? Fam Med. 2011;43(3):179.
- 47. Jay M, Gillespie C, Schlair S, Sherman S, Kalet A. Physicians' use of the 5As in counseling obese patients: is the quality of counseling associated with patients' motivation and intention to lose weight? BMC Health Serv Res. 2010;10:159.
- Vallis M, Piccinini-Vallis H, Sharma AM, Freedhoff Y. Modified 5 as minimal intervention for obesity counseling in primary care. Can Fam Phys. 2013;59(10):27–31.
- 49. Dulloo AG, Montani JP. Pathways from dieting to weight regain, to obesity and to the metabolic syndrome: an overview. Obes Rev. 2015;16 Suppl 1:1–6.
- 50. Montani JP, Schutz Y, Dulloo AG. Dieting and weight cycling as risk factors for cardiometabolic diseases: who is really at risk? Obes Rev. 2015;16 Suppl 1:7–18.
- Dulloo AG, Jacquet J, Montani JP, Schutz Y. How dieting makes the lean fatter: from a perspective of body composition autoregulation through adipostats and proteinstats awaiting discovery. Obes Rev. 2015;16 Suppl 1:25–35.
- 52. Fisher EB, Strunk RC, Highstein GR, et al. A randomized controlled evaluation of the effect of community health workers on hospitalization for asthma: the asthma coach. Arch Pediatr Adolesc Med. 2009;163(3):225–32.
- 53. Benzo R, Vickers K, Novotny PJ, Tucker S, Hoult J, Neuenfeldt P. Health coaching and COPD re-hospitalization: a randomized study. Am J Respir Crit Care Med. 2016. First published online March 08, 2016 as doi:10.1164/ rccm.201512-2503OC [Epub ahead of print].
- 54. Oliver JW, Kravitz RL, Kaplan SH, Meyers FJ. Individualized patient education and coaching to improve pain control among cancer outpatients. J Clin Oncol. 2001;19:2206–12.
- Edelman D, Oddone EZ, Liebowitz RS, Yancy WS, Olsen MK, Jeffreys AS. A multidimensional integrative medicine intervention to improve cardiovascular risk. J Gen Intern Med. 2006;21:728–34.
- Vale MJ, Jelinek MV, Best JD, Dart AM, Grigg LE, Hare DL. Coaching patients on achieving cardiovascular health (COACH). Arch Intern Med. 2003;163:2775–83.
- Whittemore R, Melkus GD, Sullivan A, Grey M. A nurse-coaching intervention for women with type 2 diabetes. Diabetes Educ. 2004;30(5):795–804.
- Sacco W, Morrison AD, Malone JI. A brief, regular, proactive telephone "coaching" intervention for diabetes. Rationale, description, and preliminary results. J Diabetes Complications. 2002;18:113–8.
- 59. Koenigsberg MA, Bartlett D, Cramer JS. Facilitating treatment adherence with lifestyle changes in diabetes. Am Fam Physician. 2004;69(319–20):323–4.
- 60. Debar LL, Ritenbaugh C, Aickin M, Orwoll E, Elliot D, Dickerson J. A health plan-based lifestyle intervention increases bone mineral density in adolescent girls. Arch Ped Adol Med. 2006;160:1269–76.

- Holland SK, Greenberg J, Tidwell L, Malone J, Mullan J, Newcomer R. Community-based health coaching, exercise, and health service utilization. J Aging and Health. 2005;17:697–716.
- Tidwell L, Holland SK, Greenberg J, Malone J, Mullan J, Newcomer R. Community-based nurse health coaching and its effect on fitness participation. Lippincotts Case Manag. 2004;9(6):267–79.
- Heimendinger J, Uyeki T, Andhara A. Coaching process outcomes of a family visit nutrition and physical activity intervention. Health Educ Behav. 2007;34:71–89.
- 64. Tucker LA, Cook AJ, Nokes NR, Adams TB. Telephone-based diet and exercise coaching and a weight-loss supplement result in weight and fat loss in 120 men and women. Science Health Promotion. 2008;23(2):121–9.
- 65. Ohashi J, Katsura T. The effects of coaching on salivary cortisol stress marker in mothers with young children, a randomized controlled trial. J Rural Med. 2015;10(1):20–8.
- 66. Hill B, Richardson B, Skouteris H. Do we know how to design effective health coaching interventions: a systematic review of the state of the literature. Am J Health Promot. 2015;29(5):e158–68.
- 67. Kivela K, Elo S, Kynga H, Kaariainen M. The effects of health coaching on adult patients with chronic diseases: a systematic review. Patient Educ Couns. 2014;92(2):147–57.
- Wolever RQ, Simmons LA, Sforzo GA, Dill D, Kaye M, Bechard EM, Southard ME. A systematic review of the literature on health and wellness coaching: defining a key behavioral intervention in healthcare. Glob Adv Health Med. 2013;2(4):38–57.
- "Curiosity." Merriam-Webster.com [Internet] Merriam Webster. c2015. [Cited 8 Mar 2016] Available from: http:// www.merriam-webster.com/dictionary/curiosity.
- 70. Kabat-Zinn J. Wherever you go, there you are. New York: Hyperion Books; 1994.
- 71. Fitzgerald FT. Curiosity. Ann Intern Med. 1999;130(1):70-2.
- 72. Dossey L. The power of premonitions. London: Hay House UK Ltd; 2009.
- Lyubomirsky S. The how of happiness: a new approach to getting the life you want. New York: The Penguin Press; 2008.
- 74. Kashdan T. Curious?: discover the missing ingredient to a fulfilling life. New York: Harpercollins Publishers; 2010.
- Kashdan TB, Rose P, Fincham F. Curiosity and exploration: facilitating positive subjective experiences and personal growth opportunities. J Pers Assess. 2004;82(3):291–305.
- Taberner K, Siggins K. The power of curiosity: how to have real conversations that create collaboration, innovation and understanding. New York: Morgan James Publishing; 2015.
- 77. Peters M. The idea of openness: open education and education for openness. In:Peters M, Besley T, Gibbons A, Žarnić B, Ghiraldelli P, editors. The encyclopaedia of educational philosophy and theory. 2010. Available from: http://eepat.net/doku.php?id=open_education_and_education_for_openness.
- Laidlaw TS, Kaufman DM, Sargeant J, MacLeod H, Blake K, Simpson D. What makes a physician an exemplary communicator with patients? Patient Educ Couns. 2007;68(2):153–60.
- Duberstein P, Meldrum S, Fiscella K, Shields CG, Epstein RM. Influences on patients' ratings of physicians: physicians demographics and personality. Patient Educ Couns. 2007;65(2):270–4.
- Cannarella LR, Jacques CH, Donovan C, Cottrell S, Buck J. Managing difficult encounters: understanding physician, patient, and situational factors. Am Fam Physician. 2013;86(6):419–25.
- 81. Teutsch C. Patient-doctor communication. Med Clin North Am. 2003;87(5):1115-45.
- Gibson S. On judgment and judgmentalism: how counselling can make people better. J Med Ethics. 2005;10:575–7.
- Vujanovic AA, Bonn-Miller MO, Marlatt GA. Posttraumatic stress and alcohol use coping motives among a traumaexposed community sample: the mediating role of non-judgmental acceptance. Addict Behav. 2011;36(7):707–12.
- Matta C. Exercises for Non-judgmental Thinking [Internet] Psych Central. 2012. [Cited 29 Feb 2016]. Available from: http://blogs.psychcentral.com/dbt/2010/06/exercises-for-non-judgmental-thinking/.
- 85. Dweck CS. Mindset: the new psychology of success. New York: Ballantine Books; 2007.
- 86. "Appreciation." Merriam-Webster.com [Internet] Merriam Webster. c2015. [Cited 9 Mar 2016]. Available at: http://www.merriam-webster.com/dictionary/appreciation.
- Bandura A. Self-efficacy. In: Ramachaudran VS, editor. Encyclopedia of human behavior. New York: Academic Press. (Reprinted in Friedman H, editor, Encyclopedia of mental health. San Diego: Academic Press; 1998); 1994. p. 71–81.
- Toback RL, Graham-Bermann SA, Patel PD. Outcomes of character strengths-based intervention on self-esteem and self-efficacy of psychiatrically hospitalized youths. Psychiatr Serv. 2016;67(5):574–7.
- Bradshaw BG, Richardson GE, Kulkarni K. Thriving with diabetes: an introduction to the resiliency approach for diabetes educators. Diabetes Educ. 2007;33(4):643–9.
- 90. Bradshaw BG, Richardson GE, Kumpfer K, Carlson J, Stanchfield J, Overall J. Determining the efficacy of a resiliency training approach in adults with type 2 diabetes. Diabetes Educ. 2007;33(4):650–9.
- Fergus S, Zimmerman MA. Adolescent resilience: a framework for understanding healthy development in the face of risk. Annu Rev Public Health. 2005;26:399–419.

- 92. VIA Character [Internet]. Values in action inventory of strengths. c2014. [Cited 9 Mar 2016]. Available from: https://www.viacharacter.org/survey/account/register.
- 93. Gander F, Proyer RT, Ruch W, Wyss T. The good character at work: an initial study on the contribution of character strengths in identifying healthy and unhealthy work-related behavior and experience patterns. Int Arch Occup Environ Health. 2012;85(8):895–904.
- Cederbaum J, Klusaritz HA. Clinical instruction: using the strengths-based approach with nursing students. J Nurs Educ. 2009;48(8):422–8.
- Knauper B, McCollam A, Rosen-Brown A, Lacaille J, Kelso E, et al. Fruitful plans: adding targeted mental imagery to implementation intentions increases fruit consumption. Psychol Health. 2011;26(5):601–17.
- 96. Paiano AM. Rose of compassion: a theological, depth psychological, and clinical consideration of the relation between personal suffering and the suffering of others. Doctoral Dissertation, Union Theological Seminary: New York; 1999.
- American Medical Association [Internet]. Chicago: The Association. c1967 [Updated June 2001]. Principles of medical ethics. Available from: http://www.ama-assn.org/ama/pub/physician-resources/medical-ethics/codemedical-ethics/principles-medical-ethics.page.
- Olson K, Kemper KJ. Factors associated with well-being and confidence in providing compassionate care. J Evid Based Complementary Altern Med. 2014;19(4):292–6.
- Hojat M, Louis DZ, Markham FW, Wender R, Rabinowitz C, Gonnella JS. Physicians' empathy and clinical outcomes for diabetic patients. Acad Med. 2011;86(3):359–64.
- Parrish 2nd RC, Menendez ME, Mudgal CS, Jupiter JB, Chen NC, Ring D. Patient satisfaction and its relation to perceived visit duration with a hand surgeon. J Hand Surg Am. 2016;41(2):257–62.
- 101. Foster A, Chaudhary N, Kim T, Waller JL, Wong J, Borish M, et al. Using virtual patients to teach empathy: a randomized controlled study to enhance Medical Students' Empathic Communication. Simul Healthc. 2016;11(3):181–9.
- 102. Walton D. Introducing emotional intelligence: a practical guide. London: Icon Books Ltd; 2013.
- 103. "Honesty". Merriam-Webster.com. [Internet] Merriam Webster. c2015. [Cited 18 Feb 2016] Available from: http://www.merriam-webster.com/dictionary/honesty/.
- 104. "Honest". Merriam-Webster.com. [Internet] Merriam Webster. c2015. [Cited 18 Feb 2016] Available from: http:// www.merriam-webster.com/dictionary/honest/.
- 105. Holland AE, Fiore Jr JF, Goh N, Symons K, Dowman L, Westall G, et al. Be honest and help me prepare for the future: what people with interstitial lung disease want from education in pulmonary rehabilitation. Chron Respir Dis. 2015;12(2):93–101.
- 106. Kraman SS, Hamm G. Risk management: extreme honesty may be the best policy. Ann Intern Med. 1999;131(12):963–7; Cohen ML. The power of apology. Fam Pract Manag. 2010;17(1):40.
- 107. Wu AW. Handling hospital errors: is disclosure the best defense? Ann Intern Med. 1999;131(12):970-2.
- 108. Boothman RC, Imhoff SH, Campbell Jr DA. Nurturing a culture of patient safety and achieving lower malpractice risk through disclosure: lessons learned and future directions. Front Health Serv Manag. 2012;28(3):13–28.
- Vincent C, Young M, Phillips A. Why do people sue doctors? A study of patients and relatives taking legal action. Lancet. 1994;343(8913):1609–13.
- 110. MacInnes D, Courtney H, Flanagan T, Bressington D, Beer D. A cross sectional survey examining the association between therapeutic relationships and service user satisfaction in forensic mental health settings. BMC Res Notes. 2014;7:657.
- 111. Rubak S, Sandbæk A, Lauritzen T, Christensen B. Motivational interviewing: a systematic review and metaanalysis. Br J Gen Pract. 2005;55(513):305–12.
- 112. Armstrong MJ, Mottershead TA, Ronksley PE, Sigal RJ, Campbell TS, Hemmelgarn BR. Motivational interviewing to improve weight loss in overweight and/or obese patients: a systematic review and meta-analysis of randomized controlled trials. Obes Rev. 2011;12:709–23.
- 113. Levensky ER, Forcehimes A, O'Donohue WT, Beitz K. Motivational interviewing: an evidence-based approach to counseling helps patients follow treatment recommendations. Am J Nurs. 2007;107(10):50–8.
- 114. Rollnick M. Motivational interviewing: helping people change. 3rd ed. New York: The Guilford Press; 2012. p. 12–29.
- 115. Fredrickson BL. The role of positive emotions in positive psychology: the broaden-and-build theory of positive emotions. Am Psychol. 2001;56:218–26.
- 116. Leong FTL, Altmaier EM, Johnson BD, editors. Encyclopedia of counseling. Vol. 2. Counseling theories and therapies; New York: SAGE Publications; 2008. p. 541–2.
- 117. Verstuyf J, Patrick H, Vansteenkiste M, Teixeira PJ. Motivational dynamics of eating regulation: a selfdetermination theory perspective. Int J Behav Nutr Phys Act. 2012;9:21.
- Patrick H, Williams GC. Self-determination theory: its application to health behavior and complementarity with motivational interviewing. Int J Behav Nutr Phys Act. 2012;9:18.

- Teixeira PJ, Silva MN, Mata J, Palmeira AL, Markland D. Motivation, self-determination, and long-term weight control. Int J Behav Nutr Phys Act. 2012;9:22.
- Cooperrider DL, Whitney D. Appreciative inquiry: a positive revolution in change. San Francisco: Berrett-Koehler Publishers, Inc.; 2005.
- 121. Ludema JD, Cooperrider DL, Barrett FJ. Appreciative inquiry: the power of the unconditional positive question. In: Reason P, Bradbury H, editors. Handbook of action research. Thousand Oaks: Sage; 2001. p. 189–99.
- McCarthy B. Appreciative inquiry: an alternative to behaviour management. Dementia (London). 2016. pii: 1471301216634921; [Epub ahead of print].
- 123. Trajkovski S, Schmied V, Vickers M, Jackson D. Implementing the 4D cycle of appreciative inquiry in health care: a methodological review. J Adv Nurs. 2013;69(6):1224–34.
- 124. Johnson SS, Paiva AL, Cummins CO, Johnson JL, Dyment SJ, Wright JA. Transtheoretical model-based multiple behavior intervention for weight management: effectiveness on a population basis. Prev Med. 2008;46:238–46.
- Prochaska JO, Velicer WF, Prochaska JM, Johnson JL. Size, consistency, and stability of stage effects for smoking cessation. Addict Behav. 2004;29:207–13.
- 126. Prochaska JO, Norcross JC, Diclemente CC. Changing for good: a revolutionary six-stage program for overcoming bad habits and moving your life positively forward. New York: Harper Collins Publishers; 1994.
- 127. Pro-Change. [Internet]. Behavior Systems, Inc. 2015. [Cited 18 Feb 2016]. Available from: http://www.prochange. com/transtheoretical-model-of-behavior-change/.
- 128. Reproduced from www.prochange.com with permission from Pro-Change Behavior Systems, Inc.
- 129. Frates EP, Crane ME. Lifestyle medicine consulting walking meetings for sustained weight loss. BMJ Case Rep. 2016;2016. Pii:bcr2015213218.
- Allender S, Hutchinson L, Foster C. Life-change events and participation in physical activity: a systematic review. Health Promot Int. 2008;23(2):160–72.
- 131. Whitmore J. Coaching for performance: GROWing human potential and purpose the principles and practice of caching and leadership. 4th ed. London: Nicholas Brealey Publishing; 2009.
- Alexander G. Behavioural coaching—the GROW model. In: Passmore J, editor. Excellence in coaching: the industry guide. 2nd ed. Philadelphia: Kogan; 2006. p. 83–93.
- Locke EA, Latham GP. Building a practically useful theory of goal setting and task motivation: a 35-year odyssey. Am Psychol. 2002;57(9):705–17.
- 134. Csikszentmihalyi M, Rathunde K. The measurement of flow in everyday life: toward a theory of emergent motivation. Nebr Symp Motiv. 1992;40:57–97.
- 135. Knowles MS. The adult learner. A neglected species. 4th ed. Houston: Gulf; 1990.
- 136. Parretti H, Aveyard P, Blannin A, Clifford SJ, Coleman SJ, Roalfe A, et al. Efficacy of water preloading before main meals as a strategy for weight loss in primary care patients with obesity: RCT. Obesisity. 2015;23(9): 1785–91.
- 137. Zhang KM, Dindoff K, Arnold JM, Lane J, Swartzman LC. What matters to patients with heart failure? The influence of non-health-related goals on patient adherence to self-care management. Patient Educ Couns. 2015;98(8):927–34.
- 138. Dennis SM, Harris M, Lloyd J, Powell Davies G, Farugi N, Zwar N. Do people with existing chronic conditions benefit from telephone coaching? A rapid review. Aust Health Rev. 2013;37(3):381–8.
- 139. Thom DH, Hessler D, Willard-Grace R, DeVore D, Prado C, Bodenheimer T, Chen EH. Health coaching by medical assistants improves patients' chronic care experience. Am J Manag Care. 2015;21(10):685–91.
- 140. Willard-Grace R, DeVore D, Chen EH, Hessler D, Bodenheimer T, Thom DH. The effectiveness of medical assistant health coaching for low-income patients with uncontrolled diabetes, hypertension, and hyperlipidemia: protocol for a randomized controlled trial and baseline characteristics of the study population. BMC Fam Pract. 2013;14:27.
- 141. Morris J, Wang W, Wang L, Peddecord KM, Sawyer MH. Comparison of reminder methods in selected adolescents with records in an immunization registry. J Adolesc Health. 2015;56(5 Suppl):S27–32.
- 142. U.S. Department of Agriculture. ChooseMyPlate.gov [Internet] Washington, DC. [Cited 9 Mar 2016]. Available from: http://www.choosemyplate.gov/.
- 143. Akers JD, Cornett RA, Savla JS, Davy KP, Davy BM. Daily self-monitoring of body weight, step count, fruit/ vegetable intake, and water consumption: a feasible and effective long-term weight loss maintenance approach. J Acad Nutr Diet. 2012;112(5):685–92.
- 144. Kong A, Beresford SA, Alfano CM, Foster-Schubert KE, Neuhouser ML, Johnson DB, et al. Self-monitoring and eating related behaviors are associated with 12-month weight loss in postmenopausal overweight-to-obese women. J Acad Nutr dIET. 2012;112(9):1428–35.
- 145. Seale SA, Sherard T. The full plate diet: slim down, look great, be healthy! Austin: Bard Press; 2009.
- 146. Association American Heart. The New American Heart Association Cookbook. In: Revised and updated with more than 150 all-new recipes. 8th ed. New York: Clarkson Potter Publishers; 2010.

- 147. Guh DP, Zhang W, Bansback N, Amarsi Z, Birmingham CL, Anis AH. The incidence of co-morbidities related to obesity and overweight: a systematic review and meta-analysis. BMC Public Health. 2009;9:88.
- 148. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med. 2004;1(3):e62.
- 149. Fiechtner L, Kleinman K, Melly SH, Sharifi M, Marshall R, Block J. Effects of proximity to supermarkets on a randomized trial studying interventions for obesity. Am J Public Health. 2016;106(3):557–62.
- 150. Marino J, Pegg J, Frates EP. The connection prescription: using the power of social interactions and the deep desire for connectedness to empower health and wellness. Am J Lifestyle Med. 2015;109:186–204.
- 151. Frank E, Bryan J, Elon L. Physician disclosure of healthy personal behaviors improves credibility and ability to motivate. Arch of Fam Med. 2000;9:287–90.
- 152. Abramson S, Stein J, Schaufele M, Frates E, Rogan S. Personal exercise habits and counseling practices of primary care physicians: a national survey. Clin J of Sports Med. 2000;10(1):40–8.
- Frank E, Carrera JS, Elon L, Hertzberg VS. Predictors of US medical students' prevention counseling practices. Prev Med. 2007;44(1):76–81.
- 154. Gallup Well-Being. [Internet] U.S. physicians set good health example. c2012. [Cited 21 Feb 2016]. Available from: http://www.gallup.com/poll/157859/physicians-set-good-health-example.aspx.
- 155. Brooks R, Goldstein S. Raising resilient children: fostering strength, hope, and optimism in your child. New York: McGraw-Hill; 2001.
- 156. Brooks R, Goldstein S. The power of resilience: achieving balance, confidence, and personal strength in your life. New York: McGraw-Hill; 2004.
- 157. Loehr J, Schwartz T. The power of full engagement: managing energy, not time, is the key to high performance and personal renewal. New York: Free Press; 2003.

Chapter 4 Effective Strategies to Help Adults Manage How Much They Eat

Mary Abbott Waite and James M. Rippe

Key Points

- Controlling energy intake to achieve energy balance, control weight, reduce risk of chronic disease, and promote health is of concern for American adults. In this population, over two-thirds are overweight or obese. In addition, approximately two-thirds of American homes house only adults.
- Individual eating behavior is governed by an interrelated complex of neurophysiological factors, psychological and cognitive factors, psychosocial factors, and external and environmental cues and influences.
- The human body has a number of biological mechanisms, such as the brain-gut neuroendocrine regulators, which ideally function to regulate hunger and satiety and keep the body in a state of energy balance, or homeostasis. Many other factors and behaviors, however, may modify these mechanisms and lead to eating in the absence of hunger.
- Genetic factors such as sensory perceptions may influence eating behavior and these factors are malleable not fixed.
- Eating palatable food triggers the brain's reward system, prompting eating in the absence of energy need. Food cues in the environment can increase endocrine signaling and the desire to eat palatable foods in the absence of hunger. This effect tends to increase with continued behavior.
- Psychological factors such as stress, mood, and emotional status may prompt greater intake of palatable, energy dense foods in some individuals but not all. Negative emotions may have a greater impact on overeating than positive emotions.
- Ineffective self-control, as exhibited in disinhibition and impulsivity, may lead to greater energy intake.
- Eating is also a social activity. Food habits and social norms, including the desire for acceptance, have an impact on food choices and food intakes.
- Food cues, such as visual appearance, smell, and taste play a major role in food choices. The variety of food available and its proximity to those eating also play a role in food intakes. The availability of larger portion sizes tends to result in greater consumption.
- Food marketing messages, which constitute external food cues, influence individual food purchases.

M.A. Waite, PhD (🖂) • J.M. Rippe, MD

Note: An earlier version of material in this chapter was prepared for the ConAgraFoodsScienceInstitute.com.

Rippe Lifestyle Institute, 21 North Quinsigamond Avenue, Shrewsbury, MA 01545, USA e-mail: mawaite100@aol.com; bgrady@rippelifestyle.com

- Strategies for positively managing food choices and energy intake must take into account the complex factors that influence individual behavior. Strategies must be individualized. One approach does not work for all.
- Creating a home food environment that supports healthful eating may result in more nutrient-dense and fewer energy-dense food choices. Home food inventories and screening tools can help individuals create such home food environments. Counseling and/or support by nutrition professionals in this process may result in better outcomes.
- Education about portion size and using portion control strategies can promote better food choices and balanced energy intake. Using nutritious, controlled-portion convenience foods as part of an overall eating plan has been shown to improve nutrient quality, help manage weight, and meet the need of many individuals and families for time-saving convenience.
- There is evidence that increasing cooking skills helps many individuals make better food choices for themselves and for their families.
- Mindful and attentive eating strategies address cognitive and self-awareness processes to help individuals exert control over how much and what foods they eat.
- Nutritional interventions based on Cognitive Behavioral Theory appear most effective in fostering changes in eating behavior by fostering individual self-regulation and self-efficacy.
- Learning to use resources in the external environment such as food and menu labels and costeffective promotions of fruits and vegetables can promote better food choices and energy intakes.

Keywords Eating behavior • Appetite and satiety regulation • Brain reward system • Dietary habits • Food decision-making • Food cues • Stress and emotional eating • Social context of eating • Nutrition and dietary assessment • Nutritional counseling and education • Cognitive behavioral therapy • Environmental interventions • Portion control • Mindful eating • Self-efficacy

Introduction

To live, the human body must consume nutrients and energy. As a consequence, the brain has a dedicated system to signal appetite and hunger to let the body know it is time to eat and to signal satiety to indicate adequate consumption has occurred. But other systems and many interrelated factors influence this basic function and overall eating behaviors. These include:

- Neurophysiological factors
- Psychological and cognitive factors
- · Psychosocial factors
- · External and environmental cues and influences

This multitude of underlying factors, therefore, plays a complex, interconnected role in individual eating behaviors. Strategies that enable an individual to eat a diet that has the right balance of nutrients and energy must take into account these complex influences on seemingly simple individual food choices and behaviors.

This chapter focuses on eating behaviors in adults for several reasons. A vast amount of research focuses on influences on eating behaviors in children and interventions to improve dietary eating behaviors and food intakes in this population. This focus is no doubt justified because eating patterns and food preferences tend to develop during childhood. In the United States today, however, approximately 69% of adults over age 18 are overweight or obese, a circumstance that increases their health risks [1]. Interestingly, in 2012, the US census indicated that just over two-thirds of US households (67.8%) contained only one or two adults, but no children under age 18 [2]. Although the overlap of these populations is unknown, clearly a large proportion of the American adult population faces

challenges in making changes to eating behaviors and food choices that will help them achieve a diet that is balanced in energy and rich in nutrients.

This chapter first examines the complex of factors that influence eating behaviors and then discusses a number of dietary interventions that research has shown are effective for adults.

Understanding the Multiple Factors that Influence Eating Behaviors

Many factors influence food decision-making and eating behaviors. Extensive research informs the generally accepted view that human biology provides mechanisms to maintain energy balance ("homeostasis") [3, 4]. These mechanisms are based on gene expression and neural and endocrine regulators but are also modified by other factors and stimuli. Some of the modifying factors are internal to the individual and some are external (environmental) [3–9].

Early research on eating biology and behavior focused on mechanisms that control eating in the presence of metabolic need. More recent research, however, has begun to examine the mechanisms and factors that contribute to eating in the absence of metabolic need and, thus, lead to overeating, weight gain, and obesity. Such continuing and emerging research is creating greater understanding of the multiple interactive factors that contribute to eating behaviors that take place in the absence of hunger and contribute to weight gain. Eating in the absence of metabolic need is also termed *hedonic* eating.

Neurophysiological Factors

A number of biological mechanisms in the human brain and body interact to influence metabolism and eating behavior.

- *Genetic Factors.* Certain eating behaviors may start with gene expressions, gene variants, or genetic predispositions. For example, genetic factors may influence such individual sensory perceptions as taste, smell, or visual impact, resulting in experiences of pleasure or dislike that influence food preferences [5, 10]. Although individual genetics may predispose certain eating behaviors, they are not fixed, but are malleable and may be modified by internal and external stimuli [4, 5, 11].
- *Hunger and Satiety Signaling System*. Sensations of hunger and satiety depend on a brain–gut connection that communicates largely through the neuroendocrine system and vagal neurons (vagus nerve). This system, often called the *homeostatic* system, monitors energy levels in blood and fat. Theoretically, when a body is in state of perfect energy balance, or homeostasis, the system signals hunger only in the presence of metabolic need and signals satiety when that metabolic need is satisfied. The hormones playing an interactive role in this signaling include ghrelin, which signals appetite or hunger, and leptin, insulin, and peptides, such as CCK and peptide YY, which signal satiety [5, 6, 12]. The fullness of the stomach (gastric stretching) also sends satiety signals via the vagus nerve [5, 6, 13].

In actuality, however, many factors and behaviors can modify the system and lead to overeating [4]. The brain's reward system, in particular, has a major impact.

• *The Brain's Reward System.* Eating palatable food triggers the brain's reward system, reinforcing the desire to eat such food and prompting eating in the absence of energy need [4, 8, 14–18]. The major neurochemical transmitter that controls this system is dopamine [14] and ghrelin may also play a role [11, 18]. Research indicates that the food reward system exerts a more powerful effect over eating behavior and food regulation than the homeostatic system. This system also adapts to

changing behavior. The more an individual eats palatable or pleasurable foods, the more the brain moves from simply experiencing a reward response when such foods are eaten to anticipating such rewards. Thus, food cues in the environment can stimulate increased dopamine and increased desire to eat before the food is consumed. This may result in increased intake of the food, particularly when eating is not immediate, but delayed for a time [16]. Over time, the food reward system adapts toward greater tolerance – the more one eats, the less sensitive the dopamine response and the more food required to trigger the reward. It is important to note, however, that individual eating response to reward signals varies widely.

It is also worth noting that many different aspects of life, not just food, stimulate the brain's reward system. These can range from hearing enjoyable music or seeing art to the body's response to drugs.

Psychological and Cognitive Factors

Psychological factors, such as stress, mood, and emotional status, and cognitive factors, such as inhibition, impulsivity, decision-making, memory, and distraction, also play a role in eating behavior.

• Impact of Stress

The general public and science have long observed that stress affects individual eating behavior [19, 20]. The stress may be emotional, such as that produced by interpersonal conflict or personal loss, or physical, such as that caused by illness or substance withdrawal (e.g., trying to quit smoking) [19]. Stress may also be either acute or long-term. Changes in eating behavior have been observed for all types of stress. Typically, the response that comes to mind is overeating highly palatable or pleasurable food, such as higher fat or higher sugar "comfort" foods. In fact, studies have observed that while many stressed individuals may select calorie-dense foods and snacks, the actual calorie intake varies. In response to stress, about 40% of individuals eat more calories, about 40% consume fewer calories, and about 20% have no change in caloric consumption [19]. Overeating in response to stress appears to occur more frequently in overweight or obese individuals than in those of normal weight [19].

Emotional and Mood Status

In general, research suggests that negative emotions and moods such as anxiety, anger, or depression may have stronger links to increased energy intake than positive emotions or moods [20]. Eating pleasurable food or "emotional eating" may be an attempt to trigger more positive feelings and mood. Individual long-term responses to chronic stress have been shown to alter brain chemistry over time to support continued overeating in many individuals [20].

Ineffective Self-control over Eating Choices – Role of Disinhibition and Impulsivity

Other factors or "domains," often related to the impact that emotional eating can have on food choice decisions, also undermine intention. Informally, this behavior may be termed "lack of control," but several factors play a role. Disinhibition may be a primary motivation [21]. Bellisle defines disinhibition as "a tendency to lose control over one's eating behavior and ingest excessively large quantities of food substances in response to a variety or cues and circumstances" [22, 23]. These cues may be interior or exterior to the individual. Impulsivity, for instance, may be a related interior influence [24–26]. Although multifaceted, impulsivity may be defined as having sudden, unplanned reactions to internal or external events without thinking about the consequences [27]. Emotional status or mood, such as low self-esteem, stress, and depression [21], may be additional interior factors, while distraction or inattention may be caused by exterior or interior circumstances. Distraction may be attributed to cognitive conditions such as attention-deficit hyperactivity disorder (ADHD) or exterior stimuli

such as eating and socializing with peers, being unaware of food cues, or doing other activities that distract from eating (watching TV, using electronic devices, and the like).

Some research suggests that chronic overeating and disinhibition actually "rewire" the neural reward circuits of the brain to heighten the response to food cues and erode control [28, 29]. Such neural changes, therefore, may contribute to the observed effect that obese individuals, particular restrained (dieting) individuals, tend to exhibit greater difficulty with controlling palatable food intakes than normal weight individuals [25, 30].

Psychosocial Factors

Eating is a behavior that individuals perform daily often in the same places with the same people. In this context, psychosocial factors such as habit and social norms play a role in food choices and eating behaviors. They also affect the possibility of an individual changing food choice behavior by intention or decision.

Habit

Habit may be defined as "learned sequences of acts that, as a result of frequent performances in similar situations, are triggered automatically by specific environmental cues" [31]. Research indicates that food habits typically override intention. When certain eating patterns or choices are habitual, individuals seek little information to make considered choices. The stronger the habit, the less likely an individual is to consider information that might prompt choices of other options particularly in the presence of situational or environmental cues [31]. For instance, if an individual usually break-fasts on a sausage biscuit, he or she is unlikely to seek information about other, potentially more varied choices, particularly when the environment gives reinforcing cues, such as smell of sausage cooking. (See also the section on Environmental Cues.)

Social Norms and Social Modeling

What other people eat has a significant impact on individual food intakes and food choices, according to a robust body of research [32–36]. Typically, individuals follow the eating norms of groups to which they belong, including family, peers, friendship groups, and nationality or culture [32]. Social norms may be defined as "implicit codes of conduct that provide a guide to appropriate conduct" [32]. Research indicates that individuals tend to adhere to norms exhibited by perceived "in groups" rather than "out groups" [35]. However, just knowing what others eat (informative norms) also appears to influence consumption when individuals are alone or not with the model group [34].

Various factors contribute to the impact of social norms on eating behavior and food choices. These include a desire for social acceptance, a goal of social facilitation (helping everyone get along), and creating or managing a desired impression [33, 37].

Social modeling correlates with increased or decreased food intake whether individuals know others in the group or are strangers to each other. Some research indicates that a stronger effect may occur when individuals are eating with a group of strangers and/or may be uncertain of appropriate eating behaviors for the specific situation or group [34].

Taken as a whole, the research shows that social norms and modeling have a significant effect on all eaters, regardless of weight status, dieting status, or gender; however, there are limitations. Reviewers note that although studies represent a wide diversity of populations, the largest group of studies, particularly trials, tends to be in women and/or young adults (i.e., college and university students). Convenient access to these populations may be part of these phenomena. Another noted limitation in trials is that food intake has been tested most often with high-energy snack foods such as small cookies rather than nutrient-dense foods such as fruits and vegetables. Therefore, additional research

is needed to confirm or modify the implications of these findings for different subgroups of the general population and a wider range of food groups with better nutrient profiles [34].

Environmental Food Cues and Influences

Reactions to food cues and environment represent primary influences on food choices and intakes. Some cues are sensory (such as visual images and odors/smells), and others are contextual and environmental (such as ambience, portion size, dietary variety, advertising and marketing messages, and food availability) [38–40]. Individual reactivity to food cues is complex because the cues are influenced by interactions of food perception, previous food experiences, motivation to eat, state of hunger or satiety, and individual neurophysiological, genetic, and psychological characteristics [41, 42].

Sensory Food Cues

Visual appearance, smell (odor), and taste are probably the primary sensory food cues that influence food choices and intakes [38]. Like other factors in eating behaviors, these sensory cues are also interrelated.

Seeing food is often the first sensory contact before an eating occasion. Visual cues include the color of the food, its visibility, presentation (arrangement), volume, and variety [38]. These cues can raise expectations about taste, flavor, and palatability and, if positive, trigger the desire to eat. Such cues and others can activate the brain's reward system [42, 43]. Sight together with smell also stimulates the anticipatory release of saliva and other digestive substances and the increase of blood insulin [38].

Research has shown that greater variety of foods available at an eating occasion leads to increased consumption [38, 44–47]. In one review of short-term trials, variety increased consumption by 22–25 % [45]. The attractive color and appearance of the food in its variety also appear to be contributing factors [38].

Proximity of the food to those eating a meal or snack may also play a role in how much is consumed. In trials where the food was positioned nearer the subjects and was more visible, participants consumed more than controls [38, 48, 49].

The odor of foods and how individuals perceive those odors play a major role in individual perception of a food's flavor or taste [50]. Preferences for particular odors, however, are not innate but are flexible, learned behaviors [50]. Smelling an odor that an individual associates with food and identifies as pleasant can trigger salivation and production of gastric acids and insulin. This reaction is similar to that evoked by visual cues. Research also suggests that olfactory sensitivity changes or adapts in different conditions. Some but not all research suggests that individuals may be less sensitive to the odor of a food they like when they are satiated rather than when they are hungry [51]. Research has also suggested that individuals with eating disorders have impaired sensitivity to odors [52]. Dieting individuals may also respond differently to odors than those not dieting [52, 53].

Portion Sizes

Substantial research indicates that larger portion sizes often result in individuals eating more food and consuming more energy [38, 54, 55]. Larger portions may have more visual appeal [38, 56]. Served with larger portions, people may also tend to eat larger bites and to eat more quickly [37, 38, 57, 58]. Bigger bites and rapid consumption may result in greater energy consumption before satiety kicks in [58].

What is served as a portion (in various settings) may also set an expectation that the portion is appropriately sized [54]. Similarly, the portion size selected by others in a group may set the perception of appropriate portion size for an individual within the group [54]. Packaging can also set up

expectation of appropriate portion size. This may occur with larger packages, which may encourage larger portions, or with single-serve packaging, such as 100-cal packs of snack foods, intended to encourage portion control [55].

Marketing Messages About Foods

Marketing communicates messages about foods through many media, including:

- Advertising (television, radio, print, Internet, social media, movie placement)
- Promotions (coupons, in store, games, contests)
- Branding (including sponsorship of sports and community events)
- Packaging (design, claims, including health claims)
- Placement (in store displays, shelf position)
- Pricing

Research, particularly in the field of marketing, has established that all these aspects of marketing communication influence what foods people buy [59, 60]. Thus, food marketing communications constitute external food cues that may influence eating behaviors [60].

Strategies for Managing How Much You Eat

The primary goal of any strategy for managing food choice and energy intake is to achieve a sustained positive impact on an individual's food choice behavior and to improve his or her knowledge of how to make those positive choices and of how to recognize and reject potentially negative influences.

The potential strategies for managing how much one eats are as complex and interrelated as the factors that influence food choice and intake behaviors. The multiple goals for managing how much and what one eats also range from promoting general healthful nutrition to support well-being and prevent chronic disease to achieving specific nutritional goals to support management of specific chronic diseases (i.e., heart disease, type 2 diabetes, hypertension, or cancers) and to manage weight. Reviewing research on such strategies suggests that the most effective interventions address the specific circumstances of an individual or family [61]. One approach does not fit all! The research summarized in the following sections provides insights and tools useful to registered dietitian nutritionists and other healthcare professionals as they work with clients.

Create a Supportive Eating Environment

Given the complexity of influences on food choices, which may often overcome intention, research suggests some modifications that individuals may make to their home environments to support preparing and eating more nutrient-rich foods in healthful amounts.

Stock the Pantry, Refrigerator, and Freezer with Nutrient-Dense Foods

• Determining what foods an individual or family usually has on hand is a good first step to change [62]. A Home Food Inventory (HFI) checklist that a client can complete provides a snapshot of what types of foods a client typically keeps on hand and enables discussion of which foods are nutrient-rich and which are not and how the client might gradually make

changes to improve dietary quality. The HFI may also be used at intervals to informally check progress. The HFI also provides a good focal point for other practical nutrition education such as using labels to judge nutrient content and portion size. Home Food Inventories have been effectively used in a number of research interventions [63–65]. A validated, self-administered Home Food Inventory [66], created by Jayne A. Fulkerson and team and frequently used in studies, may be downloaded at http://appliedresearch.cancer.gov/mfe/instruments/fulkerson_home_food_inventory.pdf;

Various screeners may help health professionals evaluate what a client likes and what he/she eats, particularly when used in conjunction with a Home Food Inventory. A variety of screeners are available through the following web site, which is maintained by the Network for a Healthy California, California Department of Public Health: https://food-hub.org/files/resources/ Network-Compendium.pdf

- *Having adequate food preparation supplies* may also contribute to more frequent and convenient preparation of home meals that are usually more nutritious [67]. However, only very limited research focuses primarily on this aspect of the home food environment [67].
- Shopping with a grocery list has been shown to help individuals select nutrient-dense foods, limit impulse purchases of energy-dense foods, and contribute to staying on budget and meal planning [68, 69]. Other studies suggest that if shoppers are reminded about their goals of weight management during shopping, their choices of higher-nutrient and lower-fat foods increase significantly [70, 71]. This research suggests that putting such a reminder on one's shopping list might be helpful.
- Stocking up on sales of nutritious foods may help save money but may also present pitfalls [72]. Some studies show that having larger quantities of food on hand may lead to greater consumption. Discussing with clients how to handle these temptations may be a useful part of nutrition education efforts.

Include Nutritious Convenience Foods that Feature Controlled Portions

Many studies have shown that individuals who use nutrient-dense, portion-controlled meal replacements (including frozen meals or entrees and liquid meal replacements) and portion-controlled snacks have greater success over time in managing weight and maintaining weight loss [55, 73–76]. Using portion-controlled meal replacements has also been shown to improve dietary quality [77–80]. This strategy of using portion-controlled meal and snack replacements may be particularly convenient for single working adults or working adult couples.

Improve Cooking Skills as Needed

Lack of cooking skills or knowledge of how to cook certain nutritious foods has been identified as a barrier to eating more nutritious foods. As a result, cooking classes have grown in popularity as an intervention to help individuals prepare more nutritious meals at home and eat out less frequently, particularly at quick-serve restaurants [81]. Although a recent review found that many studies of such interventions are not very rigorous [81], there is evidence that for many individuals taking one or more cooking classes to learn new cooking techniques or to explore cooking unfamiliar foods may be help-ful [81–83].

Practice Portion Control Strategies

Controlling portion sizes is a major challenge for individuals in controlling how much they eat. Yet estimation of appropriate portion sizes appears to vary from individual to individual [55, 82, 85–87]. Most individuals, however, tend to underestimate how much they are eating [88]. As a result, counselors may wish to draw from a variety of portion control strategies to find a combination that works for an individual client or family.

Educating about portion size and "portion distortion." A number of portion-size measurement aids (PSMAs) have been available for many years [89, 90]. Many popular PSMAs use photographs, such as the interactive Portion Distortion Quizzes from the National Heart Lung and Blood Institute (NHLBI). http://www.nhlbi.nih.gov/health/educational/wecan/eat-right/portion-distortion.htm Such aids may be a useful place to start but questions remain about their accuracy and what lasting impact they may have on behavior [89, 91, 92]. As of early 2016, the most recent review assessing the validity of PSMAs specifically was published in 1997 [89].

Learning about appropriate portion sizes has been an observed result in studies that used replacement meals either to teach portion size [93] or to assist in improvement of nutrient intakes and weight management [77, 84].

- *Making nutrient-rich, low-energy-density foods the main focus of meals.* A number of studies suggest reducing energy intake without reducing the actual weight or volume of food eaten by shifting the proportion of foods eaten at meals toward larger servings of vegetables and fruits and smaller servings of energy-dense foods (such as higher fat foods, foods made with refined grains, and foods with added sugars) [55]. Several strategies appear effective in multiple studies.
 - Serving a nutrient-rich, low energy density salad or soup before the main entrée [55, 94]
 - Increasing the amount of fruits and vegetables in a meal, lowering the amount of fat, or increasing the amount of water [55, 95, 96]
- Using portion-controlled meal replacements and snack packs. A growing body of research indicates that regularly using portion-controlled prepared entrees and snacks can be a useful strategy to help control energy intake [55].
- Using smaller dishware or divided dishware. This strategy for eating smaller portions is frequently recommended. Recent reviews of the research literature, however, have found that the impact is more mixed than the results of many individual studies might suggest [97, 98]. The effect was found to be greatest for bowls rather than plates and when subjects were eating in distracted conditions. Before dismissing the potential usefulness of smaller dishware, it is important to note several obvious limitations of the studies reviewed. First, in a majority of the studies, subjects served themselves and were able to return for additional servings ("ad libitum"). This practice could influence results [97]. Reviewers also suggest that in a laboratory setting participants may pay more attention to food selection than when eating in "real life" settings [48, 98, 99]. Informal observation notes that many recommendations to use smaller plates and bowls also advise serving meals away from the table and storing leftovers before eating. This latter strategy addresses the problem of proximity (in this case readily available extra servings) [48] which may counter the potential small plate or bowl effect.

In at least three studies, using divided plates designed to help control portions resulted in decreased energy intake and better nutrient intakes [100–102].

Adopt Mindful Eating and Attentive Eating Techniques

Strategies such as mindful eating and attentive eating address cognitive processes to help individuals exert control over how much and what foods they eat. Overall, the research literature suggests that techniques used by each of these methods may help individuals (particularly overweight/obese, emotional, and binge eaters) control their dietary intakes [103–107]. Many successful interventions also included Cognitive Behavioral Therapy or other counseling therapies discussed in the next section [103].

Mindful eating appears to have the broadest focus and its targets and techniques overlap attentive eating strategies. Intuitive eating strategies also appear to be similar to mindful eating [108]. Mindful eating may be defined as nonjudgmentally paying attention to the food one is eating, to how the body is experiencing the food and eating process, and to physical and emotional sensations associated with the eating experience [103, 109]. Attentive eating focuses on avoiding distractions while eating and responding to internal cues such as satiety and emotions (e.g., boredom, anxiety) and to external food cues (particularly inappropriate food cues) [110, 111]. Attentive eating also focuses on enhancing food memories of earlier eating occasions during the day [110, 111].

Sample techniques for mindful and attentive eating include:

- Eating without distractions, such as watching TV, reading email or a book, or using a computer
- · Taking time to savor food and to eat slowly
- Triggering positive food choices by asking mental questions about reasons for eating (hungry or some other reason) and external foods cues to which one may be reacting
- Using mindfulness meditation to reduce stress eating [112, 113]

The following validated questionnaires used in conjunction with counseling may help clients explore factors related to mindless/distracted eating or emotional eating.

- Mindful Eating Questionnaire [109]
- Emotional Eater Questionnaire [23]
- Perceived Stress Scale [114]

Use Counseling and Education Strategies to Support Actions to Achieve Nutrition Goals

Theory-based counseling and education have long been major tools for nutrition professionals [115]. Extensive and diverse research shows that a variety of strategies and models based on Cognitive Behavioral Theory (strategies collectively termed Cognitive Behavioral Therapy [CBT]) have been the most successful in helping individuals make positive changes in eating behaviors, particularly in the short term [116–119]. Research into ways to extend the positive effects of interventions long-term has led to the expansion of Cognitive Behavioral Theory to include interventions that incorporate acceptance and commitment therapy (ACT) [119, 120]. ACT strategies focus on helping individuals use core values and acceptance skills to cope with today's tempting, pleasure-pushing (hedonistic) food environment [119, 120].

Nutrition interventions based on CBT appear most effective in fostering changes in eating behaviors because they are problem-focused and goal-directed with the end objective of fostering individual self-regulation and self-efficacy [117, 118, 121–123]. Change models and techniques typically use variants of five typical CBT strategies [116]:

• Goal-setting and self-monitoring. Evaluation and goal-setting typically take place in face-to-face counseling sessions (individual or group) but there are increasing experiments with online or

mobile app intervention programs [124, 125]. Goal-setting using acceptance theory identifies core values that anchor the individual's commitment to action goals [119, 120]. Motivational interviewing is one technique that has been shown to be effective in working with clients on goal-setting and self-help [118].

The effectiveness of self-monitoring food and activity behavior is well-supported [116–118, 126–128]. Monitoring techniques include daily diaries or trackers [paper, digital, mobile device, online] for behavior such as food intake, physical activity, and regularly weighing. Monitoring could also include techniques for mindfulness or stress management.

- *Stimulus control*. These strategies help individuals identify and modify external cues associated with overeating or with nutritionally unbalanced food intake [116, 117]. Coping techniques range from avoidance (not having tempting food in the house) and distraction (thinking about something else or substituting another activity) to, more recently, techniques to promote learning how to accept food temptations or cravings and move through them [119, 120].
- Cognitive restructuring and acceptance. Thoughts and beliefs about food and about oneself in
 relationship to food may be among the greatest challenges in adopting healthful eating behaviors.
 Various techniques can help individuals become more aware of their beliefs about food, overeating,
 weight management, and body image. Fostering realistic expectations, positive self-talk, and
 acceptance of the challenges of making changes are goals for techniques in this domain [116, 119].
- Stress management. Stress is an acknowledged predictor of relapse for individuals who are trying
 to change eating behaviors or lose weight/maintain weight loss [116, 117]. Stress management
 techniques such as relaxation exercises and mindfulness exercises such as brief timeouts or mindfulness meditation may help many individuals succeed in reaching or maintaining nutrition and
 weight management goals [113].
- Social support. Recent research suggests that an important component to long-term success to
 improve nutrition and weight management may be in continued support from nutrition professionals and healthcare professions [63–65, 129–131]. Various studies suggest that such support
 might come from follow-up phone contact, online support programs, and the like. Support from
 family, friends, and counselors is a long-recognized requirement for success in active interventions [116, 117]

Make Use of Resources in the External Environment that Encourage Healthful Food Choices

How the food environment affects consumers' food choices and eating behaviors is a very complex issue [132, 133]. Research to date may offer some insights into how individuals may take advantage of opportunities for healthy food choices that may be currently available in local stores and restaurants. They may also take advantage of any interventional programs to make healthy foods more available.

- Use food labels. Food labels provide consumers with information needed to choose packaged foods and prepared foods that are higher in nutrients and lower in saturated fats, added sugars, and refined grains. National survey data indicate that label users tend to report better nutrient intakes than nonlabel users [134]. Discussion and research continues about ways to improve the usefulness of both the Nutrition Facts Panel and front-of-package labels [135, 136].
- Take advantage of store promotions of fruits, vegetables, and other nutrient-dense foods. Although a large body of literature describes the potential impact of poor food environments (food deserts) on residents' food intakes, the results of interventions to improve healthy food choices in local food environments has been mixed. Successful programs to increase healthy food access in small

stores around the country required buy-in from both store management and employees and consumers [137–141]. Point-of-purchase promotions alone were not as effective as promotions with more elements. These findings are consistent with studies of consumers that indicate that food shoppers, even those in neighborhoods with limited food outlets, chose stores based on more than proximity to home or work. Consumers often made the effort to go to more distant stores that provided better availability and quality of fruits, vegetables and meats, cleaner and more helpful store environments, and lower prices [132, 142, 143].

Such findings, though not conclusive, suggest that there are opportunities to help consumers look for promotions of healthful foods that can support nutrition goals and to assess the value or actual affordability of healthful foods such as fruits and vegetables [144, 145].

• Use menu labels for restaurant or prepared meals. Several research studies conclude that calorie counts for foods on the menus of fast-casual and full-serve restaurants appear to have had little effect on consumer food choices [146–148]. However, some consumers also report that calorie counts do enable them to make less energy-dense food choices [149, 150]. Because such calorie counts are required by law, they represent a potential tool that individuals may use to help improve food choices [151].

Conclusion

A multitude of factors play a complex, interconnected role in individual eating behaviors. These include genetic and neurophysiological factors, psychological and cognitive factors, psychosocial factors and external, environmental factors. Possibly as a result of this complexity, multicomponent interventions have been among the most successful in helping individuals improve the nutrient intakes and energy balance of their daily diet. These helpful components include helping individuals create a supportive eating environment in their homes, improve portion control, adopt mindful eating and stress relief techniques, learn and use cognitive behavioral techniques and skills, and learn how to make healthful food choices working within the external environments they encounter.

References

- Centers for Disease Control and Prevention. Obesity and Overweight. Fast Facts Webpage. http://www.cdc.gov/ nchs/fastats/obesity-overweight.htm. Accessed 11 Sept 2015.
- Vespa J, Lewis JM, Kreider RM. America's Families and Living Arrangements 2012. U.S. Census Bureau, August 2013. https://www.census.gov/prod/2013pubs/p20-570.pdf.
- 3. Shin AC, Zheng H, Berthoud HR. An expanded view of energy homeostasis: neural integration of metabolic, cognitive, and emotional drives to eat. Physiol Behav. 2009;97:572–80.
- 4. Kanoski SE. Cognitive and neuronal systems underlying obesity. Physiol Behav. 2012;106:337-44.
- 5. Grimm ER, Steinle NI. Genetics of eating behavior: established and emerging concepts. Nutr Rev. 2011;69:52–60.
- Ahima RS, Antwi DA. Brain regulation of appetite and satiety. Endocrinol Metab Clin North Am. 2008;37:811–23.
- 7. Berthoud HR. Metabolic and hedonic drives in the neural control of appetite: who is the boss? Curr Opin Neurobiol. 2011;21:888–96.
- 8. Berthoud HR. The neurobiology of food intake in an obesogenic environment. Proc Nutr Soc. 2012;71:478–87.
- Farr OM, Li CS, Mantzoros CS. Central nervous system regulation of eating: Insights from human brain imaging. Metabolism. 2016;65:699–713.
- Feeney E, O'Brien S, Scannell A, Markey A, Gibney ER. Genetic variation in taste perception: does it have a role in healthy eating? Proc Nutr Soc. 2011;70:135–43.

- Huang T, Hu FB. Gene-environment interactions and obesity: recent developments and future directions. BMC Med Genomics. 2015;8 Suppl 1:S2.
- Hsu TM, Suarez AN, Kanoski SE. Ghrelin: a link between memory and ingestive behavior. Physiol Behav. 2016. doi:10.1016/j-physbeh.2016.03.039.
- Smeets PA, Charbonnier L, van Meer F, van der Laan LN, Spetter MS. Food-induced brain responses and eating behaviour. Proc Nutr Soc. 2012;71:511–20.
- Berthoud HR, Lenard NR, Shin AC. Food reward, hyperphagia, and obesity. Am J Physiol Regul Integr Comp Physiol. 2011;300:R1266–77.
- Berthoud HR, Münzberg H, Richards BK, Morrison CD. Neural and metabolic regulation of macronutrient intake and selection. Proc Nutr Soc. 2012;71:390–400.
- Alonso-Alonso M, Woods SC, Pelchat M, Grigson PS, Stice E, Farooqi S, Khoo CS, Mattes RD, Beauchamp GK. Food reward system: current perspectives and future research needs. Nutr Rev. 2015;73:296–307.
- 17. Khanh DV, Choi YH, Moh SH, Kinyus AW, Kim KW. Leptin and insulin signaling in dopaminiergic neurons: relationship between energy balance and reward system. Front Psychol. 2014;5:Art. 846.
- Perello M, Dickson SL. Ghrelin signaling on food reward: a salient link between the gut and the mesolimbic system. J Neuroendocrinol. 2015;27:424–34.
- 19. Yau YH, Potenza MN. Stress and eating behaviors. Minerva Endocrinol. 2013;38:255–67.
- 20. Singh M. Mood, food, and obesity. Front Psychol. 2014;5:925.
- 21. Bryant EJ, King NA, Blundell JE. Disinhibition: its effects on appetite and weight regulation. Obes Rev. 2008;9:409–19.
- Bellisle F. Assessing various aspects of the motivation to eat that can affect food intake and body weight control. Encephale. 2009;35:182–5. abstract.
- Garaulet M, Canteras M, Morales E, López-Guimera G, Sánchez-Carracedo D, Corbalán-Tutau MD. Validation of a questionnaire on emotional eating for use in cases of obesity: the Emotional Eater Questionnaire (EEQ). Nutr Hosp. 2012;27:645–51.
- Jasinska AJ. Automatic inhibition and habitual control: alternative views in neuroscience research on response inhibition and inhibitory control. Front Behav Neurosci. 2013;7:25.
- Houben K, Nederkoorn C, Jansen A. Eating on impulse: the relation between overweight and food-specific inhibitory control. Obesity (Silver Spring). 2014;22:E6–8.
- Meule A, Kübler A. Double trouble. Trait food craving and impulsivity interactively predict food-cue affected behavioral inhibition. Appetite. 2014;79:174–82.
- Jasinska AJ, Yasuda M, Burant CF, Gregor N, Khatri S, Sweet M, Falk EB. Impulsivity and inhibitory control deficits are associated with unhealthy eating in young adults. Appetite. 2012;59:738–47.
- He Q, Xiao L, Xue G, Wong S, Ames SL, Schembre SM, Bechara A. Poor ability to resist tempting calorie rich food is linked to altered balance between neural systems involved in urge and self-control. Nutr J. 2014;13:92.
- 29. Ely AV, Winter S, Lowe MR. The generation and inhibition of hedonically-driven food intake: behavioral and neurophysiological determinants in healthy weight individuals. Physiol Behav. 2013;121:25–34.
- 30. Cornier MA, McFadden KL, Thomas EA, Bechtell JL, Eichman LS, Bessesen DH, Tregellas JR. Differences in the neuronal response to food in obesity-resistant as compared to obesity-prone individuals. Physiol Behav. 2013;110–111:122–8.
- van't Riet J, Sijtsema SJ, Dagevos H, De Bruijn GJ. The importance of habits in eating behaviour. An overview and recommendations for future research. Appetite. 2011;57:585–96.
- 32. Higgs S. Social norms and their influence on eating behaviours. Appetite. 2015;86:38-44.
- 33. Herman CP, Higgs S. Social influences on eating. An introduction to the special issue. Appetite. 2015;86:1-2.
- 34. Cruwys T, Bevelander KE, Hermans RC. Social modeling of eating: a review of when and why social influence affects food intake and choice. Appetite. 2015;86:3–18.
- 35. Robinson E, Thomas J, Aveyard P, Higgs S. What everyone else is eating: a systematic review and meta-analysis of the effect of informational eating norms on eating behavior. J Acad Nutr Diet. 2014;114:414–29.
- Robinson E, Blissett J, Higgs S. Social influences on eating: implications for nutritional interventions. Nutr Res Rev. 2013;26:166–76.
- 37. Herman CP. The social facilitation of eating. A review. Appetite. 2015;86:61-73.
- Wadhera D, Capaldi-Phillips ED. A review of visual cues associated with food on food acceptance and consumption. Eat Behav. 2014;15:132–43.
- Boswell RG, Kober H. Food cue reactivity and craving predict eating and weight gain: a meta-analysis review. Obes Rev. 2016;17:159–77.
- McCrickerd K, Forde CG. Sensory influences on food intake control: moving beyond palatability. Obes Rev. 2016;17:18–29.
- Mela DJ. Determinants of food choice: relationships with obesity and weight control. Obes Res. 2001;4(9 Suppl 4):249s–55.

- 42. Higgs S. Cognitive processing of food rewards. Appetite. 2015. doi:10.1080/13803395.2015.1121969.
- Ho A, Kennedy J, Dimitropoulos A. Neural correlates to food-related behavior in normal-weight and overweight/ obese participants. PLoS One. 2012;7:e45403.
- Levitsky DA, Iyer S, Pacanowski CR. Number of foods available at a meal determines the amount consumed. Eat Behav. 2012;13:183–7.
- 45. McCrory MA, Burke A, Roberts SB. Dietary (sensory) variety and energy balance. Physiol Behav. 2012;107:576–83.
- Keenan GS, Brunstrom JM, Ferriday D. Effects of meal variety on expected satiation: evidence for a 'perceived volume' heuristic. Appetite. 2015;89:10–5.
- Martin AA. Why can't we control our food intake? The downside of dietary variety on learned satiety responses. Physiol Behav. 2016. doi:10.1016/j.physbeh.2016.04.010.
- Wansink B, Painter JE, Lee YK. The office candy dish: proximity's influence on estimated and actual consumption. Int J Obes (Lond). 2006;30:871–5.
- 49. Privitera GJ, Zuraikat FM. Proximity of foods in a competitive food environment influences consumption of a low calorie and a high calorie food. Appetite. 2014;76:175–9.
- 50. Yeomans MR. Olfactory influences on appetite and satiety in humans. Physiol Behav. 2006;89:10-4.
- Albrecht J, Schreder T, Kleemann AM, Schöpf V, Kopietz R, Anzinger A, Demmel M, Linn J, Kettenmann B, Wiesmann M. Olfactory detection thresholds and pleasantness of a food-related and a non-food odour in hunger and satiety. Rhinology. 2009;47:160–5.
- Stafford LD, Tucker M, Gerstner N. A bitter sweet asynchrony. The relation between eating attitudes, dietary restraint on smell and taste function. Appetite. 2013;70:31–6.
- Zoon HF, He W, de Wijk RA, de Graaf C, Boesveldt S. Food preference and intake in response to ambient odours in overweight and normal-weight females. Physiol Behav. 2014;133:190–6.
- Peter Herman C, Polivy J, Pliner P, Vartanian LR. Mechanisms underlying the portion-size effect. Physiol Behav. 2015;144:129–36.
- 55. Rolls BJ. What is the role of portion control in weight management? Int J Obes (Lond). 2014;38 Suppl 1:S1-8.
- Burger KS, Fisher JO, Johnson SL. Mechanisms behind the portion size effect: visibility and bite size. Obesity (Silver Spring). 2011;19:546–51.
- Almiron-Roig E, Tsiountsioura M, Lewis HB, Wu J, Solis-Trapala I, Jebb SA. Large portion sizes increase bite size and eating rate in overweight women. Physiol Behav. 2015;139:297–302.
- Robinson E, Almiron-Roig E, Rutters F, de Graaf C, Forde CG, Tudur Smith C, Nolan SJ, Jebb SA. A systematic review and meta-analysis examining the effect of eating rate on energy intake and hunger. Am J Clin Nutr. 2014;100:123–51.
- 59. Chandon P, Wansink B. Does food marketing need to make us fat? A review and solutions. Nutr Rev. 2012;70:571–93.
- Cohen DA, Babey SH. Contextual influences on eating behaviours: heuristic processing and dietary choices. Obes Rev. 2012;13:766–79.
- Trapp GS, Hickling S, Christian HE, Bull F, Timperio AF, Boruff B, Shrestha D, Giles-Corti B. Individual, social, and environmental correlates of healthy and unhealthy eating. Health Educ Behav. 2015;42:759–68.
- 62. Kegler MC, Alcantara I, Haardörfer R, Gazmararian JA, Ballard D, Sabbs D. The influence of home food environments on eating behaviors of overweight and obese women. J Nutr Educ Behav. 2014;46:188–96.
- Kegler MC, Alcantara I, Veluswamy JK, Haardörfer R, Hotz JA, Glanz K. Results from an intervention to improve rural home food and physical activity environments. Prog Community Health Partnersh. 2012;6:265–77.
- 64. Sisk C, Sharkey JR, McIntosh WA, Anding J. Using multiple household food inventories to measure food availability in the home over 30 days: a pilot study. Nutr J. 2010;9:19.
- Gorin AA, Raynor HA, Fava J, Maguire K, Robichaud E, Trautvetter J, Crane M, Wing RR. Randomized controlled trial of a comprehensive home environment-focused weight-loss program for adults. Health Psychol. 2013;32:128–37.
- Fulkerson JA, Nelson MC, Lytle L, Moe S, Heitzler C, Pasch KE. The validation of a home food inventory. Int J Behav Nutr Phys Act. 2008;5:55.
- 67. Appelhans BM, Waring ME, Schneider KL, Pagoto SL. Food preparation supplies predict childrens family meal and home-prepared dinner consumption in low-income households. Appetite. 2014;76:1–8.
- Dubowitz T, Cohen DA, Huang CY, Beckman RA, Collins RL. Using a Grocery list is associated with a healthier diet and lower BMI among very high-risk adults. J Nutr Educ Behav. 2015;47:259–64.
- 69. Au N, Marsden G, Mortimer D, Lorgelly PK. The cost-effectiveness of shopping to a predetermined grocery list to reduce overweight and obesity. Nutr Diabetes. 2013;3:e77.
- Saarela AM, Lapveteläinen AT, Mykkänen HM, Kantanen TT, Rissanen RL. Real-life setting in data collection. The role of nutrition knowledge whilst selecting food products for weight management purposes in a supermarket environment. Appetite. 2013;71:196–208.
- Saarela AM. Change of behaviour when selecting food products in a supermarket environment after reminding consumers about weight management. Public Health Nutr. 2014;17:1147–55.

- Poelman MP, de Vet E, Velema E, Seidell JC, Steenhuis IH. Behavioural strategies to control the amount of food selected and consumed. Appetite. 2014;72:156–65.
- Carney D, Schultz S, Lim J, Walters W. Successful medical weight loss in a community setting. J Obes Weight Loss Ther. 2015;5:248.
- 74. Reimers K, Pardo S, Kawiecki D, Rippe J. Portion controlled frozen meals at lunch are associated with high compliance and perceived benefits during a weight management program. JADA. 2011;111(9): A83.
- Reimers K, Sinnett S, Papadopoulos T, Nguyen V, Yu Z, Rippe J. Commercially prepared single serving meals as lunch meal replacement produce energy deficit and weight loss. FASEB J. 2012;26:636.
- 76. Summers A, Nguyen V, Brosnahan J, Zukley L, Lowndes J, Pohl R, Reimers K, Angelopoulos TJ, Rippe JM. Improved weight and body composition following a structured diet plan incorporating healthy convenient prepared meals. FASEB J. 2009;23:550.10.
- 77. Raynor HA, Anderson AM, Miller GD, Reeves R, Delahanty LM, Vitolins MZ, Harper P, Mobley C, Konersman K, Mayer-Davis E, Look AHEAD Research Group. Partial meal replacement plan and quality of the diet at 1 year: action for health in diabetes (look AHEAD) trial. J Acad Nutr Diet. 2015;115:731–42.
- Levitsky DA, Pacanowski C. Losing weight without dieting. Use of commercial foods as meal replacements for lunch produces an extended energy deficit. Appetite. 2011;57:311–7.
- Nguyen V, Summers A, Brosnahan J, Zukley L, Lowndes J, Reimers K, Angelopoulos TJ, Rippe JM. Healthy convenient prepared meals improve dietary quality during structured dietary intervention for heart disease risk factor reduction. FASEB J. 2009;23:550.10.
- Cheskin LJ, Mitchell AM, Jhaveri AD, Mitola AH, Davis LM, Lewis RA, Yep MA, Lycan TW. Efficacy of meal replacements versus a standard food-based diet for weight loss in type 2 diabetes: a controlled clinical trial. Diabetes Educ. 2008;34:118–27.
- Reicks M, Trofholz AC, Stang JS, Laska MN. Impact of cooking and home food preparation interventions among adults: outcomes and implications for future programs. J Nutr Educ Behav. 2014;46:259–76.
- Hersch D, Perdue L, Ambroz T, Boucher JL. The impact of cooking classes on food-related preferences, attitudes, and behaviors of school-aged children: a systematic review of the evidence, 2003–2014. Prev Chronic Dis. 2014;11:E193.
- Archuleta M, Vanleeuwen D, Halderson K, Jackson K, Bock MA, Eastman W, Powell J, Titone M, Marr C, Wells L. Cooking schools improve nutrient intake patterns of people with type 2 diabetes. J Nutr Educ Behav. 2012;44:319–25.
- 84. Poelman MP, de Vet E, Velema E, de Boer MR, Seidell JC, Steenhuis IH. PortionControl@HOME: results of a randomized controlled trial evaluating the effect of a multi-component portion size intervention on portion control behavior and body mass index. Ann Behav Med. 2015;49:18–28.
- English L, Lasschuijt M, Keller KL. Mechanisms of the portion size effect. What is known and where do we go from here? Appetite. 2015;88:39–49.
- Lewis HB, Forwood SE, Ahern AL, Verlaers K, Robinson E, Higgs S, Jebb SA. Personal and social norms for food portion sizes in lean and obese adults. Int J Obes (Lond). 2015;39:1319–24.
- Brogden N, Almiron-Roig E. Estimated portion sizes of snacks and beverages differ from reference amounts and are affected by appetite status in non-obese men. Public Health Nutr. 2011;14:1743–51.
- Almiron-Roig E, Solis-Trapala I, Dodd J, Jebb SA. Estimating food portions. Influence of unit number, meal type and energy density. Appetite. 2013;71:95–103.
- 89. Cypel YS, Guenther PM, Petot GJ. Validity of portion-size measurement aids: a review. J Am Diet Assoc. 1997;97:289–92.
- Hollands GJ, Shemilt I, Marteau TM, Jebb SA, Lewis HB, Wei Y, Higgins JP, Ogilvie D. Portion, package or tableware size for changing selection and consumption of food, alcohol and tobacco. Cochrane Database Syst Rev. 2015;9:CD011045.
- 91. Ball GD, Friedman A. Dice, golf balls, and CDs: assumptions about portion size measurement aids. Can J Diet Pract Res. 2010;71:146–9.
- Byrd-Bredbenner C, Schwartz J. The effect of practical portion size measurement aids on the accuracy of portion size estimates made by young adults. J Hum Nutr Diet. 2004;17:351–7.
- Macdiarmid JI, Loe J, Kyle J, McNeill G. "It was an education in portion size". Experience of eating a healthy diet and barriers to long term dietary change. Appetite. 2013;71:411–9.
- Roe LS, Meengs JS, Rolls BJ. Salad and satiety. The effect of timing of salad consumption on meal energy intake. Appetite. 2012;58:242–8.
- Williams RA, Roe LS, Rolls BJ. Comparison of three methods to reduce energy density. Effects on daily energy intake. Appetite. 2013;66:75–83.
- Rolls BJ, Roe LS, Meengs JS. Portion size can be used strategically to increase vegetable consumption in adults. Am J Clin Nutr. 2010;91:913–22.
- Robinson E, Nolan S, Tudur-Smith C, Boyland EJ, Harrold JA, Hardman CA, Halford JC. Will smaller plates lead to smaller waists? A systematic review and meta-analysis of the effect that experimental manipulation of dishware size has on energy consumption. Obes Rev. 2014;15:812–21.

- Libotte E, Siegrist M, Bucher T. The influence of plate size on meal composition. Literature review and experiment. Appetite. 2014;82:91–6.
- Robinson E, Hardman CA, Halford JC, Jones A. Eating under observation: a systematic review and meta-analysis of the effect that heightened awareness of observation has on laboratory measured energy intake. Am J Clin Nutr. 2015;102:324–37.
- Kesman RL, Ebbert JO, Harris KI, Schroeder DR. Portion control for the treatment of obesity in the primary care setting. BMC Res Notes. 2011;4:346.
- Pedersen SD, Kang J, Kline GA. Portion control plate for weight loss in obese patients with type 2 diabetes mellitus: a controlled clinical trial. Arch Intern Med. 2007;167:1277–83.
- 102. Huber JM, Shapiro JS, Wieland ML, Croghan IT, Vickers Douglas KS, Schroeder DR, Hathaway JC, Ebbert JO. Telecoaching plus a portion control plate for weight care management: a randomized trial. Trials. 2015;16:323.
- O'Reilly GA, Cook L, Spruijt-Metz D, Black DS. Mindfulness-based interventions for obesity-related eating behaviours: a literature review. Obes Rev. 2014;15:453–61.
- Godfrey KM, Gallo LC, Afari N. Mindfulness-based interventions for binge eating: a systematic review and metaanalysis. J Behav Med. 2015;38:348–62.
- 105. Robinson E, Aveyard P, Daley A, Jolly K, Lewis A, Lycett D, Higgs S. Eating attentively: a systematic review and meta-analysis of the effect of food intake memory and awareness on eating. Am J Clin Nutr. 2013;97:728–42.
- 106. Higgs S. Manipulations of attention during eating and their effects on later snack intake. Appetite. 2015;92:287–94.
- 107. Papies EK, Pronk TM, Keesman M, Barsalou LW. The benefits of simply observing: mindful attention modulates the link between motivation and behavior. J Pers Soc Psychol. 2015;108:148–70.
- 108. Schaefer JT, Magnuson AB. A review of interventions that promote eating by internal cues. J Acad Nutr Diet. 2014;114:734–60.
- Framson C, Kristal AR, Schenk JM, Littman AJ, Zeliadt S, Benitez D. Development and validation of the mindful eating questionnaire. J Am Diet Assoc. 2009;109:1439–44.
- Robinson E, Higgs S, Daley AJ, Jolly K, Lycett D, Lewis A, Aveyard P. Development and feasibility testing of a smart phone based attentive eating intervention. BMC Public Health. 2013;13:639.
- 111. Robinson E, Kersbergen I, Higgs S. Eating 'attentively' reduces later energy consumption in overweight and obese females. Br J Nutr. 2014;112:657–61.
- 112. Corsica J, Hood MM, Katterman S, Kleinman B, Ivan I. Development of a novel mindfulness and cognitive behavioral intervention for stress-eating: a comparative pilot study. Eat Behav. 2014;15:694–9.
- 113. Katterman SN, Kleinman BM, Hood MM, Nackers LM, Corsica JA. Mindfulness meditation as an intervention for binge eating, emotional eating, and weight loss: a systematic review. Eat Behav. 2014;15:197–204.
- 114. Cohen S. A global measure of perceived stress. J Health Soc Beh. 1983;24:385-96.
- 115. Belli BB, Beto JA. Nutrition counseling and education skills for dietetics professionals. 6th ed. Philadelphia: Wolters Kluwer/Lippincott, Wiliams & Wilkens; 2014.
- 116. Foreyt JP, Poston 2nd WS. What is the role of cognitive-behavior therapy in patient management? Obes Res. 1998;6 Suppl 1:18S–22S.
- 117. Foreyt JP, Poston 2nd WS. The role of the behavioral counselor in obesity treatment. J Am Diet Assoc. 1998;98(10 Suppl 2):S27–30.
- 118. Spahn JM, Reeves RS, Keim KS, Laquatra I, Kellogg M, Jortberg B, Clark NA. State of the evidence regarding behavior change theories and strategies in nutrition counseling to facilitate health and food behavior change. J Am Diet Assoc. 2010;110:879–91.
- Forman EM, Butryn ML. A new look at the science of weight control: how acceptance and commitment strategies can address the challenge of self-regulation. Appetite. 2015;84:171–80.
- Forman EM, Butryn ML, Manasse SM, Bradley LE. Acceptance-based behavioral treatment for weight control: a review and future directions. Curr Opin Psychol. 2015;2:87–90.
- Thoma N, Pilecki B, McKay D. Contemporary cognitive behavior therapy: a review of theory, history, and evidence. Psychodyn Psychiatry. 2015;43:423–61.
- 122. Prestwich A, Kellar I, Parker R, MacRae S, Learmonth M, Sykes B, Taylor N, Castle H. How can self-efficacy be increased? Meta-analysis of dietary interventions. Health Psychol Rev. 2014;8:270–85.
- 123. Teixeira PJ, Carraça EV, Marques MM, Rutter H, Oppert JM, De Bourdeaudhuij I, Lakerveld J, Brug J. Successful behavior change in obesity interventions in adults: a systematic review of self-regulation mediators. BMC Med. 2015;13:84.
- Van Dorsten B, Lindley EM. Cognitive and behavioral approaches in the treatment of obesity. Med Clin North Am. 2011;95:971–88.
- 125. Hedman E, Andersson E, Lekander M, Ljótsson B. Predictors in Internet-delivered cognitive behavior therapy and behavioral stress management for severe health anxiety. Behav Res Ther. 2015;64:49–55.
- 126. Michie S, Abraham C, Whittington C, McAteer J, Gupta S. Effective techniques in healthy eating and physical activity interventions: a meta-regression. Health Psychol. 2009;28:690–701.

- 127. Burke LE, Wang J, Sevick MA. Self-monitoring in weight loss: a systematic review of the literature. J Am Diet Assoc. 2011;111:92–102.
- 128. Zheng Y, Klem ML, Sereika SM, Danford CA, Ewing LJ, Burke LE. Self-weighing in weight management: a systematic literature review. Obesity (Silver Spring). 2015;23:256–65.
- 129. Desroches S, Lapointe A, Deschênes SM, Bissonnette-Maheux V, Gravel K, Thirsk J, Légaré F. Dietitians' perspectives on interventions to enhance adherence to dietary advice for chronic diseases in adults. Can J Diet Pract Res. 2015;76:103–8.
- Kirk SF, Penney TL, McHugh TL, Sharma AM. Effective weight management practice: a review of the lifestyle intervention evidence. Int J Obes (Lond). 2012;36:178–85.
- Montesi L, El Ghoch M, Brodosi L, Calugi S, Marchesini G, Dalle GR. Long-term weight loss maintenance for obesity: a multidisciplinary approach. Diabetes Metab Syndr Obes. 2016;26:37–46.
- 132. Krukowski RA, Sparks C, DiCarlo M, McSweeney J, West DS. There's more to food store choice than proximity: a questionnaire development study. BMC Public Health. 2013;13:586.
- 133. Larson N, Story M. A review of environmental influences on food choices. Ann Behav Med. 2009;38 Suppl 1:S56–73.
- Ollberding NJ, Wolf RL, Contento I. Food label use and its relation to dietary intake among US adults. J Am Diet Assoc. 2011;111(Suppl):S47–51.
- Miller LM, Cassady DL. The effects of nutrition knowledge on food label use. A review of the literature. Appetite. 2015;92:207–16.
- Roberto CA, Khandpur N. Improving the design of nutrition labels to promote healthier food choices and reasonable portion sizes. Int J Obes (Lond). 2014;38 Suppl 1:S25–33.
- Gittelsohn J, Laska MN, Karpyn A, Klingler K, Ayala GX. Lessons learned from small store programs to increase healthy food access. Am J Health Behav. 2014;38:307–15.
- 138. Gittelsohn J, Lee-Kwan SH, Batorsky B. Community-based interventions in prepared-food sources: a systematic review. Prev Chronic Dis. 2013;10:E180.
- 139. Gittelsohn J, Rowan M, Gadhoke P. Interventions in small food stores to change the food environment, improve diet, and reduce risk of chronic disease. Prev Chronic Dis. 2012;9:E59.
- 140. Escaron AL, Meinen AM, Nitzke SA, Martinez-Donate AP. Supermarket and grocery store-based interventions to promote healthful food choices and eating practices: a systematic review. Prev Chronic Dis. 2013;10:E50.
- 141. Olstad DL, Ball K, Abbott G, McNaughton SA, Le HN, Ni Mhurchu C, Pollard C, Crawford DA. A process evaluation of the Supermarket Healthy Eating for Life (SHELf) randomized controlled trial. Int J Behav Nutr Phys Act. 2016;13:27.
- Krukowski RA, McSweeney J, Sparks C, West DS. Qualitative study of influences on food store choice. Appetite. 2012;59:510–6.
- 143. Webber CB, Sobal J, Dollahite JS. Shopping for fruits and vegetables. Food and retail qualities of importance to low-income households at the grocery store. Appetite. 2010;54:297–303.
- 144. Martin KS, Ghosh D, Page M, Wolff M, McMinimee K, Zhang M. What role do local grocery stores play in urban food environments? A case study of Hartford-Connecticut. PLoS One. 2014;9:e94033.
- 145. Holmes AS, Estabrooks PA, Davis GC, Serrano EL. Effect of a grocery store intervention on sales of nutritious foods to youth and their families. J Acad Nutr Diet. 2012;112:897–901.
- 146. Swartz JJ, Braxton D, Viera AJ. Calorie menu labeling on quick-service restaurant menus: an updated systematic review of the literature. Int J Behav Nutr Phys Act. 2011;8:135.
- 147. Sinclair SE, Cooper M, Mansfield ED. The influence of menu labeling on calories selected or consumed: a systematic review and meta-analysis. J Acad Nutr Diet. 2014;114:1375–88.
- 148. Long MW, Tobias DK, Cradock AL, Batchelder H, Gortmaker SL. Systematic review and meta-analysis of the impact of restaurant menu calorie labeling. Am J Public Health. 2015;105:e11–24.
- Breck A, Cantor J, Martinez O, Elbel B. Who reports noticing and using calorie information posted on fast food restaurant menus? Appetite. 2014;81:30–6.
- 150. Oh A, Nguyen AB, Patrick H. Correlates of reported use and perceived helpfulness of calorie information in restaurants among U.S. adults. Am J Health Promot. 2016;30:242–9.
- Lee-Kwan SH, Pan L, Maynard LM, McGuire LC, Park S. Factors associated with self-reported menu-labeling among US adults. J Acad Nutr Diet. 2016. doi:10.1016/j.jand.2015.12.015.

Chapter 5 Critical Evaluation of Nutrition Research

Andrew W. Brown and Michelle M. Bohan Brown

Key Points

- Nutrition is a broad field, touching many disciplines and fields of practice that may or may not communicate with similar terminology.
- Numerous study designs can be employed to study acute and chronic exposures to nutrition, requiring precision in describing exactly what and how it was studied.
- The personal nature of nutrition in daily life can make reading, interpreting, and communicating nutrition science onerous, making it important to make things as simple as possible but no simpler, avoiding logical fallacies, and focusing on the science rather than extrascientific factors.

Keywords Nutrition • Research reporting • Scientific integrity • Scientific rigor • Study design

Abbreviations

- AEI Actual Energy Intake
- RCT Randomized Controlled Trial
- SNAP Supplemental Nutrition Assistance Program
- SREI Self-Reported Energy Intake
- USDA United States Department of Agriculture
- WIC Special Supplemental Nutrition Program for Women Infants, and Children

A.W. Brown, PhD (🖂)

Office of Energetics and Nutrition Obesity Research Center, University of Alabama at Birmingham, 435 Lister Hill Library, 1700 University Blvd, Birmingham, AL 35294, USA e-mail: awbrown@uab.edu

M.M. Bohan Brown, PhD Food, Nutrition, and Packaging Sciences Department, Clemson University, 219 Poole Agricultural Center, Clemson, SC 29634, USA e-mail: mbohanb@clemson.edu

Introduction

Despite the increasing focus placed on the importance of nutrition for optimal health, nutrition as a science is often criticized in lay media and casual conversation as constantly conflicting with itself, and experts are ridiculed for seeming to change their minds. This unease is most pithily stated as, "if you don't like what nutrition science says about good nutrition today, just wait until tomorrow; it'll change." This has played out with supporting "super" foods or demonizing "toxic" foods; changes in the perceived healthfulness of food categories like meats, fruits, and dairy; and even macronutrients in general, such as carbohydrates or fat. Although there is much that is uncertain about the effects of foods on health, some of these seeming contradictions can come from misunderstanding several things about nutrition science. First, there are people who share amazing anecdotes as though they are the generalizable truth. These people have interesting stories to share, but it is not science. Second, our understanding of the natural world, by the very nature of the scientific method, is expected to change. Therefore, at least in some cases, our collective understanding of nutrition and health does change from one day to the next. This is the purpose of scientific pursuits.

However, there are still issues with the conduct and communication of science itself that may contribute to the confusion. This chapter is designed to be a primer for readers of scientific literature to identify some of the challenges in evaluating what was studied, determining how it was studied, and interpreting what we can conclude from studies. Throughout we will use case studies to show potential sources of confusion and illustrate that problems do occur in practice. However, we stress that these are cases and anecdotes to help readers identify what to look for and should not be interpreted as declarations of representativeness or frequencies of occurrence.

What Exactly Was Studied?

How does one look for what we know about nutrition? Today, one can type into any search engine the topic of interest. However, what the search yields can be a list of varying results from a variety of studies. For example, when one searches for nutritive value of a particular food, the results can contain studies that investigate the calories of that food or micronutrient content of the food. Confusion over findings that appear erroneous or conflicting can arise from the definition of the language used.

Using the Same Language for Different Topics

The impact nutrition has on human health is a topic area that interests scientists, policy makers, and the public, each using different subsets of language. Words can have multiple definitions (Table 5.1) or be inherently vague (e.g., high protein, low carbohydrate, breakfast, snacking). Clearly defining the exposure and the outcome is the first step in knowing what was studied in nutrition research. On the surface, defining the outcome and the exposure appears easy. However, several factors complicate defining exposures and outcomes. Is the appropriate definition the one a researcher-contrived for a specific study, the definition a politician used to set policy, or a connotation generally accepted by society at a particular time? Is it broadly defined or highly specified? Is it defined chemically or abstractly? Using the same words but referring to different topics studied in nutrition research can influence the conclusions and application of the results from nutrition studies. Below are several case studies.

Word	Interpretation 1	Interpretation 2	
Nutritive	Containing micronutrients	Containing calories	
Significant	Important	<i>p</i> <0.05	
Reduce	Decrease	Gaining electrons	
Toxicity	Acutely hazardous	Capacity to cause harm	
Uncertainty	Anything is possible	Constraining the known	
Diet	Restrictions on eating behaviors	Eating behaviors	
Bias	Mathematical deviation from true results	Human distortion	

 Table 5.1 Examples of words frequently used to describe foods and nutrients with different interpretations

Case Study: Reducing Cholesterol

Reduction of cholesterol has at least two denotations. The more commonly used meaning in nutrition research studying cholesterol is to decrease blood concentrations of cholesterol. However, another use of reduction of cholesterol in nutrition research is the chemical reduction of cholesterol to coprostanol, which occurs, for instance, from metabolism by gut microbes. Reduce here has two meanings: the first case is "to decrease" and the second case is a chemical reaction (the gaining of electrons). The difference is often clear by context, particularly for cholesterol, but if there is uncertainty the questions can be rephrased, such as "how do we decrease cholesterol concentrations in humans" or "how do we reduce cholesterol to coprostanol." Defining exactly what was done by clear and detailed language use is crucial in the understanding, interpreting, and meta-analyzing of results from nutrition research.

Case Study Snacks, Breakfast, and Meals as Exposures

Breakfast definitions across lay and scientific publications range from specifying foods and times (e.g., oatmeal no later than 9 a.m.) to an assortment of 32 separate definitions of eating breakfast and skipping breakfast depending on types of foods and number of days consumed [1]. In the case of breakfast, the researcher must create an operational definition of what in their study constitutes breakfast, which may or may not match the definition of the next researcher, clinician, or member of the public. Breakfast itself has several components that one must think about: time of day, number of times per week, amount eaten, and composition of the meal. The wide array of possible definitions of breakfast make the current research more difficult to synthesize and apply when either the denotation is not clear or is not specified [2].

Similarly, snacking versus meals have disparate denotations depending on the group defining them [3]. Snacking versus meals has been defined by researchers either using the time of day the food was eaten with meals given prespecified times (e.g., 8–10 a.m. is breakfast) or using a food taxonomy and amount of calories eaten [4]. Others have noted that a number of different contexts guide what an individual thinks is a snack versus a meal: respondents associated snacking with paper napkins, standing, and "unhealthy" foods, while associating meals with cloth napkins, sitting, and healthy, prepared foods [5]. Meals and snacking are, therefore, complex topics that can be oversimplified. We address this oversimplification in section "Oversimplification of Complex Topics".

Case Study: Obesity as an Outcome

Just as with poorly defined exposures, a poorly defined outcome does not lend well to understanding nutrition research. One such outcome that is broadly defined is obesity. Many nutritional interventions have been investigated in an effort to decrease obesity, but what a given researcher defines as an

obesity-related outcome can vary widely. Obesity is defined by professional societies in a number of ways, including descriptions of "excess body fat" (which, of course, raises additional questions of how "excess" is characterized) or metabolic dysfunction. However, researchers operationalize obesity by measuring body weight, body mass index, fat mass, fat mass index, body fat percentage, adiposity, waist circumference, waist-to-hip ratio, skinfold thickness, and categorized overweight/obesity defined by any of these methods, among others. When investigating whether or not a particular food or nutrient will have an impact on obesity, a more precise description other than simply the word "obesity" must be employed.

Oversimplification of Complex Topics

When communicating results from nutrition research, simplistic statements are common in an effort to present the results in a manner that the audience understands. Making simplistic conclusions from complex research may superficially seem to be the best way to convey the research to the intended audience. At other times, oversimplification is unintended, such as in the cases described in trying to define breakfast, meals, or snacks. The meaning of the research can be lost or misleading when overly simplified. One such statement is "eat more fruits and vegetables," exemplified by the "more matters" campaign [6], when in actuality the goal is often "eat more fruits and vegetables in place of energy dense foods." Einstein's Razor, stated as "make things as simple as possible, but not simpler," can aptly be applied to nutrition research. By distilling a statement about a topic to an oversimplified snippet, the meaning and value have changed.

Case Study: Empty Calories, Nutritive Value, and Junk Food

The United States Department of Agriculture (USDA) defines empty calories as calories contained in a food that do not have vitamins or minerals associated with them [7]. This is a distillation of the idea that when creating a diet, the diet should contain nutrient dense foods (here, specifically referring to micronutrients). The concept of empty calories was born to account for the calories in foods that are associated with extra calories coming from added sugars and fats. The concept of minimizing the amount of calories ingested that are not associated with other nutritive components is not a new concept. In the old USDA Food Guide Pyramid, these types of energy-containing items were located at the top with oils, sugars, and sweets being items one should consume sparingly. In Table 5.2, some commonly eaten foods are listed with the total calories, total empty calories, and percent empty calories each food contains. Empty calories do not just arise from the individual adding items such as sugar or fats to a food but also portion size. Furthermore, there are recommendations on how many empty calories a man or a woman should consume in a day.

Thus far, the concept of nutrient density and limiting calories—specifically empty calories—may seem straight forward and consistent with years of recommendations. However, when one looks at the numbers in Table 5.2, a woman between the ages of 31 and 50 who has a glass of wine (consistent with interpretations of the Mediterranean Diet or moderate intake of alcohol in US dietary recommendations) cannot consume anything else in the table on the same day, with the exception of white bread, because she is limited to a total of 160 empty calories.

Adding to the confusion is the use of nonquantitative or poorly defined terms, such as "excess" (e.g., excess sugar and fat) or "nutritive value." Currently, foods that are high in calories but low in micronutrients are often considered to have little nutritional value. However, in 1905, the USDA stated that "[t]he common fruits, because of their low nutritive value, are not, as a rule, estimated at their real worth as food" [8]. Fruits today are typically considered "good" and nutrient dense,

Food	Total calories	Empty calories	Percent empty
5 oz table wine	121	121	100
2 slices, white bread	138	0	0
2×1 oz patties of pork sausage	204	96	47
3 oz roasted chicken thigh w/skin	209	47	22
1 c frozen yogurt	224	119	53
3 oz regular, 80 % lean ground beef	229	64	28
1 medium, 2 oz croissant	231	111	48
1 c fruit flavored, low-fat yogurt	250	152	61
1 small, 2 oz blueberry muffin	259	69	27
3×1 oz slices of beef bologna	261	150	57

Table 5.2 Examples of total calories, empty calories, and percent empty calories for a sample of foods [7]

containing 0 empty calories, but using this older connotation of nutritive value would have seemingly classified them as were junk food.

But then what makes a food junk? Two common definitions are a food that contains little nutritive value or a food with added calories from sugar and fat. Thus, empty calories might often be used as a euphemism for junk food. However, the Physicians Committee for Responsible Medicine repeatedly refers to cheese, meat, dairy, and other nutrient-dense foods as "junk foods." Such a classification starkly contrasts with the USDA recommendations for selecting meats and cheeses to meet the nutrient requirements that come from the protein and dairy groups while decreasing the number of empty calories. Terms such as "empty calories," "nutritive value," and "junk food," therefore, are conceptually appealing to discriminate "good" from "bad" foods, but without a comprehensible and consistent definition that fits within practical limits, their value in research and practice remains questionable and ripe for abuse by organizations pushing agendas against certain foods.

Case Study: Whole Versus Processed Foods

A hot topic in nutrition is decreasing the amount of processed food in the diet, but food processing is a complex issue. What constitutes a processed versus whole food occurs along a spectrum, rather than a discrete dichotomization. There is no definition supplied by the USDA on the MyPlate recommendations, and a previous spokesperson for the Academy of Nutrition and Dietetics cautions that, "We get really caught up in the word processed without realizing what it truly means" [9]. Processing includes actions such as cutting, washing, removing nuts from shells, and deboning of fish. Some processing of foods such as washing and irradiation are done to minimize food-borne illnesses.

Such nebulous definitions of processing are also important in research. A study titled "Postprandial energy expenditure in whole-food and processed-food meals: implications for daily energy expenditure" [10] may be more aptly titled, "Tale of Two Cheese Sandwiches." Although the title implies that the study investigated the effect of whole versus processed foods, a more accurate description is that it studied thermogenic differences between two kinds of cheese sandwiches. In all fairness, comparing whole versus processed foods is conceptually difficult. First, as already mentioned, what defines a food as whole or processed? Second, once one finds foods fitting the definition, how can one make comparable treatments in energy and nutrient content given that the only difference of interest is processing? Third, the scope of inference may be limited to the whole and processed foods used in the study and not extrapolated to all whole versus all processed foods. First, the "whole food" sandwich was made with whole grain bread, the "processed" with white bread; the former using sliced cheddar cheese and the latter using American singles. Do whole versus white breads and cheese versus cheese product adequately represent whole versus processed foods? Second, besides the nominal

classification of processing, the sandwiches differed in several other important ways. The authors reported some differences in nutrients, such as a 5% difference in protein content, a 10% difference in carbohydrate content, and a 6% difference in lipid content, which will influence the thermic effect of the foods. Also, the sandwiches differed in overall weight. Third, this is a study of a whole-grain bread, cheddar cheese sandwich versus a white bread, American-singles sandwich. Although the study seemed fairly well conducted and controlled, the conclusion that the results reflect whole versus processed meals may generalize too far.

How Exactly Was It Studied?

Causal Inference and a Hierarchy of Evidence

Much of the biomedical literature is interested in determining causation so that policy or clinical recommendations can change an exposure to improve an outcome. There are many great primers and in-depth discussions of causation [11] that go far beyond the constraints of this chapter. Here, we will simply say that causal inference is the scientific exercise of determining whether one thing (an outcome) occurs as a result of another thing (an exposure) happening or being changed. Questions fitting this form include: Does increasing exercise intensity decrease obesity? Does eating omega-3 fatty acids decrease cardiovascular events? Does providing free school breakfast improve school performance? These all take the logical form of "an exposure causes an outcome," "A causes B," or simply abbreviated as $A \rightarrow B$.

One of the more thoughtful and oft-invoked discussions of causation in biomedicine comes from Sir Austin Bradford Hill's nine viewpoints [12]. Here are some of the most commonly discussed viewpoints: *strength*—the size of the association or effect of the exposure on the outcome is large enough that alternative explanations are unlikely; *consistency*—the association or the effect is seen repeatedly with different studies and study types; *temporality*—the purported cause (exposure) must come before the effect (outcome); and *biological gradient* (i.e., dose–response)—the benefit or detriment of the exposure changes with amount of exposure. Each viewpoint needs to be considered in the context of the question at hand. For instance, many things in biology, and particularly with nutrition, are U-shaped; a low amount of a vitamin may represent a deficiency, while a high amount may cause toxicity. Thus, the biological gradient may be nonlinear for nutrients, but monotonic for toxins. Furthermore, these viewpoints often need to be considered in toto, rather than independently. Hill himself indicated that none of the viewpoints are necessary or sufficient to conclude causation; indeed, when they are considered in isolation, they can quickly become fodder for logical fallacies (see section "Logical Fallacies").

Of particular importance in these viewpoints is Hill's identification that with experimentation "the strongest support for the causation hypothesis may be revealed," which is reflected in hierarchies of evidence. Hierarchies of evidence are often depicted as triangles or pyramids, with randomized controlled trials (RCTs) at the top. Sometimes, systematic reviews and meta-analyses of RCTs will be placed at the top, which is a means of recognizing the need to consider a body of evidence when making conclusions. Other versions of the hierarchy also include animal studies or cell culture studies, placed at different levels of importance, but always below RCTs and controlled longitudinal studies; case reports; case–control studies; noncontrolled longitudinal studies; controlled longitudinal studies; and randomized-controlled studies. The idea is that, from bottom to top of the pyramid, there is an increase in causal inference in humans. The oft-unstated assumption, however, is that the studies are really comparing apples to apples: all study types are looking at scientifically sound measurements of exposures, meaningful outcomes, and human-relevant models (ideally in living humans). When these three characteristics are not met, it is not immediately clear which study design would necessarily give the most causal inference for human outcomes. For example, which provides the best evidence for the causal effects of a nutrient on cardiovascular disease in humans: a randomized experiment with exposure to reasonable levels of a nutrient and measured cardiovascular outcomes in a well-characterized animal model or the association between the self-reported consumption of the nutrient and an intermediate cardiovascular endpoint (blood pressure) in humans? It is unclear a priori which will prove to point toward the ultimate truth: the study with well-defined exposures and meaningful outcomes, but in animals; or the one in humans, but with mediocre measures of exposure and surrogate endpoints.

Given that an RCT is testing a well-characterized exposure, measuring meaningful endpoints, and in humans, the RCTs or systematic meta-analyses thereof provide the best evidence for causation in humans. RCTs decrease bias in at least two ways. First, the assignment of a treatment to individuals removes many of the concerns about the directionality of a relationship discussed with observational studies. For instance, in observational studies there is always the possibility that although A is associated with B, A may not cause B. Second, randomization minimizes the influence of confounding factors on outcomes across RCTs. The use of stratification in RCTs can help ensure that such a balance is reached (e.g., by making sure that there are an equal number of men and women enrolled, or that subjects have a representative distribution of incomes between groups).

It is important to stress that this hierarchy of evidence is for determining causation between A and B, which is a reductionist principle. Some in epidemiology or systems science might argue that creating the constraint of $A \rightarrow B$ is too simplistic, owing to the fact that often A may also cause changes in other outcomes, that in turn might alter the final change in B. This criticism is indeed a concern for lifestyle medicine: telling an individual to eat a low fat diet will undoubtedly change many other characteristics of the diet beyond just fat; telling an individual to exercise may result in decreases in spontaneous physical activity. We discuss this more in section "Conclusions Versus Decisions". However, the criticism of $A \rightarrow B$ causal models must also consider that much individualized or public health advice is communicated in such a reductionist paradigm: "eat more fruits and vegetables to prevent obesity" takes the form of "fruits and vegetables \rightarrow lower obesity"; "schools need to include physical education to improve scholastic performance" takes the form of "physical education \rightarrow better grades." We will discuss challenges in communicating lifestyle sciences in section "Communicating Science".

Can the Evidence Inform What Is Sought?

As the hierarchy of evidence demonstrates, not all studies are created equal and certainly quality may vary within a study type.

Surrogates: Is the Study Measuring What It Means To?

Actually measuring the exposures and outcomes that researchers want can often be insurmountably difficult, whether because of time, money, or ethics, especially in human research. In many cases, intermediate endpoints, biomarkers, or surrogate exposures are used (Table 5.3). These limitations do not necessarily invalidate a study's findings, but are important to consider when communicating the results of a study. In some cases, though, the limitations can be invalidating, as described below.

Exposure or outcome of interest	Exposure or outcome measured/administered
Obesity	Energy intake at a single subsequent meal
Fruit and vegetable consumption	Self-reported intake of fruits and vegetables
Poverty	Feeling of relatively lower buying power in a controlled, acute experiment
Cardiovascular disease	Low-density lipoprotein cholesterol
Diabetes	Fasting blood glucose
Chronic breakfast skipping	Skipping during a single morning intervention
Habitual exercise	Controlled exercise intervention with a research trainer/observer

Table 5.3 Examples of exposures or outcomes of interest versus what was actually studied or administered

Case Study: Self-Reported Energy Intake The use of self-reported energy intake (SREI) has been used for years to make conclusions about actual energy consumption, but some methodologists of late (including the authors here) have made an attempt to explain why SREI is invalid as an estimate of actual energy intake (AEI) and is no longer fit for scientific conclusions [13]. SREI involves a researcher asking participants through food frequency questionnaires, 24-h dietary recalls, food diaries, or other methods, to report what he or she has consumed. The researcher then assigns energy values to each reported food and drink, such as by extracting energy information from a standardized food database. At least as early as 1953, researchers demonstrated that different people report their energy differently. In one particular study, overweight women reported eating fewer calories than their normal weight counterparts when compared to an external observer [14]. Although an external observer still employs some degree of subjectivity, this observation was confirmed when an objective measurement of energy balance (the doubly labeled water technique) was applied to humans [15]. Indeed, different people misreported their consumptions, with larger individuals underreporting more. Misreporting of energy intake has also been associated with age, sex, and obesity. Clearly, SREI does not measure AEL

Bias: Is Our Study as Rigorous as We Think?

RCTs are often placed atop the hierarchy of studies because of their ability to decrease bias. However, this is predicated on RCTs actually being designed and executed in a way that decreases bias. This can be difficult in nutrition experiments, where blinding or masking the participants is often not possible, particularly in studies using foods: participants and researchers alike know when participants are getting sandwiches versus salads. There are ways to decrease biases, though, like concealing allocation until after randomization, blinding those taking outcome measurements, and masking the treatments from those performing statistical analyses. When controls are not in place, there is an increased risk of bias, which in this context refers to humans influencing the results, intentionally or unintentionally.

Case Study: Lanarkshire Milk Experiment In an attempt to see whether milk helped children grow, a massive RCT was conducted involving 20,000 children at 67 schools in which students were randomized to receive milk (pasteurized or raw) or not [16]. Unfortunately, those administering the treatments reconfigured the randomization such that they "must have unconsciously made too large a substitution of the ill-nourished among the 'feeders' and too few among the 'controls,'" which in turn resulted in a large baseline imbalance between the two groups. Thus, the randomization scheme was undermined and so too was the causal inference to be drawn from the study.

Confounding: Is the Study Actually Comparing Apples to Giraffes?

Frequently, the comparisons of interest are binary in nature. In some cases, it is implicit: "does eating apples improve pulse wave velocity?" requires the additional question of "compared to what?" Eating any apples versus no apples? Increasing the number of apples someone consumes? Substituting apples for some other fruit? In each case, however, the assumption is that the study being conducted is testing the desired question, but sometimes additional factors can unwittingly be introduced into a study. Take, for instance, the concept of an attention-placebo. In studies involving lifestyle interventions, in which the researchers have hours of contact time with the participants, it is necessary to be sure that the control subjects are also receiving a similar amount of attention, ostensibly in a way that is not expected to affect the outcome of interest. The attention-placebo is part of a greater concept of making sure the treatment group is as similar as possible to the control group except for the factor of interest. In the present example, if the control group received no attention, then it would be unclear whether having hours of health-related attention would be sufficient to bring about any differences between the treatment and control groups or whether there is something unique about this particular lifestyle intervention. The existence of a factor that is difficult to isolate from another is sometimes referred to as "confounding," though it is important to note that there is also a related statistical concept (when a third factor is correlated with the two factors of interest).

Case Study: Plant-Based Diets Versus Restriction of Dietary Options An RCT was conducted to determine if the degree of plant-based content of the diet influenced weight [17]. The researchers chose five diets: omnivorous, semivegetarian, pesco-vegetarian, vegetarian, or vegan. The researchers concluded that "[v]egan diets may result in greater weight loss than more modest recommendations." However, the researchers neglected to consider another, completely reasonable alternative explanation: the extent to which dietary options are restricted may result in greater weight loss. When looking again at the five diets, as they increase in plant-based content they also decrease in the dietary options available to the participants; stated another way, the dietary option restriction increases concurrent with plant-based content. The two are confounded, and therefore conclusions about the plant-based content cannot be made without also making conclusions about the restriction of dietary options.

Communicating Science

Conclusions Versus Decisions

The concepts of "evidence-based medicine" and "evidence-based policy" revolve around using the best available evidence to inform what decisions should be made with respect to medical care, medical coverage, and policies of all sorts. The narrative often involves discussing the importance of evaluating evidence and letting the science decide what action should be pursued. Although such a narrative represents a lofty and idyllic standard, scientific evidence is designed to make conclusions, not necessarily decisions. Conclusions can often be empirically tested and confirmed, whereas decisions involve value structures and concerns beyond the scientific conclusions [18, 19]. Such differences in value structures have played out, for instance, in nutrition assistance programs in the United States, namely the Special Supplemental Nutrition Program for Women, Infants, and Children program (WIC) and the Supplemental Nutrition Assistance Program (SNAP). In one (WIC), the nutritional support is highly prescriptive, with specific foods being allocated, sometimes including size, type, and

other characteristics. In the other (SNAP), there is more flexibility, and efforts to restrict foods some people consider unhealthy or nonessential have been met with discussions of the importance of autonomy and freedom of choice. The evidence of the health effects may or may not be different between the two populations of beneficiaries, but the policy decisions of what to do with that evidence is quite different.

Additionally, many of the questions asked by science are in the form of $A \rightarrow B$. That is, will lowering food insecurity lead to decreased crime? But, A may also lead to C or to not-B [19]. For example, alerting patients of their weight status (A) was intended to lead to greater awareness of the problem of weight (B) but actually lead to stigmatization of the patients, depression and consumption of more food and avoidance of future doctor's appointments (C) [20]. In another study, the message of eat more fruits and vegetables was placed on "unhealthy" foods (A) in an effort to decrease consumption of the "unhealthy" food in favor of a "healthy" food (B) and as a result of the study, participants consumed more of a test "unhealthy" food (not-B) [21].

It is therefore important to separate the "science" and the "should": just because *science* says that a particular targeted outcome may improve with a certain set of policies or behaviors does not mean those necessarily *should* be implemented if they violate other ethical principles or have other undesirable consequences; the converse is also true.

Letting the Data Speak

Many-a-headline, press release, or study conclusion describe in no uncertain terms the sensational value of the latest research.

Averages Versus Anecdotes

Anecdotes are often buried low on the hierarchy of evidence, but they have a very important place in determining what an individual should do in daily life. For instance, it does not matter what the health benefits of nuts may be if an individual has an allergy: nuts are unhealthy for that person. Such statements may seem obvious, but are often overlooked when making policy or recommendations. Scientific research in most cases provides an estimate of what would be expected to happen within a certain population on average, not for each person. This distinction has become increasingly popular with the advent of "precision" or "personalized" medicine. Thus, conclusions need to be stated with appropriate caveats, including characteristics of the population studied.

Association or Causation

Remember the limitations in how a study was conducted. If a study statistically regresses selfreported intake of a particular food with self-reported weight, then the study only showed an association: people who self-reported eating more of that food also self-reported having a higher weight. This does not show that getting people to eat more of that food will result in them having lower weights for at least two reasons. First, as the maxim says, "correlation does not equal causation." This has been described above and is further addressed below with "Logical Fallacies" (section "Logical Fallacies"). Second, self-report does not necessarily adequately reflect truth; thus the most proximal causal conclusion would be that getting people to *self-report* eating more of that food will result in them *self-reporting* a lower weight. Going further would require extrapolating beyond both the self-reported exposure and outcome.

Extrapolation

Some forms of extrapolation were already discussed (e.g., section "Surrogates: Is the Study Measuring What It Means To?"), where biomarkers or related endpoints (e.g., blood cholesterol or short term energy intake) were assumed to represent a reliable substitute for ultimate endpoints (e.g., cardiovascular events or obesity). In some cases, the error can only be demonstrated through additional experimentation. An acute change in energy intake from a particular stimulus may or may not continue with a chronic stimulus. One study looking at a low-calorie version of a food showed that energy intake decreased acutely; when they looked more chronically, energy intake was not different (meaning total mass of intake increased). Stopping at the acute study would not have revealed this.

Another type of extrapolation has been termed the "monotonic mind" [22]. Monotonic is a term used to describe a trend that continues in one direct; a line would always increase or stay flat. This comes in to play when thinking of foods deemed "good" and "bad." Salt is frequently considered "bad," and in large quantities that is often true; but, just because a lot of salt is bad does not mean that a little salt is bad. Salts are essential minerals, but using the monotonicity argument, if a lot is bad, then a little is bad. Similarly, if a little of a food is good, then a lot of it is better. This played out tragically with the death of a contestant in a water drinking contest from hyponatremia. Despite tragic cases demonstrating the follies of monotonic thinking, studies implicating benefits from drinking water are still paired with headlines like "More you drink water, more dietary benefit you get."

Other types of extrapolation can lead conclusions astray, as well. One particular linear extrapolation is known as the 3500 kcal rule, which assumes that increasing or decreasing one's current energy balance by 3500 kcal will result in 1 lb of weight change. Such thinking ignores the adaptive nature of biological organisms. The errors in this particular extrapolation are much more apparent than some other types because simple arithmetic can demonstrate the absurdity: if a man walked enough daily to burn an additional 100 kcal/d, he would lose a pound in 35 days; in a year, over 10 lb; and, in 10 years, 100 lb. In 20 years, he would likely be a negative weight. Several models have been developed to estimate expected final weight with a sustained change in energy intake (e.g., decreasing consumption of energy from current levels daily) [23, 24]; they each show that weight asymptotes in response to changes in energy demands from the final body mass and composition.

Generalizability can be considered as another form of extrapolation, which involves taking results from one population, model, or setting, and assuming that it will work in others. Assuming animal models will perfectly recapitulate humans, for instance, is unlikely. However, subtler examples also exist. Differences in metabolism among ages, between sexes, and across races make translating results between populations difficult. The use of data in other populations is an important place to start when investigating new populations, but assuming direct translatability is often premature.

Logical Fallacies

Logical fallacies represent flaws in argumentation. When arguments are built on such fallacies, the arguments themselves no longer adequately support the concept being argued for. There are numerous fallacies, with a few addressed below.

Some fallacies come from the study design itself. For example, cross-sectional studies may suffer from a fallacy known as *cum hoc*, *ergo propter hoc* (with this, therefore because of this; sometimes abbreviated *cum hoc* fallacy), in which two things that occur together are assumed to represent a causal relationship. Indeed, if a cross-sectional study shows that A and B are correlated (that is, we see that one factor increases as another factor increases), it is unclear if $A \rightarrow B$, $B \rightarrow A$, or if A and B are caused by something else entirely. Does eating breakfast result in lower obesity, does obesity cause a disinterest in eating breakfast, or do people with obesity not eat breakfast because of other factors (genes, income, time, culture, etc.)? Without knowing which occurs first, it is impossible to tell.

Though as Sir Austin Bradford Hill pointed out, temporal order is necessary to establish causation, it is insufficient. Similar to the *cum hoc* fallacy, *post hoc*, *ergo propter hoc* (after this, therefore because of this; sometimes abbreviated *post hoc* fallacy) says that an earlier observation is the cause of a later one. Such argumentation is common in thinking about health, particularly about obesity. For instance, many arguments about the causes of obesity have used temporal fallacies of this sort to support the argument, whether it be the introduction of a particular food, agricultural practice, or technology around the time the obesity epidemic began to rise in the United States. If this line of reasoning were valid on its own, then all other temporally related factors would be just as plausible, including the invention of Hacky Sacks, food processors, and automated teller machines.

Other fallacies are also evident. For example, a paper studying the amount of sugar in ultraprocessed foods uses circular logic when the addition of sugar itself contributes to the definition of a food as ultra-processed. Raw food, whole food, paleo diets, and other terms draw on the naturalistic fallacy, or an appeal to nature, in which things that are considered "natural" are also considered "good." When a communication concludes that attempting a particular lifestyle change is imperative to combat the sad state of childhood obesity, it is often appealing to emotion and a sense of duty, rather than relying on the strength of the evidence.

False analogies occur when an argument generalizes from similarities. This is similar to the discussion of oversimplification above. For example, lay media reported on a high fat/low carbohydrate diet on weight and glucose tolerance and insulin sensitivity in polygenic obese mice [25]. A press release of the study simplified the topic to one of the experts warning against the paleo diet and lay media reporting paleo diets could lead to weight gain. Although the gross macronutrient content of the diet may reflect some "paleo diets," the content of the diet itself (canola oil, cocoa butter and ghee as sources of fat, sugar as the source of carbohydrate, and casein as the source of protein) does not match what is commonly considered a paleo diet.

The use of false analogies in other settings is used to evoke references to Nazism and Hitler to the point that a philosopher coined the term *reductio ad Hitlerum* or reducing an argument to a distasteful comparison to Hitler [26]. Here, we propose a similar phenomenon in health of comparing foods to tobacco and suggest the fallacy be called *reductio ad tabacum*. Indeed, recent examples have shown comparisons of bacon, sitting, and sugar to tobacco. Frequently, the comparison is used to evoke the same certainty of causation and magnitude of effect of tobacco on lung cancer to a situation involving much less certainty and a much smaller effect size.

Ad hominem (focusing on the originator of an argument) or genetic fallacies (deciding whether to accept an argument because of from whom or where it comes) are also frequently used. This typically comes in the form of giving a pass to academic researchers or government funded research, while attacking industry funded research. The common accusation is that individuals affiliated or funded by a for-profit industry are under undue pressure to obtain results favorable to the organization. Unfortunately, such logic fails to note the pressures within academia to "publish or perish," or to support a professional viewpoint, such as those supported by professional or public health associations. Furthermore, the pressures of the individual or the funding of the study do not address any faults with the data, methods, or conclusions, and are therefore extra-scientific considerations. Indeed, Kesselheim et al. demonstrated that such disclosures are actually bias inducing, with physicians rating identical studies as less rigorous or reliable when the study was affiliated with an industry [27]. The converse can also occur, in which some studies from particular individuals or prestigious institutions may be presumed to be of high quality. Reductio ad tabacum may also be able to be applied here, but instead of comparing the foods and the outcomes, the comparisons are of the industries funding research being comparable to the unethical practices of the tobacco industry in promoting smoking. Though trust in an institution or an individual can be a useful heuristic to evaluate science at a personal level in day-to-day life, it becomes fallacious when used to make conclusions.

The ultimate challenge in evaluating arguments that use fallacies, however, is the "fallacy fallacy" or "argument from fallacy," which is dismissing an argument as false because it contains a fallacy. Instead, the conclusion is much more nuanced: "the argument does not support the contention being argued." This is akin to concluding that a nonsignificant two-group *t*-test provides insufficient evidence of is a difference, rather than concluding that the groups are the same; or a person on trial being found not guilty rather than innocent. Identifying logical fallacies in communicating or conducting research leaves open the door for alternative hypotheses but does not immediately falsify the conclusions of the research. For example, take the case study of SREI in section "Surrogates: Is the Study Measuring What It Means To?". If someone concluded based on SREI that tall people consumed more energy than short people, we can only conclude that those data are unreliable and are not able to support the conclusion. This is not equivalent to saying their conclusions is false: by the laws of thermodynamics (to which biology is beholden), taller people (who tend to have greater mass) likely do consume more to maintain mass than do shorter people. The SREI data simply are unreliable to confirm the contention.

Moving Forward: Avoiding Lemons in Nutrition Science

The scientific literature has been likened to a "market of lemons" [28]: "the scientific literature is mixed, with the reader unable always to distinguish the good articles from the bad" [29]. Putting aside the purported health benefits of lemons and other citrus, "lemon," in this case, refers to the American idiom of a used car passed off as being of much higher quality than it is, with problems arising after purchase and with seemingly no recourse for the buyer. A scientific lemon market would be characterized by an asymmetry of information, in which the reader cannot accurately assess the value of research while the researcher has an incentive to pass of a low-quality study as a higher quality one. It is exacerbated when researchers have no means to disclose credibility, and readers are sufficiently pessimistic about study quality. Furthermore, there are insufficient public quality assurances.

To avoid a nutrition science lemon market requires efforts on the part of all relevant stakeholders, from the researchers, to the knowledge consumers, to those that apply the science. For researchers, complete methodological reporting, stating conclusions within a realistic scope of inference, and limiting simplification can help readers understand exactly what can be gained from a given study. Furthermore, scientists have a vested interest in ensuring that quality information is produced and shared. If the consumers of scientific knowledge (other scientists, the public, policymakers, and others) no longer see a value in it, then the market will collapse. Scientists therefore also have a vested interest in conducting and communicating science in a way that will help assure the long term stability of the knowledge market, which can only occur when credible information is being generated. Knowledge consumers, on the other hand, must first become educated consumers, and we hope the current chapter helps readers expect an appropriate amount of information from a given study and the scientific process in general. After all, researchers can only disclose the credibility of their science if readers know what to look for and how to interpret it. Those who use and apply scientific results, such as clinicians and policymakers, need to accurately communicate decisions versus conclusions. Too often, a reasonable decision is conflated with generalizable science, which not only undermines current and future science that may be contrary to the decision, but also may confuse those who could most benefit from the decisions required for idiosyncratic cases.

On a daily basis, people make decisions, clinicians make judgments, and policymakers make policies from imperfect or incomplete information. But the decision to eat a food, perform a particular exercise, or support a given policy, even on the basis of the best-yet-limited scientific evidence should be seen as a decision and must not be mistaken for a scientific conclusion. Perhaps the most valuable thing an individual can do to better understand nutrition is to more critically evaluate nutrition research, especially research that concurs with one's own proclivities.

References

- 1. Dialektakou KD, Vranas PB. Breakfast skipping and body mass index among adolescents in Greece: whether an association exists depends on how breakfast skipping is defined. J Am Diet Assoc. 2008;108(9):1517–25.
- Brown AW, Bohan Brown MM, Allison DB. Belief beyond the evidence: using the proposed effect of breakfast on obesity to show 2 practices that distort scientific evidence. Am J Clin Nutr. 2013;98(5):1298–308.
- Casazza K, Brown A, Astrup A, Bertz F, Baum C, Brown MB, et al. Weighing the evidence of common beliefs in obesity research. Crit Rev Food Sci Nutr. 2014;55(14):2014–53.
- 4. Gregori D, Maffeis C. Snacking and obesity: urgency of a definition to explore such a relationship. J Am Diet Assoc. 2007;107(4):562; discussion -3.
- 5. Wansink B, Payne CR, Shimizu M. "Is this a meal or snack?" Situational cues that drive perceptions. Appetite. 2010;54(1):214–6.
- 6. Foundation PfBH. Fruits & Veggies More Matters: Produce for Better Health Foundation. 2016. Available from: http://www.fruitsandveggiesmorematters.org/.
- 7. United States Department of Agriculture. Empty calories. Available from: http://fnsweb01.edc.usda.gov/super-tracker-tools/empty-calories-chart.html.
- Parloa M. Canned fruit, preserves, and jellies: United States Department of Agriculture household methods of preparation. Washington: GPO. 1905 https://books.google.com/books?id=88pFAQAAIAAJ
- 9. Academy of Nutrition and Dietetics. Processed foods: what's OK, what to avoid 09 NOV 2015. Available from: http://www.eatright.org/resource/food/nutrition/nutrition-facts-and-food-labels/avoiding-processed-foods.
- Barr SB, Wright JC. Postprandial energy expenditure in whole-food and processed-food meals: implications for daily energy expenditure. Food Nutr Res. 2010;54. doi:10.3402/fnr.v54i0.5144
- 11. Paul LA, Hall EJ. Causation: a user's guide. Oxford: Oxford University Press; 2013.
- 12. Hill AB. The environment and disease: association or causation? Proc R Soc Med. 1965;58:295-300.
- Dhurandhar NV, Schoeller D, Brown AW, Heymsfield SB, Thomas D, Sorensen TI, et al. Energy balance measurement: when something is not better than nothing. Int J Obes. 2015;39(7):1109–13.
- 14. Beaudoin R, Mayer J. Food intakes of obese and non-obese women. J Am Diet Assoc. 1953;29(1):29-33.
- Schoeller DA, Bandini LG, Dietz WH. Inaccuracies in self-reported intake identified by comparison with the doubly labelled water method. Can J Physiol Pharmacol. 1990;68(7):941–9.
- 16. Student. The Lanarkshire milk experiment. Biometrika. 1931;23(3/4):398–406.
- Turner-McGrievy GM, Davidson CR, Wingard EE, Wilcox S, Frongillo EA. Comparative effectiveness of plantbased diets for weight loss: a randomized controlled trial of five different diets. Nutrition (Burbank, Los Angeles County, Calif). 2015;31(2):350–8.
- ten Have M, de Beaufort ID, Teixeira PJ, Mackenbach JP, van der Heide A. Ethics and prevention of overweight and obesity: an inventory. Obes Rev (An Official Journal of the International Association for the Study of Obesity). 2011;12(9):669–79.
- Brown AW, Allison DB. Unintended consequences of obesity-targeted health policy. Virtual Mentor (VM). 2013;15(4):339–46.
- Puhl R, Peterson JL, Luedicke J. Motivating or stigmatizing? Public perceptions of weight-related language used by health providers. Int J Obes. 2012;37:612–9.
- 21. Werle COC, Cuny C. The boomerang effect of mandatory sanitary messages to prevent obesity. Marketing Lett. 2012;23(3):883–91.
- Rozin P, Ashmore M, Markwith M. Lay American conceptions of nutrition: dose insensitivity, categorical thinking, contagion, and the monotonic mind. Health Psychol (Official Journal of the Division of Health Psychology, American Psychological Association). 1996;15(6):438–47.
- Hall KD, Butte NF, Swinburn BA, Chow CC. Dynamics of childhood growth and obesity: development and validation of a quantitative mathematical model. Lancet Diabetes Endocrinol. 2013;1(2):97–105.
- Thomas DM, Martin CK, Heymsfield S, Redman LM, Schoeller DA, Levine JA. A simple model predicting individual weight change in humans. J Biol Dyn. 2011;5(6):579–99.
- 25. Lamont BJ, Waters MF, Andrikopoulos S. A low-carbohydrate high-fat diet increases weight gain and does not improve glucose tolerance, insulin secretion or beta-cell mass in NZO mice. Nutr Diabetes. 2016;6:e194.
- Strauss L. The Social Science of Max Weber. Measure: A Critical Journal. 1951;2(2):204–230. H. Regnery Company https://books.google.com/books?id=6z3WAAAAMAAJ
- Kesselheim AS, Robertson CT, Myers JA, Rose SL, Gillet V, Ross KM, et al. A randomized study of how physicians interpret research funding disclosures. N Engl J Med. 2012;367(12):1119–27.
- 28. Akerlof GA. The market for "lemons": quality, uncertainty and the market mechanism. Q J Econ. 1970;84(3):488–500.
- 29. Cottrell RC. Scientific integrity and the market for lemons. Res Ethics. 2013;10(1):17-28.

Chapter 6 Nutritional Genomics: The Wave of the Future for Nutrition and Dietetics

Judith A. Gilbride and Bridget L. Wardley

Key Points

- Sequencing the human genome created a need to know more about genetics and genomics as it applied to nutritional health and disease.
- Dietitians and nutritionists already practice nutritional genomics for inborn errors of metabolism and other metabolic disorders.
- The increase in genetic information since 2003 is beginning to guide food and nutrition information and offers promise for tailoring diets to individuals in the future.
- Ethical, legal, and social issues (ELSI) of genetics and genomics need to be considered in research and practice.
- Family pedigrees, teamwork, and genetic testing together with clinical evaluation will provide the evidence to alter practice until nutritional genomics research advances our knowledge base.
- Dietetics training needs to strengthen nutritional genomics content throughout all levels of accredited dietetics education and credentialing exams.

Keywords Nutritional genomics • Genetics education • Dietitian/nutritionists • Human Genome Project • Ethical legal and social issues

Introduction

Nutritional genetics and genomics are of interest to health practitioners, physicians, nutrition scientists, dietitians, and nutritionists. Connected to lifestyle medicine, these emerging disciplines are part of a pioneering effort to integrate diverse fields and strengthen the evidence to lower disease risk and promote health for children and adults. Sparked by the mapping of the Human Genome in 2003, the potential of genetics and genomics to personalize and predict nutritional needs and dietary requirements is promising, yet progress is still in its formative stages. The public at large is interested in uncovering definitive predictions of health and disease risk based on genetic profiles. Knowing more

J.A. Gilbride, PhD, RDN, FAND (🖂) • B.L. Wardley, MS, RDN, CSP

Department of Nutrition and Food Studies, New York University,

⁴¹¹ Lafayette Street, 5th Floor, New York, NY 10003, USA e-mail: judith.gilbride@nyu.edu; bridget.wardley@nyu.edu

about genes, genotypes, and phenotypes may help individuals target their own health and disease trajectories. Moreover, increased information and excitement about disease susceptibility by consumers may help them pursue healthier lifestyles.

In this chapter we present some of the history and background of genetics as it relates to nutritional genomics, provide the reader with insight into areas of practice that incorporate nutrigenomics, and highlight the needs for more integration of genetics and genomics into dietetic education at all levels. We do not provide a comprehensive review of the subject but have endeavored to clarify issues and direct the reader to other resources for more in-depth information. Nutritional genomics is an exciting and rapidly developing field that will be the future of nutrition and dietetic practice; exactly when and how this will occur is still to play out!

Background

Watson and Crick discovered DNA in the 1950s and caused a stir about finding the secrets to genetic information [1]. Fifty years later, the completion of the Human Genome Project (HGP) was applauded as the answer to the details of every life process [2].

The human genome is made up of nucleotides (base pairs) within the DNA structure that is organized into 23 chromosome pairs within the cell nucleus. Within the DNA, the bases of adenine, thymine, guanine, and cytosine (abbreviated A, T, G, C) form pairs that become sequences of functional units or genes. A single gene can be a few hundred bases or more than two million bases, and humans have about 25,000 genes establishing the blueprint for the formation of specific proteins. A and T pair up and C and G pair up and the pattern of these pairs of nucleotides is very important in forming the genetic code. Structurally, there are approximately 6 ft of human DNA with close to 3.2 billion base pairs. Genes are expressed and information passed on through a process of transcription and translation to make proteins. Genes only make up part of overall DNA. With the sequencing of the human genome, a realization of the importance of junk DNA has opened new horizons for the understanding of diseases. The various twists and permutations of DNA allow evolution to take place: short term as a life span change and long term by passing from one generation to the next [3].

Genetic variation links us to health and disease. About 99.9% of all base pairs are identical in humans. Single nucleotide polymorphisms (SNPs) are the most common type of genetic variation. Polymorphisms are natural variations in a gene, DNA sequence, or chromosome. An SNP is a variation in a single base pair within a gene. For example, a SNP may replace the nucleotide C with a nucleotide T at a certain position on DNA. SNPs occur at about every 300 base pairs, thus there are almost ten million SNPs, most frequently found in the junk DNA. SNPs act as biological markers, some normal variations, such as eye color, are expressed as phenotypes and some cause disease. SNPs may indicate to scientists the location of genes associated with disease [4]. Single-gene diseases or Mendelian diseases have been studied since the early 1900s, namely as inborn errors of metabolism. Several single-gene defects involve nutritional management. In this century, common chronic diseases are the focus of genetic research, presenting a much more complex genetic picture.

Human Genome Project

Using powerful computer modeling, the first draft of the human genome was published in Science and Nature in 2001 with a detailed visual of the genetic code [5, 6]. Touted as "decoding the book of life," it heralded a new era for detecting and understanding human disease. The question remained—How soon and how much will be learned about "the book of life?" [7].

Great promise was anticipated for this international collaborative effort to decipher the human genome, launched by the National Institute of Health (NIH) and the US Department of Energy in 1990 and involving numerous other groups from around the world. The results of this unique project gave rise to the human genome blueprint being made available via the Internet. Anyone with an Internet connection could view the entire human genome sequence. Prior to the HGP completion scientists thought that there were about 100,000 genes and that "junk DNA" was unimportant in the inheritance of genetic traits and diseases. Finding how genes were expressed was relatively easy but time has shown how much there was to learn. Only about 25,000 genes make up the human genome; repeats in junk DNA are meaningful and help to reveal our genetic potential, especially in relation to health and disease [8]. According to scientists at the time, the benefits of the HGP were likely to be many providing information on human development, insight on genetic conditions, and development of new treatments and diagnostic tools plus the creation of new jobs, services, and medical technology. The sequencing project not only led to new knowledge but also created many more questions than answers and bolstered medical and health research.

Ethical, Legal, and Social Issues

A unique feature of the HGP was the allocation of 3–5% of the US\$3 billion investment set aside for addressing the ethical, legal, and social issues (ELSI) of mapping the genome [9]. This funding also supported education of health professionals and helped augment the services of genetic counselors in the United States. The ethical, social, and psychological concerns generated a tension on the misuse of genetic information to discriminate or stigmatize individuals. Legal concerns arose surrounding job discrimination, privacy and ownership of genetic data, and genetic determinism. Gene patents seemed to advance too quickly for public and political discourse and consensus and impeded research efforts. Gene therapy held promise for the treatment of disease to find cures for conditions such as Alzheimer's, Parkinson's, and Huntington's Diseases. Early gene therapy was not successful due to side effects from treatments and poor outcomes and was discontinued for a brief period. Biologists worried about reduced funding for future programs and the overcommercialization of research to accelerate genetic discoveries [10]. Today gene therapy is being studied in clinical trials and applied to treatment of some rare diseases.

The National Human Genome Research Institute and concerned patients and families dealing with genetic conditions fought hard to pass a bill in Congress to enact the Genetic Information Nondiscrimination Act (GINA). Finally passed in 2008 the bill prohibited discrimination by insurers and employers on the basis of genetic data and protected confidentiality and privacy of genetic information [11]. The question prevailed: Should genetic information be treated differently in health records? Genetic exceptionalism sought to protect and keep private information that identified family relationships, predicted future health events and could be recovered in biological specimens for future research.

Terminology and Definitions

Once the HGP was sequenced, a period of elation and exploration followed and research expanded with the quick discovery of 1800 disease genes in the next few years. Genomics is the study of functions and interactions of all the genes in the genome of a given species [9]. Several new genomic fields emerged—genomic medicine, pharmacogenomics, nutrigenomics, public health genomics, and nutritional epigenomics.

Genomic medicine was meant to predict, prevent, and personalize care and create a better understanding of the relationship between genetic and environmental factors in health and disease. Its use emphasized health maintenance and a willingness to decipher the consequences of a patient's life, health, and longevity by knowing the positive and negative aspects of genetic susceptibility to disease. Genomic medicine also allowed for genetic engineering to advance and establish new diagnostics and therapies for patients.

Pharmacogenomics arose quickly from genomic medicine allowing the production of medications to target specific genotypes. Medications could be formulated for specific genotypes to maximize effectiveness and minimize side effects. Presymptomatic medical therapies could be designed before a disease developed (i.e., antihypertensive agents before hypertension develops). Some obstacles existed for pharmacogenomics—adequate timing for new clinical trials and approvals for patient use, finding mutations that alter drug effectiveness and may limit their use, cost and safety issues for patients, and precise interpretation of gene-drug test data.

Nutritional genomics is a broad term that helps us to recognize the effects of one's genome in nutrient metabolism and how diet alters an individual's genes and health. Nutritional genomics (often shortened to nutrigenomics) was originally defined as the scientific study of the way certain genes interacted with bioactive food components [11]. Knowing more about genes and food may realize "personalized nutrition" that targets treatment interventions for disease, ameliorates and prevents certain conditions and diseases.

Nutritional genomics refers to both nutritional genetics and nutritional genomics with the goal of using diet to prevent or treat disease. Nutritional genetics or nutrigenetics is defined as genetic variations that affect response to nutrients and other dietary components on the health status and risk for disease. Other definitions vary slightly but all include alterations in normal metabolism. Nutrigenomics includes interactions and synergies between dietary components and the genome, and thus, the functional interaction of diet and its components with the genome and its proteomic and metabolomic changes. How do genetic polymorphisms affect nutrient requirements? How does nutrition influence gene expression and metabolic pathways? How is regulation altered or disturbed in diet-related diseases? [10]

According to Stover, nutritional genomics focuses on applying genomic techniques with highthroughput technology and holds more promise for measuring interactions and synergies between dietary components and the human genome [12]. Nutrigenomics has expanded our knowledge through research on polygenic diseases, including obesity, diabetes, cardiovascular disease, cancer, and other complex conditions. As the scientific community learns more, they are also realizing how much more needs to be learned. Nutritional genomics is concerned with using diet to prevent, mitigate, or treat disease [12]. Some common genes have been identified (BRCA1 and BRCA2), the genes for apolipoprotein E (APOE), methyleneterahydrofolate reductase (MTHFR), and diabetes are examples of nutrigenomic tests. As the discipline advances, researchers and nutrition scientists have called for a uniform nomenclature to clarify nutrigenomic definitions and concepts [13].

Epigenetics, first discovered in the 1970s, has grown in importance for comprehending the science of heredity. Epigenetics is the study of heritable changes in gene activity that do not involve the genetic code but are passed to at least one successive generation. The effects of environmental stressors on genes can leave an imprint on genetic material in the developing fetus. Epigenetic marks like diet, stress, and prenatal nutrition can imprint and pass from one generation to the next predisposing children to certain diseases and early mortality [14].

Nutritional epigenomics focuses on changes in gene expression without changing the DNA sequence. It is defined as identifying and understanding the reversible, heritable chemical modifications on chromosomes (DNA methylation and histone acetylation) that alter gene expression and lead to phenotypic variation in health outcomes. Thus, nutritional epigenomics is the influence of diet on changes in gene expression without altering the underlying DNA sequence [15].

After completion of the Human Genome Project, DNA was analyzed with advanced technological methods to scrutinize all the genes, proteins, and metabolites in a genome. Omics means "complete" or "all." Proteomics is the study of a complete collection of proteins in a cell at one time, including both protein-encoding and nonprotein-encoding RNAs. Metabolomics or metabolite profiling is the investigation of metabolic pathways using noninvasive biomarkers and determines how genetic differences influence metaprofiling, metabolic functions, and metabolic response to nutrients [13]. Transcriptomics profiles gene expression and patterns of thousands of genes or an entire genome in a single experiment using microarray analysis. The omics translates genetic information into phenotype within a given environment [16].

Personalized nutrition also emerged as a concept for reaching the public with information that connects diet and genetic makeup to an individual's predisposition for selected monogenic and polygenic diseases. The public wants personalized nutrition along with designer foods and genealtering diets. However, it has become clear that modulation of diet and nutrients/food constituents based on genetics is difficult and some of the original claims for "personalization" have been tempered [17].

Health Professionals and the Genomic Revolution

Path of the Nutrition and Dietetics Profession

Nutritionists and dietitians began to approach genetics in a new way with the hope of identifying genetic profiles of patients and customizing nutrition therapy. DNA testing held the potential of taking the guesswork out of medical nutrition therapy (MNT). Entrepreneurial practitioners promised many answers in 5–10 years but academic researchers warned that it was premature to give science-based nutritional genomics advice without sufficient research verification and agreement by the scientific community. In-depth family histories could elucidate more about disease risk than a battery of genetic tests. Genetic tests provided a percentage risk of developing certain diseases and conditions, but more analysis and interpretation were necessary to help consumers/clients follow prevention regimens and make lifestyle changes. DNA kits collected and analyzed only a small number of individual genetic variants, often not enough to be meaningful to permit individualized treatment.

The study of genetics and diet had traditionally focused on single genetic variations or SNPs (single nucleotide polymorphisms). SNPs were embedded in our understanding of single-gene disorders also known as inherited metabolic diseases or inborn errors of metabolism. Mutations in single genes that caused disease were based on Mendel's law of inheritance and included thousands of diseases. A good reference is the online website of Mendelian Inheritance in Man (www.ncbi.nlm.nih. gov/omim).

Newer concepts began to affect the study of nutrigenomics—finding the function and interactions of all genes in the human genome, the number of SNPs in chronic disease, and genomic technologies applied to nutrition and diet. Nutritional genomics broadened our perspective related to common chronic diseases such as breast cancer, colorectal cancer, diabetes, obesity, cardiovascular disease, and hypertension [18–20]. Gene sequencing revealed that not one or two but a number of genes were related to diseases and that lifestyle factors could alter gene regulation and expression.

Sequencing the genome in humans is beginning to impact nutrition therapy for chronic conditions. With the plethora of new technology and genetic understanding, it is imperative for dietitians to keep abreast of new research that impacts nutritional genomics in their specialty practice area.

Organization of Health Professionals

Approximately 3300 genetic counselors were available and trained to receive referrals and assist patients with genetic testing and to help interpret probability of disease. Genetic counselors had long understood the vital role of family pedigree in interpreting health and disease predisposition. But, with the growing awareness that new genetic technology was developing very quickly, more trained professionals were needed to guide patients on the diagnosis, treatment, and psychosocial implications [21] of genetic tests. Training programs through the ELSI grants were used to prepare health professionals who were not trained in genetics. With the exception of those involved in caring for patients with inherited metabolic diseases, most health providers did not practice with genetics and genomics at the forefront of care and the relevance to their practices and the provision of quality care was not understood.

In 1996, a group started to organize and fill the gap in health provider preparedness for genetics. The organization, partially funded by the National Human Genome Research Institute (NHGRI) of NIH, responded to issues confronting health professionals. Although health professionals were free to provide some genetic services to their clients, they had limited formal education in genetics and were not confident in their ability to continue the provision of genetic services within a rapidly changing research environment [9]. Most of the genetic information was coming from the media and not being taught or monitored within any professional disciplines.

To deal with education and communication issues, the national coalition moved beyond physicians and nurses and attracted over 100 member organizations. Fully structured with bylaws and membership categories, the National Coalition for Health Professional Education in Genetics (NCHPEG) sought to integrate genetics into the knowledge and training of practicing professionals and students and developed a website (www.nchpeg.org) that highlighted tools and resources. The organization expanded and interested individuals, and profit and nonprofit groups, committed to genetics education, joined the coordinated effort and held annual meetings with national and international representatives.

By 2001, core competencies were proposed and vetted by voting members of NCHPEG to increase genetic literacy across the professions. Another major concern was the proliferation of misinformation on the web, and thus, a detailed review process was designed for approving reliable genetic resources and sound curricula. NCHPEG constructed a comprehensive online genetics information center; genetics resources on the web (www.grow.org) formulated a core curricula template for adoption by each health discipline and integrated genetics content in education programs and licensure and certification examinations. At a minimum, NCHPEG's goals were to have professionals appreciate their own extent or limits of genetics expertise, comprehend the social and psychological implications of genetic services, and know when and how to refer to a genetics professional when necessary [22].

The nutrition and dietetics profession became a member of the NCHPEG. Around the same time the Academy of Nutrition and Dietetics (formerly the American Dietetic Association) members encouraged an assertive campaign to advance genetics into dietetics practice. Members with genetics expertise or active with NCHPEG conducted workshops at the national or regional meetings and appealed to the leadership to pave the way for dietetics professionals to become the credible, leading professional relying upon the translation of genetics, health, and nutrition research and technology into clinical practice [11]. Some dietetic practice group members promoted using genotyping to guide nutrition interventions and identifying food components that increase or decrease gene expression.

Nutrition and Dietetics Education

As early as 2002, the accrediting body for dietetics education added a knowledge statement for undergraduate education, "an understanding of human genetics" based on [23] the competency that graduates will have knowledge of genetics. Many educators began to scrutinize their course content and determine how to address the integration of genetics into the dietetics curriculum. Dietetic students routinely study nutrition and food science, physiology, and chemistry. In the new model for establishing an in-depth background in nutrigenomics, they would need training in genetics and molecular biology, informatics, and computational biology. Broadening nutrition and dietetics education should also encompass the effect on nutrient requirements by genetic polymorphisms, the influence of food and nutrition on genetic expression and metabolic pathways, and determining whether gene regulation is disturbed in diet-related diseases. Nutrigenomics could be embedded across the curriculum and help students learn to target nutritional advice not only based on food preferences, lifestyle, diet, and readiness to change but also based on the patient's genetic information [24]. Table 6.1 outlines the suggested content of different levels of dietetic education.

The Human Genome Education Model (HuGEM) Project developed a needs assessment survey, Emerging Issues Confronting Health Professionals. The Academy of Nutrition and Dietetics participated in this multidisciplinary survey of six health professions to determine the genetics education needs and priorities of dietitians, occupational therapists, physical therapists, psychologists, speech–language–hearing specialists, and social workers. After the assessment survey, two additional phases followed: education and training of 60 professionals in a 1-week core course for adding genetics into teaching and practice and regional workshops within each of the six professions. An evaluation of education effectiveness on practicing professionals was the final phase of the 3-year funded project.

Findings from the HuGEM survey of 1958 professionals indicated that some genetics content (14%) was included in their academic preparation. However, over a third (35%) had no formal education and only 14% had a full course in genetics. Close to 70% reported discussing the genetic component of health and disease, 30% did some counseling in genetic concerns, 19% made referrals for genetic counseling, and 16% made referrals for genetic testing. For the 600 nutrition and dietetic respondents, the numbers were similar but lower than the entire group of 1958 on genetic services. Dietitians differed from the other professionals on referrals. They were only 6% who referred for genetic counseling and testing compared to 19% for the total group. The services for genetic and environment factors by dietitians were 8% higher than the entire group (43% vs. 35%). Overall, the major findings of the HuGEM survey found low confidence and low involvement in the provision of genetic services to individuals and families, inadequate knowledge of genetics, and a gap in practice and genetics education [25].

Replication of some of the items on the HuGEM survey would be interesting 15 years later, possibly by the Academy dietetic practice groups who work closely with individuals with genetic conditions and their families. An examination could provide direction for the proposed 2017 revised standards for dietetic education from the Accreditation Council for Education in Nutrition and Dietetics (ACEND), which states, graduates are able to describe basic concepts of nutrigenomics [26].

Vickery and Cotugna conducted a survey of the use of the NCHPEG competencies by practitioners in 2005 [23]. They found that respondents were least likely "to refer patients for genetic counseling to specialists." The lack of referrals may reflect the work environment where nutritionists and dietitians do not counsel on genetics but physicians and genetic specialists do. A questionnaire about nutrigenomics and continuing education needs of dietitians revealed the belief that applying nutrigenomic principles would strengthen practice [27]. They perceived that nutritional genomics advice would affect practice by giving greater individualization of diet prescriptions (84%), realizing a stronger foundation for nutrition recommendations (76%), and allowing diet prescriptions that could help manage and prevent certain diseases (75%).

Whelan et al. conducted a survey in the UK on the involvement, confidence level, and knowledge of dietitians regarding genetics and diet–gene interactions. Using the HuGEM survey for some of the involvement and confidence measures, a national survey was sent to a sample of 600 UK dietitians with a 65 % response rate. Most of the 390 respondents indicated no involvements in genetic activities and a lack of confidence in undertaking the genetic activities. Knowledge was 41 % indicating a low level of knowledge in genetics and diet–gene interactions. The authors concluded a need to improve the parameters measured in this survey [28].

Table 6.1	Nutrition and	dietetics education	on in the	postgenome era
-----------	---------------	---------------------	-----------	----------------

Undergraduate/graduate education
Basic genetics and nutritional genomics concepts
Human genetics and molecular biology
Genetics in physiology and biochemistry
Diet gene interactions in chronic disease progression
Nutrition care process and nutritional genomics
Bioengineering and functional foods in food science
Dietetic internship
Nutrition care process in patient care with genetic conditions
Nutritional genomics and medical nutrition therapy
Disease risk assessment and counseling based on family histories, genetic testing and clinical evaluation
Practical aspects of ethical, legal and social issues in nutritional genomics
Advanced practice or doctoral degrees
Interdisciplinary approaches in genetics and genomics
Nutrigenomics in advanced clinical nutrition practice
Nutritional genomics research methods
Clinical nutrition cases using informatics
Genetic counseling and testing referrals
Genetic testing and management of patient outcomes
Policy and public health implications

In 2007, the Academy of Nutrition and Dietetics was awarded a NCHPEG grant to work toward a web-based genetics curriculum. Funding for the project was limited, so various nutrition professionals from the United States and the UK donated their time to draft, review, rewrite, and critique is four case scenarios, working with a genetics counselor from the NCHPEG staff. The objectives of the scenarios were to assist dietetic students/interns and practicing dietitians in applying nutritional genetics and nutritional genomics in practice. Each case had a unique focus for learning. The cases included an example of a fatty acid oxidation disorder, medium chain acyl-CoA dehydrogenase deficiency (MCADD), where the scenario stressed interpretation of family histories, newborn screening, and inheritance patterns. A celiac disease case focused on genes the environment, genetic variants, susceptibility, and genetic testing. The cystic fibrosis scenario showed the effect of genetic mutations on body systems and growth and the impact of early nutrition therapy.

A variation in the DNA due to an SNP causing less efficient functioning of the enzyme of methylenetetrahydrofolate reductase (MTHFR) is the final case. This example demonstrated the role of MTHFR in common disorders and has been reiterated in many articles and workshops. MTFHR has a role in folate metabolism and regulates usage of one-carbon units between methylation reactions and nucleic acid synthesis [29]. In this case, aspects of personalized nutrition therapy based on genetic diagnosis, family history, and clinical/nutritional evaluation are highlighted. The nutrition case scenarios can be found on the NCHPEG website www.nchpeg.org/nutrition. However, the coalition of health professionals for genetics education was discontinued in 2005 and the website is now maintained by Jackson Labs.

Implications for Nutrition and Dietetic Practice

Consensus of those involved in the early days of genetics education envisioned an opportunity to infuse genetics and genomics into the training of dietetic students and interns. However, a need existed to update current practicing nutrition and dietetics professionals to understand basic

genetics as a foundation for practice. Also important in the ever-changing world of genetic discoveries was a renewed focus on several chronic diseases and conditions. Metabolic dietitians were skilled in managing inborn errors of metabolism in children and could assist in this effort. Critical topics for nutrition practice included genetic testing and counseling, how genetic differences among individuals can alter risk and health outcomes, working with genetics professionals, and the ethical, legal, and social issues. Knowledge of how food and nutrition combine with genes in multifactorial diseases and genotyping to guide food and nutrition selection has become progressively important (see Table 6.2).

Diet is a key environmental component of health and has the potential for achieving better health outcomes with increased genetic data at the population and individual levels. Health care costs may be reduced if nutritional genomics is a low-cost prevention strategy and can show large effects and statistical significance. Its greatest impact could be for high-risk diseases and within population subgroups at greatest risk. Dietary factors may become the most important environmental factor modulating gene expression throughout the lifespan. Genetic testing may become a lifetime investment for each person (done more than once), although a cohesive regulatory strategy is an issue for further research in clinical applicability. GINA, since its passage in 2008, is helping to control fairness in the use of genetic information by insurers, employers, adoption agencies, schools, courts, and in other settings who have access to personal genetic information and how it will be used protecting privacy and confidentiality. Public surveys from the International Food Information Council (IFIC) have shown that consumers want both personalization from genetic testing and privacy of data, but the public debate continues with concerns and confusion about ownership of genetic information for health care and research [30, 31].

A recent study examined consumer perceptions of personalized nutrition (PN) and information provided by genetic tests. A randomized clinical trial of 138 men and women (ages 20–35) was provided with either a DNA-based dietary report or a general report of dietary advice for caffeine, vitamin C, added sugar, and salt. Both groups were surveyed three times at baseline, at 3 months, and at 12 months. The researchers found that perceptions of personalized nutrition changed over the course of the study. Patients preferred to receive PN results from a university research laboratory or a health care professional. Dietitians/nutritionists were considered the best providers of personalized nutrition advice (56%) compared to physicians (27%) and nurses (8%). More studies are needed to clarify the value and method of disclosing genetic-based information along with dietary advice to consumers [32].

 Table 6.2
 What you should know for practice in nutritional genomics

- Basic understanding of genetics (terminology and principles including individual and population inheritance patterns)
- Knowledge of normal metabolism and potential disruptions in metabolism due to genetic disorders of enzymes, transporters, etc.

Nutritional needs and diet modifications of selected traditional genetic conditions such as inborn errors of metabolism and chromosomal defects

Application of family histories/family pedigrees (at least three generations) to interpret genetic predisposition to disease

Understanding of the impact of nutrient-gene interactions on normal and aberrant phenotypes and chronic disease progression

Ability to know where to find up-to-date, sound evidence-based information on nutritional genomics and genetic testing

Appreciation of the ethical, legal, and social issues surrounding genetic testing, privacy protection issues and those affecting one's practice

Integration of dietitian's role/expertise in nutritional genomics with other disciplines and making appropriate referrals for genetic counseling

Genetic Testing as Part of Disease Management

Newborn Screening

Newborn screening (NBS) is a public health initiative that should be considered an early form of genetic testing. Although not developed to look specifically at an individual's genetic sequence, NBS does identify genetic disorders at a very early stage in the newborn infant, mostly through detection of metabolites in the blood. In some instances the initial metabolite screen is supported by genetic mutation analysis. Cystic fibrosis (CF) is an example of this [33, 34].

NBS originated in the 1960s, beginning with the work of Dr. Robert Guthrie, who developed a test to detect phenylketonuria (PKU). He was commissioned to devise a test shortly after the discovery that PKU as a genetic condition, if untreated, causes mental retardation and developmental delays. PKU could be treated by diet, which if initiated early in life prevented these consequences. The early dietary experiment establishing the success of the diet is documented in a historic video record [35]. The history of PKU and the development of newborn screening are reported in the literature [36–38].

Newborn screening gradually expanded into every state. The simple blood collection method developed by Dr. Guthrie is still in use today. Blood is collected from the infant using a minimally invasive heel stick, dropped onto a filter paper, and then mailed to the state's screening laboratory for testing. Guthrie's method involved bacterial inhibition testing, allowing only one disease to be tested per individual blood spot and thus limited the number of diseases that could be tested. Newer methods for testing and more conditions were later developed. With the advent of tandem mass spectrometry (MS/MS) where multiple metabolites can be examined on one single blood spot, NBS was capable of screening for many conditions.

By 2005, the American College of Medical Genetics (ACMG) released a report calling for all states to adopt a core screening panel of 29 primary disorders and 25 additional secondary disorders that would be detected incidentally when screening for the core disorders. In 2008, the Newborn Screening Saves Lives Act was enacted and brought expansion and standardization of NBS as well as requiring education and coordination of follow-up care. Today, all states have a NBS laboratory and screen for a minimum 29 core conditions. Many of the conditions are single-gene defects requiring dietary management as a significant or sole therapy. If a screening test is positive, the infant is referred to a genetic center where confirmatory testing will take place. When a diagnosis is confirmed and diet is required it will be initiated immediately. Not all positive newborn screens result in a diagnosis. The history of NBS and discussion on future possibilities are described in a publication from the Association of Public Health Laboratories [39] (see Table 6.3). Conditions screened on a NBS panel that require nutritional therapy include PKU, maple syrup urine disease (MSUD), homocystinuria, some urea cycle disorders, fatty acid oxidation disorders, galactosemia, and cystic fibrosis.

Genetic Metabolic Dietitians

The dietitian's involvement in the care of nutritionally managed single-gene defects can provide all practitioners with insight into some aspects of nutrigenomics practice. Dietitians working in the field of genetic metabolic dietetics (inborn errors of metabolism) have been using genetic information to direct nutritional care for many years. The genetic disorders, where most of the experience has accrued are single-gene defects (Mendelian disorders), many inherited through an autosomal recessive pattern of inheritance. Single-gene defects refers to conditions involving mutations in the DNA sequence of genes, resulting in an alteration or deficiency of a protein such as an enzyme involved in the

Table	6.3	Resources
-------	-----	-----------

Resource and web addresses	Comments
Online Mendelian Inheritance in Man www.ncbi.nlm.nih.gov/omim	Catalogue of human genes and genetic disorders and traits. Most current and updated information on known human genetic variation and expression
National Human Genome Research Institute www.genome.gov	Offers many thorough resources on human genetics fo students, health professionals, patients, and educators as well as information on research, issues, and news in the field
GeneTests www.genetests.org	Useful resource for information on genetic testing. Search by specific disorder, gene, test or laboratory and find clinics
Centers for Disease Control and Prevention (CDC) Office of Public Health Genomics: Training Programs and courses http://www.cdc.gov/genomics/training/	Listings of useful courses and training programs, CDC supported and others
US National Library of Medicine, Genetics Home Reference. http://ghr.nlm.nih.gov/	An excellent resource which includes a handbook of basic information about genetics with links to online resources. Also includes sections on: definitions of genetic terms; descriptions of genetic conditions grouped alphabetically and also by category (such as food, nutrition, and metabolism) providing an overview of the condition and links to further references and resources; information about newborn screening; what's new and in the spotlight in genetics
The New York—Mid-Atlantic Consortium for Genetic and Newborn Screening Services. Understanding genetics: The New York—Mid-Atlantic guide for patients and health care professionals. 2009. http://www.ncbi.nlm.nih.gov/books/NBK115563/	Printed booklet (can be downloaded as a pdf free of charge). Includes information on diagnosis of genetic disorders and genetic testing, taking a family pedigree, newborn screening, genetic counseling, indications for genetic referrals, ethicat legal, and social issues. In addition includes some patient stories as examples
NCHPEG http://www.nchpeg.org/nutrition/	Site written specifically for dietitians. Includes overview of genetic terminology and definitions. Several case study examples to illustrate genetics practice relating to nutrition, includes aspects of taking a family history and using genetics with clinical information and family history to direct nutritional management
Genetic Metabolic Dietitians International (GMDI) http://www.gmdi.org/	Provide standards of excellence and leadership in nutrition therapy for genetic metabolic disorders through clinical practice, education, advocacy, and research. Excellent resources for clinicians on nutritional management of inborn errors of metabolism, contacts to other dietitians working in this area of practice, offers networking and peer support. Also organizes regular educational conferences
State Newborn Screening Laboratory	Each state has at least one designated newborn screening laboratory. Their website provides valuable resources for clinicians and families. Contacts for referral clinics in the state with experienced health care practitioners can be found at these sites. Good local resource and starting point for clinical information and local contacts

Table 6.3 (continued)

Resource and web addresses	Comments
American College of Medical Genetics (ACMG) ACT sheets and algorithms http://www.ncbi.nlm.nih.gov/books/NBK55832/	These algorithms and information sheets describe actions required to communicate positive NBS tests with families and the steps in follow up required to confirm diagnosis. Links to further information and resources are also included. The site is maintained and updated as new information becomes available
Academy of Nutrition and Dietetics http://www.eatright.org/	 Available position paper and resources on nutrigenomics. A series of articles published in the Academy journal between 2005 and 2009 Position statement: Position of the Academy of Nutrition and Dietetics: Nutritional Genomics. J Acad Nutr Diet. 2014;114:299–312
Journal of Nutrigenetics and Nutrigenomics http://www.karger.com/Journal/Home/232009	International journal focusing on research and education in nutrigenetics and nutrigenomics since 2007. Endorsed by the "International Society of Nutrigenetics/Nutrigenomics" (ISNN)

metabolism of amino acids, carbohydrates, fats, vitamins or minerals or disrupting a transporter function at the gastrointestinal or cellular level or impairing another functioning protein. Cystic fibrosis, for example, is a disruption of transmembrane conductance regulator (CTFR) that is responsible for movement of chloride ions in and out of cells [33, 34].

Dietitians also work closely with patients with chromosomal disorders. For example, Prader–Willi syndrome (PWS) is a chromosomal disorder affecting genes on chromosome 15. Knowledge of the genetic diagnosis directs the dietitian to nutritional interventions specific for PWS. Nutrition interventions support the underweight, hypotonia, and feeding difficulties in infancy and later dietary regimens are adjusted in anticipation of later childhood hyperphagia and obesity.

Genetic metabolic dietitians are advanced practitioners, whose responsibilities include developing and implementing dietary treatments for a range of genetic conditions with variable phenotypes. The dietitian develops and implements dietary treatments based on the genetic diagnosis to prevent or minimize adverse health outcomes such as severe cognitive impairment or developmental delays and other significant medical problems. In many instances diet is the primary treatment option, in others it is an adjunct to pharmacological therapy and in others it is an intervention to optimize nutrition where there is a physical inability to eat, for example, tube feeding. For most patients diet therapy is required for life and the dietitian plays a critical role in giving care along with other members of the pediatric genetics team, including physicians, genetic counselors, social workers, and biochemists. Working on the team, the dietitian uses knowledge of the genetic defect, with an understanding of the metabolic pathway affected, and the impact on nutrition to develop individual medical nutrition therapy (MNT) for the affected patient. In addition, consideration of the overall nutritional requirements for that individual is necessary based on age, growth, and daily activity. The nutrition care plan is then developed taking into account food availability, food preferences, cultural or religious beliefs as well as family socioeconomic circumstances [40, 41].

PKU, a Single-Gene Condition

The management principles of inborn errors of metabolism are outlined below using PKU as an example. Phenylketonuria (PKU) is an autosomal inherited disorder of metabolism of the amino acid phenylalanine. Its incidence is approximately 1:10,000 live births, however, this varies by population.

PKU is a condition on the NBS in all states in the United States and in many countries around the world. Upon indication of a positive screen the baby is referred to a genetic center where further blood amino acid analysis confirms the high level of phenylalanine. Genetic mutation analysis is undertaken to confirm diagnosis and sometimes also for research purposes to further understand the genetics of this condition. The family sees all members of the genetic team. A genetic counselor will take a detailed generational family history, although no other relatives may be identified as affected due to the autosomal nature of inheritance in PKU.

The main principles of managing inborn errors of amino acid metabolism are (1) to reduce blood levels of toxic metabolites by reducing intake of nutrients that promote their production due to the inherited enzyme defect and (2) to provide deficient products (nutrients) created by the block in this metabolic pathway through supplementation. In some, particularly where there is residual enzyme activity, provision of a nutritional "cofactor" such as a vitamin is also important. In all cases individualization is necessary due to the variability in metabolic tolerance between individuals with the same condition [40]. For an overview and review of other inborn errors of protein, fat, or carbohydrate metabolism refer to *nutrition management of patients with inherited metabolic disorders* [41]. Upon diagnosis the dietitian will take a full detailed nutrition assessment, including a feeding history, assess the usual family eating pattern and foods, and then develop an initial feeding plan. The goals for PKU are to provide a diet limited in phenylalanine to lower blood levels, replacing tyrosine (the deficient product of metabolism and now a conditionally essential nutrient) thus maintaining blood phenylalanine levels within a treatment range (slightly above normal physiologic levels to ensure no deficiency), while ensuring overall nutrient needs are met for growth and development [42, 43].

In the PKU infant, the diet is composed of a special formula free from phenylalanine, high in tyrosine, and containing a balance of other essential and nonessential amino acids to meet protein needs. The formula also contains fat, carbohydrates, vitamins, and minerals. Phenylalanine is an essential amino acid and therefore a small amount must be provided to meet essential needs; in the infant this is provided as breast milk or infant formula.

In older children and adults, the diet is similar with utilization of a phenylalanine-free supplemental amino acid based formula, specialized foods free from protein and small amounts of measured low protein foods to provide essential phenylalanine. Diet treatment is lifelong and adjustments are made based on knowledge of diagnosis, clinical evaluation including levels of amino acids in the blood, dietary intake, and needs for growth. Dietary advice will change as a child gets older, types of foods and formulas may be adjusted and nutrient needs will vary. Particular attention is required before and during pregnancy when the fetus is at risk of damage if phenylalanine levels are high [42, 43]. A toolkit for practical application was developed by Genetic Metabolic Dietitians International (GMDI) in 2016 (refer to www.gmdi.org).

Direct to Consumer Genetic Testing

The era of genetic testing started in 2003 with one company reporting thousands of multigenetic tests available directly to consumers (DTC). DTC testing without interpretation from a qualified medical geneticist or genetic counselor was questioned. Most DTC DNA tests analyzed only 4–19 genes and included some enquiries on sex, age, weight, and lifestyle. A comprehensive family history was not required and US Food and Drug Administration (FDA) stopped DTC tests for further review.

Besides a lack of FDA review, concerns expressed in overseeing DTC testing were clinical validity and utility, and lack of privacy and research protection for consumers. State protections were thought to be inadequate and federal regulations often did not apply. Moreover, the GINA advocates wondered whether there was sufficient knowledge about genetic risks and whether correct interpretations could be made among both consumers and health care providers. There is an increase in the amount of genetic data available to consumers today and it will probably double with actions taken by the FDA to allow limited DTC tests that became effective in 2015. Regulators have determined that "one-size-fits-all" rules are not appropriate for genetic tests, raising issues for nutrigenomic testing [44, 45]. Genetic tests from state and federal newborn screening are not part of DTC testing and remain a separate issue.

Genetic tests will proliferate due to new FDA guidelines. Although the first tests had covered up to 250 health issues, a recent release was narrowed to 36 hereditary diseases in order to obtain FDA approval. Also included in the genetic profile were some personal traits, ancestry, and "general wellness information" like sensitivity to caffeine, sugar, and salt. When making decisions about genetic tests, one should consider the potential outcome. Is the purpose to compare or validate a family history for a specific disease, help with decisions on cancer treatment, or begin a health and disease record for a family member? Information on risk is provided to consumers but often is not explained fully because a health professional is not involved or there may not be a treatment available for some conditions. Often a consumer learns more from a comprehensive family history that reveals environmental or behavioral risk factors than a genetic test for predicting disease risk.

DTC tests are considered safe for genealogical or paternity testing but implications for health and disease should be examined carefully and used cautiously. On the positive side, if consumers know more about their disease susceptibility, they may seek health care and screen for at-risk conditions more regularly. They could even alter their lifestyle habits including diet and use data to make reproductive decisions.

The federal government has established guidelines on the marketing and advertising of DTC genetic tests by prohibiting some types of marketing and permitting the sale of low risk tests and certain advertising claims. The FDA is enforcing truth-in-advertising and encouraging education and accountability by the industry. State laws may further restrict DTC requiring approval by a physician or medical practitioner, especially for predictive genetic test.

A variety of different types of tests exist on the market. The American Society of Human Genetics (ASHG) has taken the stand that a one-size-fits-all approach does not work for DTC tests. ASHG is concerned that predicted risk may occur in the future, risks may be passed to progeny, and consumers need help in therapy selection similar to what is done in pharmacogenomics. Often it is more difficult to guide "lifestyle" choices such as diet because accurate measurement of food intake is often not possible [45].

Academy of Nutrition and Dietetics Position

The Academy of Nutrition and Dietetics raised a number of issues on commercialization of DTC testing in a 2014 position paper, especially related to quality control and clinical utility that could undermine public confidence [44]. What if consumers question the quality of the test or insurers ponder the validity of certain measures? Personalized nutrition and preventative medicine could be in jeopardy. The collection of biological samples also triggered privacy and discrimination issues although GINA, the nondiscrimination act, helps protect the unauthorized use of personal genetic information.

Cautions for DTC tests were the analytic validity and clinical utility with certain dilemmas. A number of questions arose. What are the genetic marker(s) and are there multiple tests of different markers? Are they patients? Are risks increased? Will the test affect a different outcome for the patient? Three criteria are applied for effective testing: analytical validity, clinical validity, and clinical utility. Analytical validity accurately measures a specific genetic variation. Clinical validity accurately predicts the presence or absence of a disease or particular phenotype. Clinical utility is the highest measure—Will the test improve patient outcomes?

A position paper of the Academy offers exploration of analytical and clinical validity and utility and provides examples based on CDC evidence-based recommendations for genomic tests. The position statement of the Academy states: "It is the position of the Academy of Nutrition and Dietetics that nutritional genomics provides insight onto how diet and genotype interactions affect phenotype. The practical application of nutritional genomics for complex chronic disease is an emerging science and the use of genetic testing to provide dietary advice is not ready for routine dietetics practice. Registered dietitian nutritionists need basic competency in genetics as a foundation for understanding nutritional genomics; proficiency requires advanced knowledge and skills." [44, p. 299]

Other major points in the position paper encourage funding access and guidance to geneticists and genetic counselors. The authors caution that genetic mutations are only part of predicting disease risk. We must use other tools or pave a new road: find empowerment with family histories/pedigrees, biochemical markers, known and suspected risk factors, and other tests and careful monitoring of patients similar to what happens in the management of inborn errors of metabolism [44].

According to the position paper, reinforcing education is a priority for practicing and future dietitians that a basic competency in genetics builds a structure for understanding nutrigenomics. To be proficient, advanced knowledge and skills are recommended to strengthen the application of a diet prescription based on a person's genotype. New studies are still appearing and practitioners should monitor effective approaches to nutrigenomics and patient outcomes. Ethical guidelines for genetic testing and research and evidence-based decision-making will produce an era for helping patients and consumers [45].

Training and time are required of professionals who counsel inpatients and outpatients about gene diet associations. Health care professionals trained in the diagnosis and treatment model may not be prepared to address personal genome data or nutritional genomics and lifestyle modifications to decrease risk and enhance health. Some companies are hiring dietetic professionals or genetic counselors to guide communication and education but companies should also help with the following: the interpretation of the test; fully disclose what the genetic test reveals to the consumer; clearly state the extent of scientific evidence for the test; use an accredited laboratory; and adopt measures to protect consumer privacy.

Nutritional Genomics Research

Nutritional genomics research is important for understanding the health status and risk of developing chronic disease and to advance the art and science of dietetics [45]. Education of current and future practitioners can help promote nutritional health and well-being of the population through incorporation of nutritional genomics into nutrition and dietetic practice. Dietetic practitioners must keep upto-date on research studies that could apply to their patients and clients and counsel responsibly and ethically [44]. To help consumers, they should monitor the potential benefits and safety of nutritional genomic tests for analytical validity and clinical validity and utility [13].

Advances in genomic and epigenetic research will have an impact on how dietary interventions are implemented, especially modification of gene expression to optimize health and reduce disease risk. Dietitians should be involved in conducting and interpreting basic and translational research and become knowledgeable about emerging research on food/nutrient–gene interactions and diet-gene and lifestyle modifications that will promote health and decrease risk of disease (see Table 6.3).

The future of personalized nutrition will derive from nutritional genomics research. It is complicated by the intricacies of diet and the intricacies of genes and the conflicting reports in the literature [47]. Early approaches have been thwarted using a reductionist approach deciphering the interactions between individual genotypes and disease biomarkers and how they are altered by particular foods or isolated components of the diet [47]. Often, there is not enough dietary information to isolate the potentially hundreds of "at-risk" gene variants in a very complex diet.

For advances in tailoring diets for individuals, new dietary strategies may need to be applied including a predetermination of genotype before studies begin. Retrospective analyses have been the norm in the rapidly changing environment that combine genotyping with large data sets from dietary studies including the Framingham Heart Study, the Nurses' Health Study, the Health Professionals Follow-up Study, and the European Prospective Investigation into Cancer (EPIC) and Nutrition Study [13]. For nutritional genomics to become useful in public health, bioinformatics and mathematical tools will need to be applied to models that combine the influence of multiple gene variations on health outcomes [47].

The influence on phenotype by genetic makeup is affected by various environmental factors, including diet as one of the central factors impacting health status [46]. A great deal of research in nutritional genomics over the past 20 years has focused on cardiovascular disease and the APOE genotype with inconsistent results. Although some mechanisms in the cause of cardiovascular disease have been identified, early detection and likelihood of disease risk are not sufficient to tailor diet recommendations to individuals and subgroups in the population with confidence [47]. Segregation analysis, linkage studies in families candidate gene approaches have been applied to see the contribution of multigenic diseases and to find effective preventive strategies and therapies [15]. Genome wide association studies (GWAS) have been used for analyzing disease susceptibility with success. Many gene loci have confirmed genes for chronic disease especially obesity, diabetes, and certain types of cancer but more needs to be done [47].

Applications for counseling, development of new software, expanded food composition tables with bioactive food components, and food marketed for specific genotypes are all predicted to be available to consumers and professionals in the next decade. Eventually dietitians will be in a position to help clients sort out appropriate food and diet choices based on sound genomic science. Today nutrition and dietetic practitioners must rely on their proficiency in assessing and quantifying dietary intakes and combining generational family histories into clinical practice [48]. As the science accumulates, several initiatives should be put in place: a framework for training undergraduate and graduate students, examination of competencies in nutritional genomics, participation in policy debates on genetic testing, and capitalizing on nutritional genomics opportunities.

Conclusion

The knowledge base in nutritional genomics is growing exponentially fueled by research advances in the public and private sectors. Practitioners will need to keep abreast of rapidly changing information and be prepared to interpret conflicting study results. Practice guidelines and solid evidence may not yet be available or agreed upon by professional societies and some uncertainty and practice dilemmas may occur. Thus, continuing education will need to be constant. Shared clinical practice networks, interdisciplinary teams, and collaborations will become crucial as new research is disseminated and deliberated. Long-standing approaches in nutrition and dietetic practice will evolve and allow the vision of nutritional genomics expertise to become the reality.

References

- Crick FHC. Special report: reflections on solving the DNA puzzle. In: Bernstein E, editor. Encyclopedia Britannica Medical and Health Annual 1994. Chicago: Encyclopedia Britannica, Inc; 1994. p. 305–8.
- Gilbride JA, Camp K. Preparation and needs for genetics education in dietetics. Top Clin Nutr. 2004;19(4):316–23.
- 3. Briere T. Nutrigenetics: the interplay of genes, diet and health. Nutrition dimension. Spring. 2015:20-5.
- 4. Shendure J, Akey JM. The origins, determinants and consequences of human mutations. Science. 2015;349(6255):1478.
- 5. The human genome. Science. 2001;291(5507):1145-434.
- 6. The human genome. Nature. 2001;409:745–964.

- 6 Nutritional Genomics: The Wave of the Future for Nutrition and Dietetics
- 7. Kay EL. Who wrote the book of life? A history of the genetic code. Stanford: Stanford University Press; 2000.
- 8. Barnes S. Nutritional genomics, polyphenols, diets, and their impact on dietetics. J Am Diet Assoc. 2008;108(11):1888–95.
- 9. Kozma C. The interface between genomics and nutrition. Top Clin Nutr. 2003;18(2):73-80.
- 10. DeBusk RM. Genetics: the nutrition connection. Chicago: The American Dietetic Association; 2003.
- DeBusk R, Fogarty C, Ordovas J, Kornman K. Nutritional genomics in practice: where do we begin? J Am Diet Assoc. 2005;105(4):589–98.
- Stover PJ, Caudill MA. Genetic and epigenetic contributions to human health: managing genome-diet interactions. J Am Diet Assoc. 2008;108(9):1480–7.
- 13. Subbiah MTR. Understanding the nutrigenomic definitions and concepts at the food-genome junction. OMICS. 2008;12(4):229–35.
- 14. Kauwell G. Epigenetics: what is it and how can it affect dietetics practice. J Am Diet Assoc. 2008;108(6):1056-9.
- West AA, Caudill MA. Applied choline-omics: lessons from human metabolic studies for the integration of genomics research into nutrition practice. J Acad Nutr Diet. 2014;114(8):1242–50.
- Afman L, Muller M. Nutrigenomics: from molecular nutrition to prevention of disease. J Am Diet Assoc. 2006;106(4):569–76.
- 17. Mutch D, Wahli W, Williamson G. Nutrigenomics and nutrigenetics: the emerging faces of nutrition. FASEB J. 2005;19(12):1602–16.
- 18. Qi L. Personalized nutrition and obesity. Ann Med. 2014;46:247-52.
- 19. Ferguson LR. Dissection the nutrigenomics, diabetes, and gastrointestinal disease interface: from risk assessment to health intervention. OMICS. 2008;12(4):237–44.
- 20. Ziesel SH. Nutrigenetics, plasma lipids, and cardiovascular risk. J Am Diet Assoc. 2006;106(7):1074-81.
- 21. Holtzman NA, Watson MS, editors. Promoting safe and effective genetic testing in the United States: final report of the Task Force on Genetic Testing. Bethesda: NIH-DOE Working Group on the Ethical, Legal and Social Implications of Human Genome Research; 1997.
- 22. Core Competencies in Genetics Essential for All Health Care Professionals. Lutherville: National Coalition for Health Professional Education in Genetics; 2001.
- 23. Vickery C, Cotungna N. Incorporating human genetics into dietetics curricula remains a challenge. J Am Diet Assoc. 2005;105(4):583–8.
- 24. Kauwell GPA. A genomic approach to dietetic practice: are you ready? Top Clin Nutr. 2003;18:81–91.
- Lapham EV, Kozwa C. Weiss JO, Benkendorf JL, Wilson MA. The gap between practice and genetics education of health professionals: HuGEM survey results. Genet Med. 2000;2(4):226–31.
- 26. ACEND Accreditation Standards for Nutrition and Dietetics Didactic Programs (DPD). Chicago: Academy of Nutrition and Dietetics; 2016.
- Rosen R, Earthman C, Marquart L, Reicks M. Continuing education needs of registered dietitians regarding nutrigenomics. J Am Diet Assoc. 2006;106(8):1242–5.
- Whelan K, McCarthy S, Pufulete M. Genetics and diet-gene interactions: involvement confidence and knowledge of dietitians. Br J Nutr. 2008;99:23–8.
- 29. Trujillo E, Davis C, Milner J. Nutrigenomics, proteomics, metabolomics, and the practice of dietetics. J Am Diet Assoc. 2006;106(3):403–13.
- IFIC Foundation. Food and Health Survey: consumer attitudes toward food, nutrition and health. 2005. http://www. foodinsight.org/Resources/Detail.aspx?topic+2008. Food health survey consumer attitudes toward food health nutrition.
- 31. IFIC Foundation. Food and Health Survey: consumer attitudes toward food, nutrition and health. 2007. http://www. foodinsight.org/Resources/Detail.aspx?topic+2008. Food health survey consumer attitudes toward food health nutrition
- Nielsen DE, Shih S, El-Sohemy A. Perceptions of genetic testing for personalized nutrition: a randomized trial of DNA-based dietary advice. J Nutrigenet Nutrigenomics. 2014;7:94–104.
- 33. Rock MJ. Newborn screening for cystic fibrosis. Clin Chest Med. 2007;28:297-305.
- 34. Wagener JS, Zemanick ET, Sontag MK. Newborn screening for cystic fibrosis. Curr Opin Pediatr. 2012;24(3):329–35.
- 35. Discovery of the diet for PKU by Dr Horst Bickel. https://www.youtube.com/watch?v=-rs0iZW0Lb0xxx. Accessed 31 Mar 2016.
- 36. Howell RR. Fifty years of newborn screening. Mol Genet Metab. 2014;113(1-2):4-5.
- 37. Dhondt JL. Neonatal screening: from the 'Guthrie age' to the 'genetic age'. J Inherit Metab Dis. 2007;30(4):418-22.
- Frazier DM. Tandem mass spectrometry newborn screening and its impact on inborn errors of metabolism. Top Clin Nutr. 2009;24(4):275–88.
- Association of Public Health Laboratories (APHL). http://www.aphl.org/AboutAPHL/publications/Documents/ NBS_2013May_The-Newborn-Screening-Story_How-One-Simple-Test-Changed-Lives-Science-and-Health-in-America.pdf.

- Boyer SW, Barclay LJ, Burrage LC. Inherited metabolic disorders: aspects of chronic nutrition management. Nutr Clin Pract. 2015;30(4):502–10.
- 41. Acosta PB. Nutrition management of patients with inherited metabolic disorders. Sudbury: Jones and Bartlett Publishers, LLC; 2010.
- Vockley J, Andersson HC, Antshel KM, Braverman NE, Burton BK, Frazier DM, et al. Phenylalanine hydroxylase deficiency: diagnosis and management guideline. Genet Med. 2014;16(2):188–200.
- 43. Singh RH, Rohr F, Frazier D, Cunningham A, Mofidi S, Ogata B, et al. Recommendations for the nutrition management of phenylalanine hydroxylase deficiency. Genet Med. 2014;16(2):121–31.
- Camp KM, Trujillo E. Position of the academy of nutrition and dietetics: nutritional genomics. J Acad Nutr Diet. 2014;114(2):299–312.
- Ries NM, Castle D. Nutrigenomics and ethics interface: direct-to-consumer services and commercial aspects. OMICS. 2008;12(4):245–50.
- 46. Zeisel SH, Waterland RA, Ordovás JM, Muoio DM, Jia W, Fodor A. Highlights of the 2012 research workshop: using nutrigenomics and metabolomics in clinical nutrition research. JPEN J Parenter Enteral Nutr. 2013;37(2):190–200.
- 47. Rimbach G, Minihane AM. Nutrition society silver medal lecture, nutrigenetics and personalized nutrition: how far have we come progressed and are we likely to get there? Proc Nutr Soc. 2009;68:162–72.
- 48. Bennett RL, French KS, Resta RG, Doyle DL. Standardized human pedigree nomenclature: update and assessment of the recommendations of the national society of genetic counselors. J Genet Counsel. 2008;17:424–33.

Part II Nutrition in Prevention and Treatment of Metabolic Diseases

Chapter 7 The Role of Nutrition and Lifestyle in the Prevention and Treatment of Cardiovascular Disease

James M. Rippe and Theodore J. Angelopoulos

Key Points

- Cardiovascular disease (CVD) is the largest source of mortality in the United States and other developed countries and results in 37% of all mortality annually in the United States.
- Positive lifestyle measures, in general, and positive nutritional practices, in particular, play a pivotal role in lowering the risk for cardiovascular disease.
- Multiple guidelines from the American Heart Association (AHA), Dietary Guidelines for Americans (DGA 2010 and 2015), and others have consistently recommended a diet with increased fruits, vegetables, nonfat dairy seafood, legumes, and nuts as consistent with a heart healthy diet.
- Nutritional practices must be placed in the context of an overall positive approach to lifestyle measures including maintaining a proper weight, not smoking cigarettes, and engaging in regular physical activity.
- Despite many years of the guidelines, Americans still fall very short. A key for action moving forward will be implementation of the knowledge that already exists in heart healthy diets.

Keywords Cardiovascular disease • DASH diet • Fruits and vegetables • Whole grains • Fish

Introduction

There is no longer any serious doubt that daily lifestyle practices and habits exert profound effects on the likelihood of developing cardiovascular disease (CVD) [1]. While many different lifestyle practices have been demonstrated to yield positive benefits, both for the prevention and treatment of

J.M. Rippe, MD (🖂)

T.J. Angelopoulos, Ph.D. School of Health Sciences, Emory & Henry College, Emory, VA 24327, USA e-mail: tangelopoulos@ehc.edu

Rippe Lifestyle Institute, 21 North Quinsigamond Avenue, Shrewsbury, MA 01545, USA e-mail: bgrady@rippelifestyle.com

cardiovascular disease, nutrition clearly plays a pivotal role [2–5]. Epidemiologic studies have demonstrated that a diet containing more fish, whole grains, fruits, and vegetables and also the caloric balance to maintain a healthy weight as well as other positive lifestyle decisions such as not smoking and engaging in at least 30 min of physical activity per day can reduce the incidence of coronary heart disease (CHD) by over 80% and diabetes by over 90% in both men and women [6, 7].

Between 1980 and 2000, mortality rates due to CHD fell by 40% [8]. Approximately half of the reduction in CVD mortality was attributed to the impact of major lifestyle-related risk factors such as increased physical activity, smoking cessation, and better control of cholesterol and blood pressure (BP). Unfortunately, increases in obesity and diabetes have moved in the opposite direction and threaten to wipe out all the gains achieved by other lifestyle-related risk factors unless these negative trends can be reversed. Clearly, nutritional factors play a role in many of the components of either positive or negative lifestyle decisions and practices.

While the major focus of this chapter will be on nutrition, we will place nutrition in a larger lifestyle context with particular emphasis on physical activity as well as energy balance and weight maintenance.

This broader approach is consistent with the nutritional guidelines offered by the American Heart Association (AHA) and also many other evidence-based documents, including the 2013 AHA/ American College of Cardiology (ACC) Guidelines on Lifestyle Management to Reduce Cardiovascular Risk [9] the AHA 2020 Strategic Plan for Improving Cardiovascular Health and Lowering Cardiovascular Risk [3], as well as the Dietary Guidelines Advisory Committee 2015 (DGAC 2015) Report [10]. We will also place emphasis on frameworks and practical strategies for implementing nutritional guidelines to improve cardiovascular health.

Background

Consensus Statements

A variety of consensus statements have been issued over the past 10 years regarding nutritional strategies for improving cardiovascular health. These statements have been very similar in terms of recommendations and often have drawn upon the same database and referred to each other. These statements have formed the basis of recommendations made in this chapter. The statements are given below:

- Diet and Lifestyle recommendations Revision 2006: A Scientific Statement form the American Heart Association Nutrition Committee [2]
- Defining and Setting National Goals for Cardiovascular Health Promotion and Disease Reduction: The American Heart Association's Strategic Impact Goal through 2020 and Beyond [3]
- 2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk: A Report from the American College of Cardiology/AHA Task Force on Practice Guidelines
- The Dietary Guidelines 2015 Advisory Committee Report [10]
- The Dietary Guidelines for Americans 2015 [5]

These guidelines are all consistent in recommending a dietary pattern that is higher in vegetables, fruits, whole grains, nonfat dairy, seafood, legumes, and nuts and for those who consume alcohol (among adults) to do so in moderate amounts if at all. In addition, the guidelines have consistently recommended diets that are lower in red and processed meat, lower in sugar sweetened foods and drinks and refined grains, and all have emphasized the importance of not overconsuming calories as a strategy for managing weight and thereby reducing cardiovascular risk. The guidelines have also uniformly recommended including increased physical activity as a key component of an overall positive lifestyle.

Broader Context

The national recommendations for nutrition and cardiovascular health have also moved to a broader context placing nutrition as a key component of overall strategies to reduce cardiovascular risk. The American Heart Association Strategic Goals for 2020 and Beyond is particularly instructive in this regard [3]. This important document introduced the concept "primordial" prevention (preventing risk factors from occurring in the first place rather than lowering them once they exist and also introducing the construct of "ideal" cardiovascular health).

Primordial prevention in the AHA Strategic Plan is distinguished from primary prevention by incorporating strategies to avoid adverse levels of cardiovascular risk factors in the first place rather than preventing the first occurrence of a clinical event or disease among individuals who are already at risk. The concept of "ideal" cardiovascular health was defined as a series of seven health behaviors and health factors, including not smoking, maintaining a healthy body mass index (BMI), achieving an appropriate level of physical activity, achieving a healthy diet score, maintaining a total cholesterol of less than 200 mg/dL, maintaining a blood pressure of less than 120/80 mmHg, and a fasting glucose of less than 100 mg/dL. (The cholesterol, blood pressure and glucose parameters were all defined as "untreated" values.)

Within the domain of nutrition, while recognizing that recommending an optimal pattern for cardiovascular risk reduction is a complicated task, the 2020 Strategic Plan defined dietary goals as "in the context diet that is appropriate in energy balance, pursuing an overall dietary plan that is consistent to DASH" (Dietary Approaches to Stop Hypertension) [11]. An eating plan including but not limited to the following was recommended:

- Fruits and vegetables \geq 4.5 cups a day
- Fish \geq two 3 $\frac{1}{2}$ servings per week (preferably oily fish)
- Fiber rich/whole grains (≥1.1 g of fiber per 10 g of carbohydrates): three 1 ounce equivalent servings per day
- Sodium ≤ 1500 mg per day
- Sugar-sweetened beverages \leq 450 kcal (36 ounces) per week

These recommendations seem reasonable and have been expanded upon in other guidelines and recent reviews.

Dietary Patterns

Over the last 20 years the emphasis in dietary guidance has moved from nutrients to individual foods and then ultimately to dietary patterns. Thus, the dietary patterns outlined above have been recommended in numerous guidelines. In addition, there is emerging evidence related to potential cardiovascular benefits from the Mediterranean Diet [12] which will be discussed in more detail later in this chapter.

Implementation

Emphasis has also shifted to the important aspect of implementing dietary guidelines. While recognizing that nutrition plays a key role in many aspects of preventing risk factors for CVD, it has also been recognized that despite decades of largely consistent recommendations in guidelines only a distinct minority of Americans are fully following these guidelines. For example, it has been estimated that less than 30% of adults in the United States consume the recommended number of servings of fruits and vegetables [13]. With regard to nutrition and hypertension, less than 20% of individuals are following the DASH diet [11]. Thus, the problem of how to encourage people to implement heart healthy guidelines in their daily lives remains an enormous challenge. This important topic has been the subject of recent reviews which will be discussed toward the end of this chapter [14].

The area of how to incorporate proven behavioral medicine techniques into nutrition and other aspects of positive lifestyle is another area of considerable ongoing research [15, 16].

American Heart Association Diet and Lifestyle Clinical Recommendations and Goals

In 2006, the AHA summarized diet and lifestyle recommendations in a scientific statement from the AHA Nutrition Committee [2]. While subsequent studies and research have refined these goals, the basic framework has stood the test of time and will be utilized in this chapter as a point of departure.

The AHA started from the premise that improving diet and lifestyle represented a critical component of an overall strategy to prevent CVD, which remains the leading cause of morbidity and mortality in Americans. The 2006 guidelines differ from previous guidelines in recommending a broader approach to not just diet, but overall lifestyle with a particular emphasis on physical activity. This framework also took into account that these recommendations have "public health and clinical applications." With this as background the following goals were set.

Consume an Overall Healthy Diet

This recommendation was based on the concept that we need to move from individual nutrients and foods to an overall dietary pattern. This concept has been further amplified in multiple guidelines and research projects since 2006. It was recognized that randomized control trials (RCTs) on the whole diet are difficult and expensive to conduct, nonetheless, abundant cohort studies and epidemiologic studies support the concept that the dietary pattern that comprises a variety of fruits and vegetables, whole grains, fat free and low fat dairy products, legumes, poultry, lean meats, and fish (preferably oily fish consumed at least twice a week) has been associated with lower risk of CVD [3, 17–20]. Specific components of this overall healthy diet will be discussed later in this chapter.

Aim for a Healthy Body Weight

The AHA document incorporates the framework from the Institute of Medicine establishing a healthy body weight defined as a body mass index (BMI) of 18.5–24.9 kg/m² while overweight is defined as a BMI between 25 and 29.9 kg/m² and obesity is defined as a BMI of greater or equal to 30 kg/m². This goal recognizes that obesity is an independent risk factor for CVD in addition to being related to other multiple risk factors such as dyslipidemias, high blood pressure, and diabetes [21, 22]. While both low levels of physical activity and over consumption of calories contribute to overweight and obesity, the Dietary Guidelines 2010 and the DGA 2015 both recognize overconsumption of calories as the leading nutritional problem in the United States.

The problem of overweight and obesity in the United States has truly reached epidemic proportions. Over two-thirds of the population in the United States are overweight or obese [23]. Current estimates reveal that 33.8% of adults, over 66 million Americans are obese (30 million men and 36 million women) while an additional 74 million (42 million men and 32 million women) are overweight. The prevalence of obesity has grown a shocking 40% over the last 30 years [24]. This problem is not exclusively found in the United States. The World Health Organization estimates that there are currently over a billion individuals worldwide who are obese and this will grow to 1.5 billion individuals by the year 2030 if current trends continue [25].

Aim for a Desirable Lipid Profile

Elevations in total cholesterol, as well as low-density lipoproteins (LDL) cholesterol, are established risk factors for CVD [26]. As levels of LDL cholesterol increase, so does the risk of developing CVD. The following levels of LDL have been defined: optimal, less than 100 mg/dL; near or above optimal, 100–129 mg/dL; borderline high, 130–159 mg/dL; high, 160–189 mg/dL; very high, \geq 190 mg/dL.

In 2013, the American College of Cardiology (ACC) and AHA issued updated guidelines for the treatment of blood cholesterol to reduce atherosclerotic cardiovascular disease in adults [27]. These guidelines recommended increased use of statin medicines to reduce atherosclerotic cardiovascular disease (ASCVD) events in primary and secondary prevention and also recommended discontinued use of specific LDL and high-density lipoprotein (HDL) treatment targets. This report identified four major groups that could benefit from statin medications:

- 1. Individuals with existing clinical ASCVD
- 2. Primary elevations of LDL-cholesterol (LDL-C)>190 mg/dL
- 3. Diabetics aged 40-75 years, with LDL-C 70-189 mg/dL and without clinical ASCVD
- 4. Diabetics with LDL-C 70–189 mg/dL who had a 10-year ASCVD risk of >7.5 %

These guidelines were immediately criticized for recommending excessive use of statins particularly in individuals with ASCVD risk of greater than 7.5%. This has remained controversial. It must be emphasized, however, that dietary changes such as following the overall AHA healthy diet plan with a particular emphasis on decreasing saturated fat and trans-fat, are recommended for all individuals.

Triglycerides and HDL cholesterol, which are related to CVD risk, can also be affected by diet and body weight [28, 29]. Major diet-related determinates of low HDL cholesterol are hyperglycemia, diabetes, hypertriglyceridemia, and very low fat diet (less than 15% energy as fat), as well as excess body weight. Triglyceride levels greater than 150 mg/dL are considered one of the criteria for classification of the metabolic syndrome [26]. Since there is an inverse relationship between triglyceride and HDL concentrations, dietary recommendations for elevated triglycerides are virtually the same as those for low HDL cholesterol.

Aim for a Normal Blood Pressure

Elevated blood pressure represents a significant risk factor for both CVD and stroke. Issues related to optimum levels of blood pressure control, however, have become somewhat controversial. Recommendations from the Joint National Commission VII (JNC VII) defined a normal blood pressure as less than 120/80 mmHg and defined 80–89 mmHg diastolic and 120–139 mmHg systolic as

prehypertension and greater than 140 mmHg as "hypertension" [30]. These were also the recommendations made by the AHA 2020 Strategic Plan [3].

These guidelines became controversial when the commission established to formulate JNC VIII guidelines made somewhat different recommendations [31]. JNC VIII guidelines made the following statement: "There is strong evidence to support treating hypertensive persons age 60 or older to a BP goal of less than 150/90 mmHg and hypertensive persons 30 through 59 years of age to a diastolic goal of less than 90 mmHg, however, there is insufficient evidence in hypertensive persons younger than 60 years for a systolic goal or in those younger than 30 years for a diastolic goal, so the panel recommends a BP of less than 140/90 mmHg for those groups based on expert opinion." These guidelines emphasized that while these targets were articulated, clinical judgment should still prevail in hypertensive therapy.

The recently completed, National Institutes of Health (NIH) funded Systolic Blood Pressure Intervention Trial (SPRINT Study) [32] demonstrated that individuals at increased risk for heart disease and kidney failure whose systolic blood pressure was controlled to ≤ 120 mmHg versus 140 mmHg achieved a 33% decrease in myocardial infarction or kidney failure and an overall 25% decrease in mortality from CVD. It should be noted, however, that to achieve these levels of blood pressure control, individuals, on average, needed three medications. This has raised issues about whether or not outside of a research setting, individuals would be willing to comply with the drug regimens required to meet these more stringent guidelines [33].

While some controversy persists regarding which recommendations to follow, it is important to emphasize that all of these guidelines emphasize nutritional interventions as a key component of the overall prevention and treatment of high blood pressure. In particular, a nutritional pattern that is consistent with the DASH diet has been clearly demonstrated to help control blood pressure [11, 34, 35]. Unfortunately, less than 20% of individuals who have high blood pressure actually follow the DASH diet [11]. Other dietary modifications demonstrated to lower blood pressure include reducing salt intake, caloric deficit to induce weight loss if necessary, moderate alcohol consumption (among those who drink alcohol), and increased potassium intake [25, 34].

Be Physically Active

Increased levels of moderate or vigorous intensity physical activity have been repeatedly shown to lower the risk of cardiovascular disease [1, 10, 36]. Guidelines such as the DGA 2015 now routinely place proper nutrition in the overall context of positive lifestyle decisions. Data related to physical activity and reduced risk of heart disease and other chronic diseases summarized in the 2008 US Department of Health and Human Services "Physical Activity Guidelines for Americans" [36]. This document was designed to complement the Dietary Guidelines for Americans. Physical activity is important for maintaining physical and cardiovascular fitness [9, 36], helping to control weight [37, 38] and helping to sustain weight loss once achieved. Unfortunately, current estimates suggest that over 60% of US adults do not engage in any regular physical activity [39].

The Physical Activity Guidelines for Americans are based on the fundamental concept that some physical activity is better than none and more is better than some. The specific recommendation is for adults to obtain at least 150 min per week of moderate intensity physical activity or 75 min per week of vigorous aerobic physical activity or some combination of the two to achieve substantial health benefits. Regular physical activity is also associated with improvement in other cardiovascular risk factors including blood pressure, lipid profiles, and blood sugar [9].

Avoid Use and Exposure to Tobacco Products

Overwhelming evidence exists from multiple sources that cigarette smoking significantly increases the risk of both heart disease and stroke [40]. This evidence has been ably summarized elsewhere and is incorporated as a recommendation in the AHA 2020 Strategic Plan as well as the AHA Diet and Lifestyle clinical recommendations and goals [3, 9]. Unfortunately, nearly 23% of US adults smoke cigarettes and the rate of decline of cigarette smoking has significantly slowed over the past 20 years [41]. Currently, risks of cigarette smoking for women are equivalent for men. Conversely substantial benefits from reduction of risk of CVD are seen in individuals who stop smoking cigarettes. These benefits can be seen over a very brief period of time [42].

AHA-Specific Nutrition and Lifestyle Recommendations

In this section we will discuss the specific AHA diet and lifestyle recommendations intended to reduce CVD risk. While these were the recommendations that were made in the AHA Scientific Statement from the Nutrition Committee in 2006 [2], a number of studies that have been carried out since that time have helped to further clarify and fine-tune these recommendations. The framework, however, remains very applicable to current knowledge about nutritional recommendations to lower the risk of CVD.

It should be emphasized, again, that while the guidance from these individual recommendations represents a consensus about current evidence related to nutritional practices to lower the risk of CVD, an overall healthy diet and physically activity lifestyle remain paramount for lowering CVD risk factors. It should also be noted that even if individual risk factors for CVD are not lowered by following the specific recommendations outlined in this section, adherence to a healthy diet and lifestyle will still lower CVD risk even if individual risk factors are not decreased.

Balance Calorie Intake and Physical Activity to Achieve or Maintain Healthy Body Weight

Achieving a healthy body weight is of paramount importance to lower the risk of CVD [2, 9, 43, 44]. Key drivers of achieving and maintaining a healthy body weight include proper portion size and regular physical activity. It is important to follow the general principles of a heart healthy diet as articulated by numerous professional organizations such as the AHA [2], DGA 2010 [45], and DGA 2015 [5].

If weight loss is desired, reduced caloric consumption is critically important. It has been estimated that the average American adult in 2010 consumed an average of over 450 calories more than in 1970 [46]. Even the reduction of 100 kcal/day would make a meaningful impact on lowering weight [47]. Numerous studies have shown that the macronutrient content of weight reducing diets does not matter [48]. It is more important to emphasize adherence to whatever reduced calorie diet the individual is following. Physical activity is also important for the maintenance of weight loss. Regular daily physical activity has been shown to be an important part of maintaining weight loss once achieved [43, 44]. A combination of moderate and vigorous activity may also be highly appropriate. In the area of weight loss, 60 min of physical activity on most days is recommended for adults attempting to lose weight or maintain weight loss [36]. The same recommendations have been made for children [36].

It is important to note that these levels of physical activity can be accumulated over the course of the day and do not need to be conducted in one session.

Consume a Diet Rich in Vegetables

Multiple RCTs that have emphasized increased consumption of fruits and vegetables improve multiple risk factors for CVD including blood pressure, lipid levels, insulin resistance, endothelial function, and weight control [4, 17–19]. In addition, diets that are high in fruits and vegetables meet both micronutrient and macronutrient and fiber requirements and help control overall energy consumption. It appears that the benefits of fruits and vegetables come from synergistic interactions of multiple phytochemicals. These benefits do not appear to be reproducible with equivalent amounts of supplements. In observational studies, greater fruit and vegetable consumption are each associated with lower incidence of CHD while higher fruit consumption is associated with lower incidence of stroke [49, 50]. While a variety of fruits and vegetables are recommended, those that are deeply colored throughout, such as peaches, carrots, spinach, and berries, should be emphasized because of their high micronutrient content. Preparation also matters, which is important for preserving nutrient and fiber content without adding unnecessary calories such as fats, sugar, and salt. Thus, fresh, frozen, or canned vegetables without high calorie sauces, or added salt or sugars, are highly recommended.

Chose Whole Grain, High Fiber Foods

Whole grains generally comprise bran, germ, and endosperm from natural cereals [4, 51]. Bran typically contains both soluble and insoluble fiber and multiple vitamins, minerals, and flavonoids, while the germ contains fatty acids, antioxidants, and other phytochemicals. Dietary patterns that are high in whole grain products and fiber have been associated with improved dietary quality and decreased risk of CVD [51]. Soluble fibers such as those found in oat products may assist in modestly reducing LDL cholesterol levels beyond those achieved through other dietary factors. It is important to note that simply because a product is whole grain, it does necessarily follow that it is high in fiber [52, 53]. It is important to carefully read the label.

The AHA Nutrition Guidelines recommends that at least half of grain intake should be from whole grains [2] while the AHA Strategic Plan for 2020 recommends fiber-rich whole grains (\geq 1.1 g of fiber for 10 g of carbohydrates) and >3 one ounce servings of fiber-rich whole grains per day [3].

Consume More Fish and Other Seafood

Fish and other seafood contain multiple helpful nutrients including long chain omega 3 polyunsaturated fats (PUFAs) as well as eicosatpentaenoic acid (EPA) and docosahexenoic acids (DHA) [4]. Multiple studies have shown that fish oil has direct antiarrhythmic effects [54, 55]. Oily fish, in particular, are recommended including wild salmon, trout, anchovies, and herring. Consuming an average of two fish meals per week has been associated in multiple studies with lower incidence of CHD, ischemic stroke, and risk of sudden cardiac death [54, 55]. The reproduction of the benefits of consuming fish by substituting fish oil supplements has not been established. Contamination of certain fish with mercury and other organic compounds may potentially be of concern particularly for children and pregnant women who have been advised to avoid eating fish with the highest level of such contamination (such as shark and swordfish) [56, 57]. Information on potential contamination of fish may be obtained from local and state authorities and the Food and Drug Administration (FDA) website. In general, the benefits of consuming fish (particularly oily fish) far outweigh the risk for most segments of the population.

Preparation methods for fish should minimize the addition of saturated and trans-fats which often increase through the use of hydrogenated fat during frying or found in cream sauces.

Limit Intake of Saturated and Trans Fat and Cholesterol

Dietary patterns low in saturated and trans-fatty acids and cholesterol have been shown in multiple studies to lower the risk of CVD largely through their effects on LDL cholesterol levels [2]. The major sources of saturated fatty acids in the US diet are animal fats (meat and dairy) while the primary sources of trans-fat are partially hydrogenated fats used in commercially fried or baked products [58–60].

Recent studies have suggested that the food metric that saturated fats are found in can greatly impact on whether or not these fatty acids increase the risk of heart disease [61, 62]. In dairy products, in particular, in some studies fatty acids found in dairy products have not been found to increase the risk of heart disease in contrast to saturated fatty acids found in meat [61, 62].

The American Heart Association recommends a goal of less than 7% of energy as saturated fat and less than 1% of energy as trans-fat and less than 300 mg of cholesterol per day [2]. Strategies to achieve these levels can be achieved by replacing whole fat versions of foods (e.g., replacing full fat dairy products with nonfat or lower fat versions, choosing lean cuts of meat, or replacing meat with vegetable alternatives (e.g., beans or fish). Efforts to reduce trans fatty acids, typically rely on the substitution of partially hydrogenated fats with those made with liquid vegetable oils except tropical vegetable oils. As of January 1, 2006, mandatory trans-fat labeling was required, which allows for easier identification to help individuals limit trans fatty acids [60]. Multiple scientific bodies have urged reduction of consumption of trans fatty acids to the lowest level possible (64). These organizations have consistently recommended a diet containing less than 1% trans fatty acids.

Polyunsaturated and monounsaturated fats may be good replacements for saturated fat to lower the risk of CHD. A range of 25–35% of total fat can fit within a healthy dietary pattern as long as the amount of saturated fat is limited to less than 7% of energy.

Moderate Your Intake of Beverages and Food with Added Sugars

The AHA has recommended that the average adult male consume no more than 150 kcal per day in added sugars and the average female consume no more than 100 kcal per day in added sugars [36]. Between 1977–1978 and 1999–2002, the percentage of energy consumed from added sugars rose from 13.1% to 16.6% [63, 64]. However, the consumption of added sugars has been in significant decline since the year 2002 [65]. These declines have amounted to approximately a 15–20% reduction in added sugars. Some epidemiologic studies have suggested that added sugars may increase the risk of heart disease, diabetes, and obesity [66–68]. However, other studies have not confirmed these findings [69, 70]. An entire chapter in the book is devoted to this controversial area (Chapter 20: Added Sugars and Health: What do we Really Know?).

Chose and Prepare Foods with Little or No Salt

There is a well-known association between salt (sodium chloride) intake and blood pressure [71, 72]. A reduced salt intake in the presence of elevated blood pressure can facilitate hypertension control and may prevent hypertension in nonhypertensive individuals.

The recommended upper limit of sodium consumption in the diet has been disputed. The AHA has recommended an upper limit of no more than 2300 mg per day and in certain population groups, no more than 1500 mg per day as mechanisms for population-wide lowering of blood pressure [73].

A recent multination study employing modeling estimated that 1.65 million deaths from cardiovascular disease, which occurred in 2010 could be attributed to sodium consumption above the reference level of 2.0 g/day [74]. However, another study of over 100,000 persons in 17 countries using estimates of 24-h sodium and potassium excretion (used as a surrogate for intake) found that an estimated sodium intake of between 3 and 6 g/day was associated with the lower risk of death and cardiovascular events than with either a higher or lower estimated level of intake [75]. The average individual in the United States currently consumes 3.4 g of sodium per day, which would fall within this optimum range. Thus, the optimum level of sodium consumption current remains in dispute.

It should be mentioned, however, that diets rich in potassium lower blood pressure and also blunt blood pressure raising effects of increased sodium intake.

If You Consume Alcohol, Do So in Moderation

The AHA recommends that if alcoholic beverages are consumed, they should be consumed in moderation [2]. This means that no more than two alcoholic drinks per day for men and one drink for women. In general, a 4 ounce glass of wine, a 12 ounce bottle of beer, or a 1.5 ounce shot of 80% spirits all contain the same amount of alcohol (one half ounce). Thus, each of these should be considered equivalent of one drink.

The rationale for this recommendation is that modest alcohol consumption has been shown in a number of studies to raise high density lipoprotein cholesterol (HDL), reduce systemic inflammation, and improve insulin resistance [76, 77]. Individuals who consume moderate alcohol experience a lower incidence of CHD and diabetes mellitus (DM). Most studies support that it is the alcohol content of these beverages that conveys these benefits. However, some nonalcoholic components such as resveratrol in wine may also have some potential benefits.

Alcohol levels above this have been shown to carry multiple adverse effects such as increase in cardiomyopathy and cardiac failure as well as high risk of atrial fibrillation [21]. Higher alcohol consumption has also been associated with motor vehicle accidents, homicides, and suicide and thus, alcohol use has an overall net adverse effect on population mortality and is not recommended as a population-based strategy to reduce CVD risk. It is also important, with regard to alcohol consumption, to avoid weight gain since the average serving of alcohol contributes between 120 and 200 kcal and because of the liquid nature may be less satiating then those from solid foods.

When Eating Foods Prepared Outside the Home Follow the AHA Guidelines

Americans often consume food prepared outside the home. Sources of "away" food include food prepared at restaurants and grocery stores, schools, day care centers, and other nonhome locations. It has been estimated that over one-third of calories consumed by Americans come from these sources [78]. Often "away" food comes in large portion sizes and contains high energy density. These foods

may also be high in saturated fat, trans-fat, sodium, and added sugars. For all of these reasons, it is important that individuals be counseled that when eating food away from the home, the same principles of the healthy AHA diet should be applied.

Implementing Heart Healthy Nutrition Plans

Given the pivotal importance of sound nutrition to reduce multiple risk factors for CVD, it is important that strategies be developed to help individuals consume a more heart healthy diet. This was the emphasis of an AHA Scientific Statement published in 2009 entitled "Implementing American Heart Association Pediatric and Adult Nutrition Guidelines [79]." This statement emphasized the complexity of factors impacting on nutritional choices and offered a multilevel framework for those factors. This framework started with individual factors and then was placed more broadly in family environment factors, the microenvironmental and finally macroenvironmental factors. Each of these interacting domains contain multiple influences, which are discussed in more detail in Chapter 1.

In order to positively impact on these multiple factors, it becomes incumbent to employ proven models from behavioral medicine which have demonstrated effectiveness in helping individuals adopt more positive behaviors in general and in the area of nutrition, in particular. These models of behavior change are beyond the scope of this chapter but have been extensively reviewed elsewhere [15, 16, 80].

Conclusions/Summary

There is no longer any serious doubt that nutritional practices strongly interact with the likelihood of developing CVD. These factors should be placed in the overall context of positive lifestyle habits and practices. This domain, which has been termed "lifestyle medicine," offers a promising framework for impacting on both nutritional factors and other lifestyle-related factors and their impact for risk factors CVD [1, 81, 82].

The evidence-based guidelines from the AHA [2] and the Dietary Guidelines for American [5] and the American College of Cardiology [9] are all consistent in recommending an overall heart healthy approach to nutrition including a dietary pattern that is rich in vegetables, fruits, whole grains, seafood, legumes, and nuts and in low and nonfat dairy products and alcohol (among adults), lowering meat and processed meats and low in sugar sweetened fruits and vegetables and refined grains. While these patterns can be achieved in many different ways, they should be tailored to individuals' cultural, biological, and medical needs.

Implementation of these guidelines remains an important challenge and will require a recognition of the multiple factors both individual, family, cultural, environmental, and public policy and interact on both individual and population-wide nutritional choices. A sophisticated understanding of emerging science and behavioral medicine will be essential to accomplish the goal of helping individuals consume a more heart healthy diet.

References

- Rippe JM, Angelopoulos TJ. Lifestyle strategies for cardiovascular risk reduction. Current atherosclerosis reports. 2014;16:444(10):1–7. Epub 2014/08/06.
- American Heart Association Nutrition C, Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. Circulation. 2006;114(1):82–96. Epub 2006/06/21.

- Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, et al. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's strategic impact goal through 2020 and beyond. Circulation. 2010;121(4):586–613.
- Mozaffarian D, Appel LJ, Van Horn L. Components of a cardioprotective diet: new insights. Circulation. 2011;123(24):2870–91.
- U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015 2020 Dietary Guidelines for Americans. 8th ed. 2015. Available at http://health.gov/dietaryguidelines/2015/guidelines/. Epub December 2015.
- Liu S, Stampfer MJ, Hu FB, et al. Whole-grain consumption and risk of coronary heart disease: results from the Nurses' Health Study. Am J Clin Nutr. 1999;70:412–9.
- 7. Stampfer MJ, Hu FB, Manson JE, et al. Primary prevention of coronary heart disease in women through diet and lifestyle. N Engl J Med. 2000;343:16–22.
- Ford ES, Ajani UA, Croft JB, et al. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. N Engl J Med. 2007;356:2388–98.
- Eckel RH, Jakicic JM, Ard JD, et al. AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2013;2013.
- Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans US Department of Agriculture Center for Nutrition Policy and Promotion. Washington DC; 2015.
- 11. Appel LJ, Brands MW, Daniels SR, Karanja N, Elmer PJ, Sacks FM. Dietary approaches to prevent and treat hypertension: a scientific statement from the American Heart Association. Hypertension. 2006;47(2):296–308.
- 12. Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, Aros F, et al. Primary prevention of cardiovascular disease with a Mediterranean diet. N Engl J Med. 2013;368(14):1279–90. Epub 2013/02/26.
- 13. Scientific Advisory Committee on Nutrition. SACN carbohydrates and health report. 2015.
- Epstein LH, Carr KA, Cavanaugh MD, Paluch RA, Bouton ME. Long-term habituation to food in obese and nonobese women. Am J Clin Nutr. 2011;94(2):371–6.
- 15. Ockene JK, Schneider KL, Lemon SC, et al. Can we improve adherence to preventive therapies for cardiovascular health? Circulation. 2011;124(11):1276–82.
- Stuart-Shor EM, Berra KA, Kamau MW, et al. Behavioral strategies for cardiovascular risk reduction in diverse and underserved racial/ethnic groups. Circulation. 2012;125(1):171–84.
- 17. Knoops KT, de Groot LC, Kromhout D, et al. Mediterranean diet, lifestyle factors, and 10-year mortality in elderly European men and women: the HALE project. JAMA. 2004;292:1433–9.
- Appel LJ, Moore TJ, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997;336:1117–24.
- 19. Appel LJ, Sacks FM, Carey VJ, et al. OmniHeart Collaborative Research Group. The effects of protein, monounsaturated fat, and carbohydrate intake on blood pressure and serum lipids: results of the OmniHeart randomized trial. JAMA. 2005;294:2455–64.
- 20. van Dam RM, Rimm EB, Willett WC, et al. Dietary patterns and risk for type 2 diabetes mellitus in U.S. men. Ann Intern Med. 2002;136:201–9.
- 21. Rippe J, Angelopoulos T. Obesity and heart disease. In: Rippe JM, Angelopoulos TA, editors. Obesity: prevention and treatment. Boca Raton: CRC Press; 2012.
- 22. Rashid MN, Fuentes F, Touchon RC, Wehner PS. Obesity and the risk for cardiovascular disease. Prev Cardiol. 2003;6:42–7.
- Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999-2008. JAMA. 2010;303(3):235–41. Epub 2010/01/15.
- 24. Center for Disease Control and Prevention (CDC). Overweight and obesity: US obesity trends. Atlanta: U.S. Department of Health and Human Services; 2015.
- World Health Organization. Risk Factor Projects. Overweight and obesity. 2005. Available at: http://www.who.int/ chp/chronic_disease_report/part2_ch1/en/index16.html. Accessed 4 Feb 2016.
- 26. Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA. 2001;285:2486–97.
- 27. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Circulation. 2014;129(25 Suppl 2):S1–45. Epub 2013/11/14.
- Wilson PW, Grundy SM. The metabolic syndrome: a practical guide to origins and treatment: part II. Circulation. 2003;108:1537–40.
- 29. Howard BV, Ruotolo G, Robbins DC. Obesity and dyslipidemia. Endocrinol Metab Clin North Am. 2003;32:855–67.
- Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo Jr JL, et al. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. JAMA. 2003;289(19):2560–72. Epub 2003/05/16.

- 31. James PA, Oparil S, Carter BL, Cushman WC, Dennison-Himmelfarb C, Handler J, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: Report from the panel members appointed to the Eighth Joint National Committee (JNC 8). JAMA. 2014;311(5):507–20. Epub 2013/12/20.
- 32. The SPRINT Research Group. A randomized trial of intensive versus standard blood-pressure control. N Engl J Med. 2015;373(22):2103–16.
- 33. Chobanian AV. Time to reassess blood-pressure goals. N Engl J Med. 2015;373(22):2093-5.
- Obarzanek E, Sacks FM, Vollmer WM, Bray GA, Miller 3rd ER, Lin PH, et al. Effects on blood lipids of a blood pressure-lowering diet: the Dietary Approaches to Stop Hypertension (DASH) Trial. Am J Clin Nutr. 2001;74(1):80– 9. Epub 2001/07/14.
- 35. Greenland P. Beating high blood pressure with low-sodium DASH. N Engl J Med. 2001;344(1):53–5. Epub 2001/01/04.
- 36. Johnson R, Appel L, Brands M, Howard B, Lefevre M, Lustig R, Sacks F, Steffen L, Wylie-Rosett J. American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity, and Metabolism and the Council on Epidemiology and Prevention. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. Circulation. 2009;120:1011–20.
- Fogelholm M, Kukkonen-Harjula K. Does physical activity prevent weight gain a systematic review. Obes Rev. 2000;1(2):95–111. Epub 2002/07/18.
- Hill JO, Wyatt HR, Phelan S, Wing RR. The National Weight Control Registry: is it useful in helping deal with our obesity epidemic? J Nutr Educ Behav. 2005;37:2016–210.
- American Heart Association. Heart Disease and Stroke Statistics–2005. Update. Dallas: American Heart Association; 2005. Available at http://www.americanheart.org/presenter.jhtml!identifier_1928. Accessed 4 Feb 2016.
- 40. The health consequences of smoking: a report of the surgeon general. Atlanta: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. 2004.
- Centers for Disease Control and Prevention. National Center for Health Statistics. National Health Interview Survey, 1997–2012, Sample Adult Core component. Available from: http://www.cdc.gov/nchs/data/nhis/earlyrelease/earlyrelease201306_08.pdf. Accessed on 4 Feb 2016.
- Jha P, Ramasundarahettige C, Landsman V, et al. 21st-century hazards of smoking and benefits of cessation in the United States. N Engl J Med. 2013;368:341–50.
- 43. Hill JO, Thompson H, Wyatt H. Weight maintenance: what's missing? J Am Diet Assoc. 2005;105 suppl 1:S63-6.
- 44. Wing RR, Phelan S. Long-term weight loss maintenance. Am J Clin Nutr. 2005;82:222S-5S.
- 45. U.S. Department of Agriculture, U.S. Department of Health and Human Services. Report of the Advisory Committee on the Dietary Guidelines for Americans 2010 7th ed. Washington, DC U.S. Government Printing Office; 2010.
- 46. USDA Department of Agriculture, Economic Research Service. Calories average daily per capita calories from the US food supply, adjusted for spoilage and other waste. Loss-Adjusted Food Availability Data. 2013.
- 47. Hill JO, Peters JC, Wyatt HR. Using the energy gap to address obesity: a commentary. J Am Diet Assoc. 2009;109(11):1848–53.
- 48. Foster G, Wyatt H. A randomized trial of a low-carbohydrate diet for obesity. N Engl J Med. 2003;348:2082-90.
- 49. Bazzano LA, Serdula MK, Liu S. Dietary intake of fruits and vegetables and risk of cardiovascular disease. Curr Atheroscler Rep. 2003;5:492–9.
- Hung HC, Joshipura KJ, Jiang R, et al. Fruit and vegetable intake and risk of major chronic disease. J Natl Cancer Inst. 2004;96:1577–84.
- 51. Hu FB, Willett WC. Optimal diets for prevention of coronary heart disease. JAMA. 2002;288:2569-78.
- Quagliani D, Felt-Gunderson P. Closing America's fiber intake gap: communication strategies from a food and fiber summit. Am J Lifestyle Med. 2015. First published on June 2, 2015. doi:10.1177/1559827615588079.
- Pereira MA, O'Reilly E, Augustsson K, et al. Dietary fiber and risk of coronary heart disease: a pooled analysis of cohort studies. Arch Intern Med. 2004;164:370–6.
- Kris-Etherton PM, Harris WS, Appel LJ, American Heart Association. Nutrition Committee. Fish consumption, fish oil, omega-3 fatty acids, and cardiovascular disease. Circulation. 2002;106:2747–57.
- 55. Wang C, Chung M, Balk E, et al. N-3 fatty acids from fish or fish-oil supplements, but not alpha-linolenic acid, benefit cardiovascular disease outcomes in primary and secondary-prevention studies: a systematic review. Am J Clin Nutr. 2006;83:5–17.
- Foran JA, Carpenter DO, Hamilton MC, et al. Risk-based consumption advice for farmed Atlantic and wild Pacific salmon contaminated with dioxins and dioxin-like compounds. Environ Health Perspect. 2005;113:552–6.
- 57. US Department of Health and Human Services, Food and Drug Administration, Center for Food Safety and Applied Nutrition. Methylmercury in fish—summary of key findings from focus groups about the methylmercury advisory. Available at: http://www.cfsan.fda.gov/dms/admehg3g.html. Accessed 4 Feb 2016.
- 58. Subcommittees on Upper Reference Levels, Institute of Medicine of the National Academies. Dietary reference intakes: energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids. Washington, DC: National Academies Press; 2005.
- 59. Ascherio A, Katan MB, Zock PL, et al. Trans fatty acids and coronary heart disease. N Engl J Med. 1999;340:1994-8.

- 60. US Department of Agriculture, Agricultural Research Service, Dietary Guidelines Advisory Committee. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans. 2005.
- 61. de Oliveira Otto MC, Mozaffarian D, Kromhout D, Bertoni AG, Sibley CT, Jacobs Jr DR, et al. Dietary intake of saturated fat by food source and incident cardiovascular disease: the Multi-Ethnic Study of Atherosclerosis. Am J Clin Nutr. 2012;96(2):397–404. Epub 2012/07/05.
- 62. Forouhi NG, Koulman A, Sharp SJ, Imamura F, Kroger J, Schulze MB, et al. Differences in the prospective association between individual plasma phospholipid saturated fatty acids and incident type 2 diabetes: the EPIC-InterAct case-cohort study. Lancet Diabetes Endocrinol. 2014;2(10):810–8. Epub 2014/08/12.
- 63. Cook AJ, Friday JE. Pyramid Servings Intakes in the United States 1999 2002, 1 Day. Beltsville: USDA, Agricultural Research Service, Community Nutrition Research Group; 2005.
- Block G. Foods contributing to energy intake in the US: data from NHANES III and NHANES 1999–2000. J Food Compost Anal. 2004;17:439–47.
- 65. Welsh JA, Sharma AJ, Grellinger L, Vos MB. Consumption of added sugars is decreasing in the United States. Am J Clin Nutr. 2011;94(3):726–34. Epub 2011/07/15.
- 66. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. N Engl J Med. 2011;364(25):2392–404.
- 67. Teff KL, Grudziak J, Townsend RR, Dunn TN, Grant RW, Adams SH. Endocrine and metabolic effects of consuming fructose- and glucose-sweetened beverages with meals in obese men and women: influence of insulin resistance on plasma triglyceride responses. J Clin Endocrinol Metab. 2009;94(5):1562–59. Epub 2009/02/12.
- 68. Antar MA, Little JA, Lucas C, Buckley GC, Csima A. Interrelationship between the kinds of dietary carbohydrate and fat in hyperlipoproteinemic patients. 3. Synergistic effect of sucrose and animal fat on serum lipids. Atherosclerosis. 1970;11(2):191–201. Epub 1970/03/01.
- Sievenpiper JL, Tappy L, Brouns F. Fructose as a driver of diabetes: an incomplete view of the evidence. Mayo Clin Proc. 2015;90(7):984–8.
- Kaiser KA, Shikany JM, Keating KD, Allison DB. Will reducing sugar-sweetened beverage consumption reduce obesity? Evidence supporting conjecture is strong, but evidence when testing effect is weak. Obes Rev. 2013;14(8):620–33.
- Sacks FM, Svetkey LP, Vollmer WM, et al. DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. N Engl J Med. 2001;344:3–10.
- Johnson AG, Nguyen TV, Davis D. Blood pressure is linked to salt intake and modulated by the angiotensinogen gene in normotensive and hypertensive elderly subjects. J Hypertens. 2001;19:1053–60.
- http://www.heart.org/HEARTORG/HealthyLiving/HealthyEating/Nutrition/The-American-Heart-Associations-Diet-and-Lifestyle-Recommendations_UCM_305855_Article.jsp#.VrS6Zhg. Accessed 5 Feb 2016.
- Mozaffarian D, Fahimi S, Singh GM, Micha R, Khatibzadeh S, Engell RE, et al. Global sodium consumption and death from cardiovascular causes. N Engl J Med. 2014;371(7):624–34.
- O'Donnell M, Mente A, Yusuf S. Sodium and cardiovascular disease. N Engl J Med. 2014;371(22):2137–8. Epub 2014/11/27.
- Flesch M, Rosenkranz S, Erdmann E, Bohm M. Alcohol and the risk of myocardial infarction. Basic Res Cardiol. 2001;96:128–35.
- 77. Goldberg IJ, Mosca L, Piano MR, et al. Nutrition Committee Co, Prevention, et al. AHA Science Advisory: wine and your heart: a science advisory for healthcare professionals from the Nutrition Committee, Council on Epidemiology and Prevention, and Council on Cardiovascular Nursing of the American Heart Association. Circulation. 2001;103(3):472–5. Epub 2001/02/07.
- Guthrie JF, Lin BH, Frazao E. Role of food prepared away from home in the American diet, 1977-78 versus 1994-96: changes and consequences. J Nutr Educ Behav. 2002;34(3):140–50. Epub 2002/06/06.
- 79. Gidding SS, Lichtenstein AH, Faith MS, Karpyn A, Mennella JA, Popkin B, et al. Implementing American Heart Association Pediatric and Adult Nutrition Guidelines: A Scientific Statement From the American Heart Association Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism, Council on Cardiovascular Disease in the Young, Council on Arteriosclerosis, Thrombosis and Vascular Biology, Council on Cardiovascular Nursing, Council on Epidemiology and Prevention, and Council for High Blood Pressure Research. Circulation. 2009;119(8):1161–75.
- Linke SE, Robinson CJ, Pekmezi D. Applying psychological theories to promote healthy lifestyles. Am J Lifestyle Med. 2014;8(1):4–14.
- 81. Rippe JM. Lifestyle medicine. 2nd ed. Boca Raton: CRC Press; 2013.
- 82. Rippe JM. Encyclopedia of lifestyle medicine and health. SAGE Publications. Thousand Oaks; 2012.
- 83. US Department of Health and Human Services, Food and Drug Administration, Center for Food Safety and Applied Nutrition. Nutrition subcommittee meeting: total fat and trans fat. Washington, DC. 2004. Available at: jttp://www. fda.gov/ohrms/dockets/ac/04/transcripts/4035t1.htm. Accessed 4 Feb 2016.

Chapter 8 Nutrition Therapy for the Prevention and Treatment of Prediabetes and Diabetes

Marion J. Franz

Key Points

- In persons with prediabetes, nutrition therapy interventions including a reduced energy intake resulting in a modest weight loss (5–7% of body weight), moderate physical activity (equivalent of 30-min brisk walking on most days of the week), education, and support have been shown to decrease the risk of converting to diabetes by 28–67%.
- The outcome of this level of lifestyle interventions is reported to persist for up to 15–20 years.
- In persons with type 2 diabetes (T2D), nutrition therapy interventions implemented by registered dietitian nutritionists (RDNs) reduced hemoglobin A1_c (A1C) levels by 0.3–2.0% and in persons with type 1 diabetes (T1D) by 1.0–1.9%, depending on the duration of diabetes and the A1C level at implementation. Improvements in A1C levels were maintained for 12 months and longer.
- A unifying focus of nutrition therapy interventions for persons with type 2 diabetes is a reduced energy intake and for persons with type 1 diabetes adjusting insulin based on carbohydrate counting.
- There is no ideal percentage of calories from carbohydrate, protein, and fat that applies to all persons with diabetes; total energy intake, rather than the source of the energy, is of importance.
- Recommended for all persons with diabetes is portion control of all foods with an emphasis on choosing nutrient-dense, high-fiber foods whenever possible instead of processed foods with added sodium, fat, and sugars; avoidance of sugar-sweetened beverages (SSB); selection of leaner protein sources and meat alternatives; and substitution of foods higher in unsaturated fat for foods high in saturated or trans-fats.
- Selecting a meal planning approach or eating pattern should be individualized and based on the individual's personal and cultural preferences, their literacy and numeracy, and their readiness, willingness, and ability to change. Collaboration between health professionals and persons with diabetes in determining nutrition therapy interventions and goals is essential.
- Physical activity is encouraged for children and adults with diabetes or prediabetes.
- Nutrition therapy interventions must be integrated into the overall diabetes management plan.
- Monitoring outcomes and providing ongoing education and support is essential; nutrition therapy
 interventions and medications may need to be adjusted over time based on changes in life circumstances, preferences, and disease course.

M.J. Franz, MS, RDN, CDE Nutrition Concepts by Franz, Inc., 6635 Limerick Drive, Minneapolis, MN 55439, USA e-mail: MarionFranz@aol.com **Keywords** Prediabetes • Type 1 diabetes • Type 2 diabetes • Nutrition therapy • Physical activity • Education/counseling and support • Monitoring • Pharmacological therapy • Collaboration • Lifestyle goals • Medical management

Abbreviations

A1C	Hemoglobin A1 _c
Academy	Academy of Nutrition and Dietetics
ACE	Angiotensin-converting enzyme inhibitor
ADA	American Diabetes Association
ARB	Angiotensin receptor blocker
AUC	Area under glucose curve
BMI	Body mass index
CGM	Continuous glucose monitoring
CSII	Continuous subcutaneous insulin input
CVD	Cardiovascular disease
DCCT	Diabetes Control and Complications Trial
DPP	Diabetes Prevention Program
DPP-4	Dipeptidyl peptidase 4 inhibitor
DPS	Diabetes Prevention Study
DSME	Diabetes self-management education
DSMS	Diabetes self-management support
GI	Glycemic index
GLP-1	Glucagon-like peptide 1 agonist
IDF	International Diabetes Federation
MDI	Multiple-dose insulin injections
MED Diet	Mediterranean diet
MNT	Medical nutrition therapy
NNS	Non-nutritive sweeteners
NTPG	
UTFU	Nutrition therapy practice guidelines
PCOS	Nutrition therapy practice guidelines Polycystic ovary syndrome
PCOS	Polycystic ovary syndrome
PCOS RCT	Polycystic ovary syndrome Randomized controlled trial
PCOS RCT RDN	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist
PCOS RCT RDN SFA	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist Saturated fatty acids
PCOS RCT RDN SFA SGLT-2	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist Saturated fatty acids Sodium-glucose cotransporter 2 inhibitor Self-monitoring of blood glucose
PCOS RCT RDN SFA SGLT-2 SMBG	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist Saturated fatty acids Sodium-glucose cotransporter 2 inhibitor
PCOS RCT RDN SFA SGLT-2 SMBG SOS	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist Saturated fatty acids Sodium-glucose cotransporter 2 inhibitor Self-monitoring of blood glucose Swedish Obese Subjects
PCOS RCT RDN SFA SGLT-2 SMBG SOS SSB	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist Saturated fatty acids Sodium-glucose cotransporter 2 inhibitor Self-monitoring of blood glucose Swedish Obese Subjects Sugar sweetened beverages
PCOS RCT RDN SFA SGLT-2 SMBG SOS SSB T1D	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist Saturated fatty acids Sodium-glucose cotransporter 2 inhibitor Self-monitoring of blood glucose Swedish Obese Subjects Sugar sweetened beverages Type 1 diabetes
PCOS RCT RDN SFA SGLT-2 SMBG SOS SSB T1D T2D	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist Saturated fatty acids Sodium-glucose cotransporter 2 inhibitor Self-monitoring of blood glucose Swedish Obese Subjects Sugar sweetened beverages Type 1 diabetes Type 2 diabetes
PCOS RCT RDN SFA SGLT-2 SMBG SOS SSB T1D T2D TZD	Polycystic ovary syndrome Randomized controlled trial Registered Dietitian Nutritionist Saturated fatty acids Sodium-glucose cotransporter 2 inhibitor Self-monitoring of blood glucose Swedish Obese Subjects Sugar sweetened beverages Type 1 diabetes Type 2 diabetes Thiazolidinedione

Introduction

As the prevalence of diabetes increases worldwide, lifestyle medicine focusing on nutrition therapy, physical activity, education/counseling and support, and monitoring interventions increases in importance. The International Diabetes Federation (IDF) estimates that 387 million adults in the world today live with diabetes type 1 or type 2, and almost half of them do not know it [1]. Furthermore, the IDF estimates that 592 million people will be living with diabetes by 2035. Of concern is that 77 % of people with diabetes live in low- and middle-income countries where only 18 % of total diabetes expenditure is spent.

In the United States in 2011–2012, the estimated prevalence of diabetes is 12-14% among adults, depending on the criteria used, with a higher prevalence among adults who are non-Hispanic black, non-Hispanic Asian, and Hispanic [2]. Of interest is that the proportion of people who have undiagnosed diabetes has significantly decreased by ~3.1–5.2%. The prevalence of prediabetes is reported to be 37-38% in the overall US population, and consequently, 49-52% of the US population is estimated to have either diabetes or prediabetes. Prevalence of diabetes among youth is also increasing. Over an 8-year period (2001–2009), the prevalence of type 1 diabetes (T1D) in youth increased by 21% and the prevalence of type 2 diabetes (T2D) increased by 30.5% [3]. Diagnostic criteria for diabetes mellitus and prediabetes are listed in Table 8.1 [4].

It is encouraging to note that rates of diabetes-related complications have declined substantially in the United States in the past 20 years although the burden of disease continues because of the continued increase in the prevalence of diabetes [5]. The improvements reflect a combination of advances and improvements in acute clinical care and health care systems and health promotion efforts directed at persons with diabetes. However, there continues to be an increased need for persons with diabetes to receive nutrition therapy and/or diabetes education [6]. National data in the United States indicate that only about half of the people with diabetes receive some type of diabetes education and even fewer see a Registered Dietitian Nutritionist (RDN). In one study of 18,404 patients with diabetes, only 9.1 % had at least one nutrition visit within a 9-year period [7].

The goals of nutrition therapy for diabetes are interventions that promote healthy eating patterns, emphasize a variety of nutrient-dense foods in appropriate portion sizes, and to assist in achieving glucose, lipid, blood pressure, and body weight goals [6]. However, the nutrition therapy interventions selected must meet the individual's goals and lifestyle, must be done in collaboration between health professionals and persons with diabetes, and must be strategies that the individual with diabetes is willing and able to make.

This chapter first reviews the evidence for the effectiveness of nutrition therapy (including physical activity) for the prevention of diabetes followed by a review of effective prediabetes nutrition therapy interventions. Summarized next is the evidence for the effectiveness of nutrition therapy in the management T1D and T2D and nutrition therapy interventions (including physical activity). Education and counseling strategies are reviewed and medications (glucose-lowering medications and insulin) summarized.

Table 8.1	Criteria for the diagno	sis of diabetes an	d categories of increased	l risk for diabetes	(prediabetes)
-----------	-------------------------	--------------------	---------------------------	---------------------	---------------

	Diabetes mellitus	Prediabetes
A1C, %	≥6.5	5.7-6.4
Fasting glucose, mg/dL (mmol/L)	≥126 (7.0)	100-125 (5.5-6.9)
2 h glucose, mg/dL (mmol/L)	≥200 (11.1)	140-199 (7.8-11.0)
Random glucose in patients with classic symptoms of diabetes, mg/dL (mmol/L)	≥200 (11.1)	Not specified

Source: Adapted from American Diabetes Association [4]

Prediabetes: Preventing Diabetes

Effectiveness of Nutrition Therapy Interventions

T2D is known to be a progressive disease and although lifestyle interventions are effective at any time in the disease process, they have their greatest impact earlier in the course of the disease; thus, the importance of intervening early with lifestyle approaches in both the prevention and treatment of diabetes.

Genetic factors that remain largely unidentified are a major factor in the development of diabetes; however, there is also a strong role for nongenetic factors. Insulin resistance, a common identifiable risk factor, is a state in which there is low or impaired biological response to insulin and, therefore, greater than normal amounts of insulin are necessary to produce a normal biological response. With insulin resistance, the pancreatic beta cells must increase their insulin secretion (hyperinsulinemia) to maintain normoglycemia. Tests to evaluate insulin resistance measure insulin sensitivity or the ability of insulin to effectively lower glucose levels and are available but not commonly used in clinical practice. Insulin resistance is associated with a higher incidence of T2D, dyslipidemia, hypertension, atherosclerosis disorders, and polycystic ovary syndrome (PCOS). T2D is first diagnosed, however, when there is insufficient endogenous insulin available to maintain euglycemia. As insulin deficiency progresses, medications and eventually insulin will be required to achieve glycemic goals.

Persons with prediabetes progress to T2D at a rate of about 5–10% per year without intervention. Five clinical trials have compared lifestyle interventions with a control group, with risk reduction for type 2 diabetes ranging from 28 to 67%. The Finnish Diabetes Prevention Study (DPS) [8] and the Diabetes Prevention Program (DPP) [9] reduced the incidence of diabetes by 58%, the Da Qing study by 46% [10], the Japanese DPP trial by 67% [11], and the Indian DPP by 30% [12]. Education and support, modest weight loss, and moderate physical activity were commonly implemented interventions. It is encouraging to note that a sustained 43% lower incidence rate over a 20-year period was reported in the Da Qing study [13], a 27% reduction over a 15-year period in the DPP [14], and a 43% reduction over a 7-year period in the Finnish DPS [15]. In the DPP, lifestyle interventions, including a weight loss of 3 kg from baseline, also improved cardiovascular disease (CVD) risk factors—hypertension and lipid profile—compared with placebo and metformin therapy [16]. A cost-effective model suggested that lifestyle interventions as delivered in the DPP are cost effective [17], and actual cost data from the DPP and US Diabetes Prevention Program Outcomes Study confirmed that lifestyle interventions are highly cost effective [18].

Although several trials have shown that medications, such as metformin, α -glucosidase inhibitors, orlistat, and thiazolidinediones (TZD), can also prevent or delay progression from prediabetes to diabetes, the American Diabetes Association (ADA) recommends that only metformin be considered as drug therapy for individuals with prediabetes [4]. Metformin is most effective in individuals with a body mass index (BMI) >35 kg/m² and who are <60 years. Cost, side effects, weight gain, and lack of persistence of effect in some studies are concerns with other medications.

The Community Preventive Services Task Force (funded by the Center for Disease Control) reviewed 53 studies (30 of diet and physical activity programs vs. usual care, 13 of more intensive vs. less intensive programs, and 13 single programs) from 66 programs and concluded that combined diet and physical activity promotion programs are effective at decreasing diabetes incidence and improving cardiovascular disease (CVD) risk factors in persons at increased risk [19]. More intensive programs are most effective. In these programs, dietitians were the primary counselors followed by exercise therapists, nurses, and laypersons.

Nutrition Therapy for Prediabetes

Goals of nutrition therapy for prediabetes emphasize the importance of lifestyle interventions in preventing or reversing the progression of prediabetes to diabetes by selection of food choices that facilitate moderate weight loss and by an increase in physical activity. A modest weight loss (5-10% of body weight) and modest physical activity (30 minutes daily) are the recommended goals. Education and continued support are necessary to accomplish these goals.

Weight Loss Interventions

In both the DPP and Finnish DPS, structured education programs emphasized lifestyle changes, including a reduced fat (\sim 30% of total energy) and energy intake, regular physical activity, and regular participant contact, resulted in a weight loss of 5–7% of baseline weight.

Of importance for weight management is a reduced energy eating pattern that an individual can follow long term. Attendance at group sessions; support from family, peers, or health care professionals; setting realistic weight loss goals; monitoring food intake by food records; and regular physical activity have been shown to assist in accomplishing required lifestyle interventions.

Carbohydrate and Dietary Fats

Although it is often assumed that a high carbohydrate intake contributes to insulin resistance, observational and interventions studies report that high-carbohydrate diets do not adversely affect insulin sensitivity and may have a beneficial effect on insulin sensitivity [20]. In low-carbohydrate diets, energy from carbohydrate is generally replaced with energy from fats, usually saturated fatty acids (SFA). Animal studies and observational studies report a relationship between total dietary fat and greater insulin resistance. Reducing intake of fat, especially SFA, may reduce risk for diabetes by producing an energy-independent improvement in insulin resistance. The effect may result from changes in membrane lipid profiles related to the type of fat ingested, enzymatic activity, insulin signaling, and gene expression [21].

Of concern is the increased consumption of sugar-sweetened beverages (SSB) which includes soft drinks, fruit drinks, iced teas, and energy and vitamin-water drinks. A meta-analysis of 11 studies concluded that individuals consuming one to two servings per day had a 26% greater risk of developing diabetes than those in the lowest quartile (none or less than one serving per month) and a greater risk for metabolic syndrome [22]. A second meta-analysis of 17 cohort studies concluded that consumption of SSB was associated with a greater incidence of T2D, independently of obesity [23]. Therefore, individuals at risk for T2D are encouraged to limit their intake of SSB.

Whole Grains and Fiber

In a number of studies, primarily observational, increased intake of whole grains and dietary fiber, especially cereal fibers, is inversely associated with insulin resistance and risk of type 2 diabetes [24, 25]. Individuals are encouraged to consume at least half of all grains as whole grains and limit the consumption of foods that contain refined grains, especially grain foods that contain solid fats, added sugars, and sodium.

Alcohol

Observational studies suggest a U- or J-shaped association between moderate consumption of alcohol and decreased risk of T2D, coronary heart disease, and stroke [26]. (One drink is commonly defined as 12 oz regular beer, 5 oz wine, or 1.5 oz 80-proof distilled spirits, each of which contains ~15 g alcohol [ethanol].) A meta-analysis of 20 observational studies identified a peak reduction in development of type 2 diabetes at 24 g/day of alcohol among women and 22 g/day among men (~1.5 drinks) [27]. The most recent meta-analysis identified 38 studies, representing 1,902,605 participants and 125,926 cases of type 2 diabetes with peak risk reduction of 18 % occurring between 10 and 14 g/day (~1 drink) and with risks increasing above this level [28]. They note, however, this risk reduction among moderate drinkers may only apply to women and non-Asian men. Although small clinical trials and observational studies have shown light to moderate amounts of alcohol improve insulin sensitivity, heavy consumption of alcohol (greater than ~60 g/day for men and greater than ~50 g/day for women [~4 to 3 drinks/day, respectively]) is associated with increased incidence of diabetes [27].

Vitamin D

Several but not all epidemiological studies have shown an association between low serum 25-hydroxy (OH) vitamin D and risk of type 2 diabetes [29]. However, in a 12-month randomized controlled trial (RCT) in individuals with prediabetes and hypovitaminois D, doses of vitamin D supplementation designed to raise serum 25-OH vitamin D levels into the upper-normal range had no effect on insulin secretion, insulin sensitivity, or the development of diabetes compared to placebo [30].

Mediterranean Diets

Adherence to a Mediterranean-style eating pattern (MED Diet) has been shown to have a beneficial role in decreasing risk for T2D [31–33] as well as being associated with a lower rate of morbidity and mortality caused by CVD, cancer, obesity, and T2D [32]. Therefore, the MED Diet may be a palatable alternative to the low-fat diet implemented in the diabetes prevention trials.

Physical Activity

Increases in physical activity significantly reduce the risk of T2D, independent of weight loss, and improve insulin sensitivity [34, 35]. In a meta-analysis of 20 prospective cohort studies, individuals who regularly engaged in physical activity had an ~30% lower risk of T2D as compared with sedentary individuals even in those who did not lose weight [35]. Protection from diabetes occurs from moderate-intensity activities, such as brisk walking as well as from vigorous physical activity. Exercise increases insulin-stimulated glycogen synthesis through an increased rate of insulin-stimulated glucose transporters and increased glycogen synthase activity [36]. In addition, increased muscle mass may contribute to beneficial effects.

Bariatric Surgery

Studies suggest that for obese subjects, bariatric surgery can reduce the incidence of diabetes. Sjoström et al. published a 10-year data from the Swedish Obese Subjects (SOS) study that included 407 morbidly obese subjects who underwent bariatric surgery and matched controls [37]. At 10 years, the

incidence of diabetes was 24% in the control group and 7% in the bariatric surgery group. Similar results were reported from another study in Sweden in which 1658 obese patients underwent bariatric surgery and 1771 obese patients received usual care [38]. At 15 years, the incidence of T2D was 22% in the control group and 7% in the bariatric surgery group, a relative risk reduction of 78%.

Combining Lifestyle Interventions to Prevent Diabetes

For all individuals, a healthy eating pattern, participation in regular physical activity, maintenance of normal body weight, and moderate alcohol intake lower risk of developing T2D [39]. However, adhering to a combination of these factors reduced the risk of developing diabetes by as much as 84% in women and 72% in men. Although weight was one of the most important factors, the researchers note that even overweight individuals can lower their risk by adopting other healthy lifestyle habits. For individuals with prediabetes, nutrition therapy recommendations for reducing risk of progression to T2D are summarized in Table 8.2.

Type 1 And Type 2 Diabetes: Management of Diabetes

Medical Management

The management of all types of diabetes includes nutrition therapy, physical activity, diabetes education and counseling, monitoring, and medications. An important goal of treatment is to provide the individual with diabetes with the necessary tools to achieve the best possible control of glucose, lipids, and blood pressure to prevent, delay, or manage the microvascular and macrovascular complication while minimizing hypoglycemia and excess weight gain.

The ADA's glycemic treatment goals are listed in Table 8.3 [4]. Achieving goals requires open communication between the health care provider and the person with diabetes and appropriate self-management education and support. Patients can assess day-to-day glycemic control by monitoring of blood glucose. Longer-term glycemic control is assessed by A1C testing. Lipid levels and blood pressure must also be monitored. In adults, a screening lipid profile is recommended at time of first diagnosis, at the initial medical evaluation, and/or at age 40 years and periodically (e.g., every 1–2 years) thereafter [4]. Blood pressure should be measured during every routine visit. Blood pressure goals are listed in Table 8.4 [4].

Table 8.2 Nutrition therapy interventions for prediabetes

Negotiate a reduced energy eating pattern with the individual at risk for diabetes

Promote an increase in physical activity

- Recommend moderate-intensity aerobic physical activity a minimum of 30 min 5 days/week (150 min/week) or vigorous-intensity aerobic physical activity a minimum of 20 min 3 days/week (90 min/week)
- Physical activity should be distribute over at least 3 days/week, with no more than 3 days without activity Muscle-strengthening activities involving all major muscle groups 2 or more days/week are also recommended
- Discourage consumption of sugar-sweetened beverages (soft drinks, fruit drinks, iced teas, and energy and vitaminwater drinks)

Encourage intake of whole grains and foods higher in dietary fiber

Consider implementing a Mediterranean-style eating pattern

Source: From Refs. [8, 9, 20-28, 31-33]

Glycemic control	Criteria
A1C	<7.0 % ^a
Preprandial capillary plasma glucose	80-130 mg/dL ^a (4.4-7.2 mmol/L)
Peak postprandial capillary plasma glucose	<180 mg/dL ^a (<10.0 mmol/L)

 Table 8.3
 Recommendation for glycemic control in nonpregnant adults with diabetes

Source: Adapted from American Diabetes Association [4]

^aMore or less stringent goals may be appropriate for individual patients. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations

 Table 8.4
 Blood pressure goals for people with diabetes

Blood pressure	Criteria	
Systolic blood pressure (SBP)	<140 mmHg ^a	
Diastolic blood pressure (DBP)	<90 mmHg ^b	

Source: Adapted from American Diabetes Association [4]

^aLower SBP targets, such as <130 mmHg, may be appropriate for certain individuals, such as younger patients, if they can be achieved without undue treatment burden ^bLower DBP targets, such as <80 mmHg, may be appropriate for certain individuals, such as younger patients, if they can be achieved without undue treatment burden

Nutrition Therapy

Effectiveness of Nutrition Therapy

The 2015 Academy of Nutrition and Dietetics (Academy) Type 1 and Type 2 Diabetes in Adults Evidence-Based Nutrition Therapy Practice Guidelines(NTPG) report that in adults with T2D, in 18 studies, medical nutrition therapy (MNT) provided by RDNs significantly lowered hemoglobin A1_c (A1C) by 0.3-2.0% at 3 months, and with ongoing MNT support, decreases were maintained or improved for over 12 months [40]. Although nutrition therapy interventions were effective throughout the disease duration, the decreases in A1C levels were the largest in studies in which participants were newly diagnosed and/or had higher baseline A1C levels. Of importance was an individualized nutrition therapy intervention that resulted in a reduced energy intake. A variety of interventions, such as individualized nutrition therapy, energy restriction, carbohydrate counting, portion control, sample menus, exchange lists, simple meal plans, low-fat vegan, were implemented and effective. All resulted in a reduced energy intake.

In adults with T1D, three studies reported that MNT provided by RDNs contributed to significant decreases in A1C levels by 1.0–1.9% at 6 months [40]. Ongoing MNT support resulted in maintenance of the reduced A1C levels at 1 year and in the Diabetes Control and Complications Trial (DCCT) throughout the 6.5 years of the trial. Individualized MNT using carbohydrate counting to determine bolus (premeal or prandial) insulin doses was the primary nutrition therapy intervention.

Studies in adults with T2D report that MNT also resulted in decreases in doses and number of glucose-lowering medications used. In adults with T1D, although the number of insulin injections increased, A1C improved without an increase in total insulin doses. A number of studies also reported improvements in quality of life. Outcomes for weight, lipids, and blood pressure were mixed. Subjects did not have or were not described as having disorders of lipid metabolism or hypertension and were confounded by use of lipid-lowering and blood pressure medications [40].

It is also recommended that people with diabetes receive diabetes self-management education (DSME) and diabetes self-management support (DSMS) [6]. Reviews and meta-analyses report that most DSME leads to clinically important improvements in glycemic control, but they also highlight the absolute need for long-term support [41–43].

Nutrition Therapy Macronutrient Percentages

In the United States, the majority of individuals with diabetes do not eat a low or high carbohydrate diet. Usual carbohydrate intake in adults with T2D in the Look AHEAD (Action for Health in Diabetes) trial was 44 % of total kilocalories [44]; in persons with T1D who were participants in the DCCT intensive treatment arm 46 % [45], and in youth with T1D or T2D, 48 % [46]. Fat intake is \sim 35–40 % of energy intake with the remained (\sim 16–18 %) from protein.

The ADA's review of evidence concluded that there is no most effective mix of carbohydrate, protein, and fat that applies broadly [6]. Macronutrient percentages should be individualized and adjusted to meet metabolic goals and preferences of the person with diabetes.

Although research trials may show benefit from changes in usual macronutrient distribution, the important question remains: can individuals with diabetes implement these recommendations into their lifestyle long-term? Research trials are often of a relatively short duration and frequent support from health professionals is provided during the trial. Of interest are comments from researchers who have conducted a minimum of 1-year clinical trials involving changes in macronutrient distribution. Iqbal et al. conducted a 24-month study in participants with T2D to determine if benefits identified from short-term, intensive low-carbohydrate studies could be achieved using a low-intensity intervention that mimics what is feasible in an outpatient practice [47]. At 6 months, the low-carbohydrate group had decreased carbohydrate intake by 4.7% (35% of kcal) but at 12 months they were back to their baseline intake (40% of kcal) and at 24 months they had increased carbohydrate intake (48% of kcal). The researchers concluded that low-carbohydrate diets may be difficult to sustain and that participants in the two groups consumed similar diets, despite the prescription of markedly different intake. A review of low-carbohydrate diets in people with T2D reported that carbohydrate intake at 1 year in very low carbohydrate diets (<50 g of carbohydrate) ranged from 132 to 162 g [48]. They concluded that very low carbohydrate diets may not be sustainable over a medium to longer term as carbohydrate intake converges toward a more moderate level.

Two researchers compared a high-protein intake (30% of kcal) to either a high-carbohydrate diet [49] or low-carbohydrate diet [50]. Krebs et al. reported that the prescribed protein of 30% was achieved in only 12 of 2017 (6%) of participants and noted that this "highlights how difficult it is to achieve and maintain prescribed change...individuals trend back to habitual intake over time" [49]. Larsen et al. commented: "under real-world conditions, variations in food selection and adherences are likely to attenuate the effect previously demonstrated in controlled feeding studies" [50]. The studies provide support for the conclusion that "how much" individuals eat is more important than "what."

However, because all three macronutrients require insulin for metabolism and influence the attaining of goals of nutrition therapy, including healthy eating, they still must be addressed. The amount rather than the type of carbohydrate and available insulin is the primary determinant of postprandial glycemia [6]. Therefore, monitoring carbohydrate intake, whether by carbohydrate counting or experience-based estimation, remains a key strategy in achieving glycemic control [6, 40]. Carbohydrate counting focuses on the total amount of carbohydrate in foods and not the source (15 g carbohydrate choice). An eating plan for adult women often begins with three to four carbohydrate choices per meal, for adult men four to five carbohydrate choices per meal, and, if desired, zero to one carbohydrate choice for a snack.

Macronutrients and Insulin Adjustments

For individuals taking insulin by injection or by insulin pumps, the bolus or premeal insulin dose covers the need of the meal carbohydrate and for three meals is approximately half of the total daily insulin dose. Therefore, the basal or background insulin dose covers the need of protein and fat for insulin as well as other insulin needs.

Insulin-to-carbohydrate ratios are used to match bolus doses of rapid-acting insulin taken before a meal to planned carbohydrate intake, which is determined by carbohydrate counting. A study done in young people using intensive insulin therapy reported that small inaccuracies in carbohydrate intake at a meal (± 10 g) do not increase the risk of hypo- or hyperglycemia and, therefore, it is not essential to count carbohydrate in grams, carbohydrate choices (servings) can be used [51]. However, a 20 g decrease from recommended carbohydrate intake resulted in one of three youths (31%) experiencing hypoglycemia, whereas, a 20 g increase was more likely to cause a blood glucose response ≥ 216 mg/ dL (12 mmol/L) compared with other meals [52].

If the premeal glucose level is not in the target goal range, rapid-acting insulin doses need to be added or subtracted from the usual bolus insulin dose. This is called the blood glucose correction insulin, also known as the insulin sensitivity factor, and is used to bring blood glucose levels back into the target range. Table 8.5 illustrates commonly used insulin-to-carbohydrate ratios and blood glucose correction doses (insulin sensitivity factors) [53].

Usually consumed meals contain moderate amounts of protein and fat and this is already accounted for by usual basal and bolus insulin doses. However, research using excessively high intakes of fat (or protein) has documented the need for additional insulin [54]. A study in young people concluded that only large meals containing 75 and 100 g protein raised glucose levels in the late postprandial (180–130 min) when compared to control [55]. Meals containing 12.5, 25, and 50 g protein did not significantly increase glucose levels. The effect of larger amounts of protein and fat is additive. Responses vary greatly, however, and research at this time does not provide guidelines for insulin adjustments [55].

Carbohydrate Intake

Because the amount of carbohydrate consumed and available insulin is the most important factor influencing postmeal glycemic response [6], monitoring carbohydrate intake, whether by carbohydrate counting or experience-based estimation, remains a key strategy in achieving glycemic control. As noted, for adults with T1D or T2D on multiple daily injections (MDI) of insulin or insulin pump therapy, carbohydrate counting is used to determine insulin-to carbohydrate ratios [6]. For adults on fixed insulin doses or on insulin secretagogues, carbohydrate consistency (timing and amount) is emphasized. For adults on MNT alone or on diabetes medications other than insulin secretagogues, a simple diabetes healthful eating plan may be appropriate.

Table 8.5 General guidelines for insulin-to-carbohydrate ratios and blood glucose correction insulin factors

	Insulin-to-carbohydrate ratio	Blood glucose correction insulin
Adult (normal weight)	1:10 to 1:15	1:40 to 1:50
Adult (overweight)	1:7 to 1:10	1:30 to 1:40
Adult (obese)	1:5	1:25

Source: American Diabetes Association, Academy of Nutrition and Dietetics [53]

Insulin-to-carbohydrate ratios are 1 unit of rapid-acting insulin for grams of planned carbohydrate intake. The blood glucose correction insulin is the approximate glucose lowering in mg/dL from an added 1 unit of rapid-acting insulin. The insulin dose is taken before the meal is eaten

For good health, carbohydrate intake from vegetables, fruits, whole grains, legumes, and dairy products is advised over intake from other carbohydrate sources, especially those that contain added fats, sugars, or sodium. Of importance are appropriate amounts and portion sizes of carbohydrate foods. Furthermore, negotiating with the individual with diabetes as to what they are willing and able to do is essential.

Glycemic Index

The types of carbohydrate, especially the role of the glycemic index (GI), have been another area of controversy and the definition of the GI is confusing. The GI measures the relative area under the postprandial glucose curve (AUC) comparing 50 g of a digestible carbohydrate from a test food to a 50 g standard, usually glucose [56]. It does not measure how rapidly blood glucose levels increase after eating different carbohydrate foods, which is the definition often given to the public. The second definition implies that a high-GI food produces a rapid, high glucose peak while a low-GI food produces a more gradual and sustained glucose response. A review compared the glucose curve from different types of carbohydrate categories (e.g., breads, cereals, potatoes, pasta, fruit, and fruit juice) of low and high GI foods in persons without diabetes. No statistical difference in the glucose response curve from different foods or food categories was shown. Glucose peaks occurred consistently at ~30 min, regardless of whether the food was categorized as low GI, medium GI, or high GI, with a modest difference in glucose peak values between high-GI and low-GI foods [56]. The authors also concluded that low-GI foods do not produce a slower rise in blood glucose nor do they produce an extended, sustained glucose response.

The ADA systematic review of macronutrients concluded that, in general, there is little difference in glycemic control and CVD risk factors between low GI and high GI or other diets. A slight improvement in glycemia may result from lower GI diets; however, confounding by a higher fiber intake is not accounted for [57]. As with carbohydrates, most individuals with diabetes likely consume a moderate GI diet, and it is unknown if reducing the usual GI by a few percentage points will result in long-term improved glycemic control.

Protein

Gram for gram protein is reported to require similar amounts of insulin for metabolism as do carbohydrates. Although essential amino acids undergo gluconeogenesis in the liver, the glucose does not enter the general circulation but instead is stored as glycogen. Adding protein to bedtime snacks is often recommended to prevent overnight hypoglycemia or to be added to the treatment of hypoglycemia, but adding protein to prevent hypoglycemia is not beneficial (because as noted above, the glucose from gluceoneogenesis does not enter the general circulation) and therefore should not be recommended to persons with diabetes [6].

Also of interest is the role of protein restriction in the treatment of diabetic kidney disease. Although reducing protein intake below usual intake in individuals with micro- or macroalbuminuria may reduce albuminuria, it does not alter the glomerular filtration rate and is not recommended [6].

Fats

Evidence is also inconclusive for an ideal amount of total fat intake for people with diabetes [6]. The type of fatty acids consumed is more important than total fat in the diet. Individuals with diabetes are encouraged to select unsaturated fats in place of saturated and trans fatty acids and are encouraged to moderate their fat intake to be consistent with weight management goals.

As noted earlier in persons with prediabetes, of concern in persons with diabetes is the effect of total fat, especially saturated fat, on insulin sensitivity. Epidemiologic data and clinical trials have reported that long-term higher total fat intake results in greater whole-body insulin resistance [58, 59]. Although not as well studied in persons with diabetes, reducing saturated fat has been shown to improve insulin sensitivity [60, 61]. The impact of long-term intake of saturated fatty acids on insulin resistance is important because as people with diabetes decrease their intake of carbohydrate, they usually increase their fat intake, especially saturated fat [62].

The ADA 2013 nutrition therapy [6] and the Academy 2015 Diabetes Type 1 and Type 2 Evidence-Based Nutrition Practice Guidelines [40] recommendations are summarized in Table 8.6.

Diabetes and Weight Management

Strong evidence exists for the benefits of moderate weight loss for the prevention of T2D. Weight loss interventions (WLI) have also been shown to be effective in improving glycemic control in individuals with newly diagnosed T2D [63, 64]. The benefit of weight loss interventions (WLI) in T2D of longer duration is controversial. To better understand the results of WLI, a systematic review and meta-analysis was conducted to answer the questions: in overweight and obese adults with T2D, what are the outcomes on A1C, lipids, and blood pressure from WLI resulting in weight losses greater than or less than 5 % at 12 months, and what are the weight and metabolic outcomes from differing amounts of macronutrients in WLI [65]? Eleven trials (eight compared two WLI and three compared a WLI to a usual care/control group) with 6754 participants met the study criteria. At 12 months, 17 WLI groups reported weight losses less than 5 % (1.9-4.8 kg) and nonsignificant benefits on A1C, lipids, or blood pressure. Only two WLI study groups, a MED Diet in newly diagnosed adults and the intensive lifestyle intervention in the Look AHEAD trial resulted in weight loss greater than 5%. Both included regular physical activity and frequent contacts with health professionals and reported significant benefits on A1C, lipids, and blood pressure. Five trials (10 study groups) compared WLI of differing amounts of macronutrients and reported nonsignificant differences in weight loss, A1C, lipids, or blood pressure. Thus, a weight loss >5% (~6 kg) appears necessary for beneficial metabolic outcomes. Achieving this level of weight loss requires intense interventions, including energy restriction, regular physical activity, and frequent contact with health professionals.

Furthermore, it appears difficult for persons with diabetes to lose weight. In a systematic review of 80 weight loss studies with 26,455 participants, primarily without diabetes, the average weight loss was 8 % (7.5 kg) from baseline [66] compared to <5 % in the majority of studies in participants with type 2 diabetes [65]. Factors that contribute to an individual's inability to loss and maintain weight loss include low socioeconomic status, an unsupportive environment, and, very importantly, physiologic changes (e.g., compensatory changes after weight loss in circulating hormones that encourage weight regain and adaptive thermogenesis) [67]. Therefore, the emphasis of nutrition therapy for individuals with T2D should be on a reduced energy intake for improved glycemic control, not the scale (in some it may lead to weight loss, in some it may maintain weight loss, and in some it may prevent weight gain), regular physical activity, and support for lifestyle changes.

Weight Loss Medications

Five obesity medications are approved for long-term use in the US: (1) orlistat, a gastrointestinal lipase inhibitor; (2) lorcaserin, the 5-hydroxytryptophan, 5-HT2c, (2c subscript) serotonin receptor agonist; (3) phentermine/topiramate extended release (ER), combining a sympathomimetic and an anticonvulsant; (4) naltrexone/bupropion ER, combining a dopamine/noradrenaline reuptake inhibitor and an opioid receptor antagonist; and (5) a high-dose (3-mg) preparation of the glucagon-like

Topic	Recommendation	Evidence rating
Energy intake	For overweight or obese adults with diabetes a reduced energy intake, healthful eating plan, with a goal of weight loss, weight loss maintenance, and/or prevention of weight gain is encouraged.	Strong, A
	For appropriate-weight adults with diabetes a healthful eating plan with a goal of weight maintenance and/or prevention of weight gain is recommended.	Consensus
Macronutrient composition	Macronutrient distribution should be individualized based on a healthful eating plan with appropriate energy intake and based on current eating patterns, preferences, and metabolic goals.	Fair, E
Eating patterns	A variety of eating patterns are acceptable for the management of diabetes.	Fair, E
Carbohydrates	Monitoring carbohydrate intake, whether by carbohydrate counting or experience-based estimation remains a key strategy in achieving glycemic control.	В
	For good health, carbohydrate intake from vegetables, fruits, whole grains, legumes, and dairy products are encouraged over intake from other carbohydrate sources, especially those that contain added fats, sugars, and sodium.	В
	Carbohydrate counting using insulin-to-carbohydrate ratios is recommended for adults using multiple daily injections of insulin or insulin pump therapy	Strong
	Carbohydrate consistency (timing and amount) is recommended for adults on fixed insulin doses or on insulin secretagogues.	Fair
	Carbohydrate counting alone, plate method/portion control/simplified meal plan, food lists/carbohydrate choices, based on adults abilities, preferences, and management goals can be recommended for adults on nutrition therapy alone or on diabetes medications other than insulin secretagogues.	Fair
Fiber and whole grains	Fiber and whole grain foods recommendations for people with diabetes are the same as for the general public.	Fair, C
Glycemic Index (GI) and glycemic load (GL)	Lowering GI and/or GL may or may not have a significant effect on glycemic control; studies longer than 12 weeks report no significant impact of GI and/or GL, independent of weight loss, on A1C.	Fair, C
Nonnutritive sweeteners (NNS)	Use of NNS has the potential to reduce overall calorie and carbohydrate intake if substituted for caloric sweeteners without compensation by intake of additional calories from other foods.	Fair, B
Protein	Adding protein to meals and/or snacks does not prevent or assist in treatment of hypoglycemia; ingested protein can increase insulin response without increasing plasma glucose concentrations.	Fair, B
	For people with diabetes and diabetic kidney disease, reducing the amount of protein below usual intake is not recommended as it does not alter glycemic measures, cardiovascular risk measures, or the course of glomerular filtration rate decline.	Strong, A
Saturated fat, dietary cholesterol, and trans-fat	A cardioprotective eating pattern, within the recommended energy intake, and the same intake of saturated fat and cholesterol as recommended for the general population is encouraged.	Strong, C
Micronutrients and herbal supplements	There is no clear evidence of benefit from supplementation in people with diabetes who do not have underlying deficiencies. Routine supplementation with antioxidants, other micronutrients, and herbal supplements is not advised.	Fair, C
Alcohol	If adults with diabetes choose to drink alcohol they should do so in moderation (one drink per day or less for woman and two drinks per day or less for men).	Е

 Table 8.6
 Nutrition therapy for diabetes. Academy of Nutrition and Dietetics (Academy) recommendations ratings and American Diabetes Association's (ADA) level of evidence ratings

(continued)

 Table 8.6 (continued)

Торіс	Recommendation	Evidence rating
Sodium	The recommendation for the general public to reduce sodium intake to less than 2300 mg/day is also appropriate for people with diabetes.	В
	For individuals with both diabetes and hypertension, further reduction in sodium intake should be individualized.	В

Source: Evert et al. [6] and Academy of Nutrition and Dietetics Evidence Analysis Library [40] The Academy of Nutrition and Dietetics (Academy) recommendations ratings are Strong, Fair, Weak, Consensus, Insufficient Evidence. American Diabetes Association's (ADA) level of evidence ratings that support each nutrition therapy recommendation use the letters A, B, C, or E

peptide 1 (GLP-1) receptor agonist liraglutide [68]. Table 8.7 summarizes the trials done in persons with T2D comparing the weight loss medication to a placebo and the 52- to 56-week weight loss and A1C outcomes [69–73]. All subjects were placed on lifestyle interventions and then randomized to placebo versus weight-loss medication. When used as an adjunct to lifestyle intervention, these agents can assist people by enhancing their ability to comply with a reduced-energy eating plan and studies report that people can sustain loss for a longer period of time [68]. In people with T2D, drug-assisted weight loss resulted in better glycemic control, while reducing the number and doses of glucose-lowering medications and generally lowering blood pressure and improving lipids. A recent phentermine/topiramate ER study in people with prediabetes reported the drug-assisted weight loss reduced the incidence of progression to T2D over 108 weeks by 79% compared to placebo [74].

Bariatric Surgery

The best long-term surgical weight loss data come from the SOS study, a prospective (>90% followup rate) evaluating effects of bariatric surgery compared with nonsurgical weight management [75]. At 15 years, the weight loss was $27 \pm 12\%$ for gastric bypass, $18 \pm 11\%$ for vertical-banded gastroplasty, and $13 \pm 14\%$ for gastric banding compared with a slight weight gain for controls. In the SOS study, the remission rate for T2D was 72% at 2 years and 36% at 10 years compared with 21% and 13%, respectively, for the nonsurgical control subjects [37].

The safety of bariatric surgery and selection of patients most likely to benefit from the surgery continue to be primary concerns in the determination of whether the potential benefits outweigh the surgery risks [76]. The Scientific Statement from the American Heart Association and the ADA concluded that bariatric surgery may be particularly suitable for patients with T2D and severe obesity (BMI \geq 35 kg/m²) because these patients may benefit from obesity comorbidity improvement and significantly improved glycemic control compared with medical therapy alone [76]. They note that the durability of these metabolic improvements (A1C and CVD risk) remains to be determined.

Physical Activity

Physical activity should be an integral part of the treatment plan for persons with diabetes. Exercise helps all persons with diabetes improve insulin sensitivity, reduce CVD risk factors, control weight, and improve well-being [4]. Given appropriate guidelines, the majority of people with diabetes can exercise safely. The activity plan will vary, depending on interest, age, general health, and level of physical fitness.

	Orlistat				
	(Xenical;	Lorcaserin	Phentermine/topiramte	Naltrexone/bupropion	Liraglutide
	Alli ^a) [69]	(Belviq) [70]	(Qnexa) [71]	(Contrave) [72]	(Saxenda) [73]
Weight los	s (%)				
Drug	6.2	4.5	9.6	5.0	6.0
Placebo	4.3	1.5	2.6	1.8	2.0
A1C chang	ge (%)				
Drug	-0.3	-0.9	-1.6	-0.6	-1.3
Placebo	+0.2	-0.4	-1.2	-0.1	-0.3

Table 8.7 Weight-loss medications in people with T2D: weight loss and glycemic control

Source: Adapted from: Cefalu et al. [68]

Brand names are in parenthesis; study lengths: 52-56 weeks

^aOver-the-counter brand name

Despite the increase in glucose uptake by muscles during exercise, glucose levels change little in individuals without diabetes [77]. Muscular work causes insulin levels to decrease while counterregulatory hormones (primarily glucagon) rise. As a result, the increased glucose needed by exercising muscles is matched with increased glucose production by the liver.

In persons with T1D, the glycemic response to exercise varies depending on overall diabetes control, glucose and insulin levels at the start of exercise, intensity and duration of the exercise, previous food intake, and previous conditioning [77]. The most common problem during and after exercise is hypoglycemia, which must be treated with carbohydrate in addition to the carbohydrate prescribed for exercise. However, high-intensity exercise and competition stress may result in hyperglycemia after exercise.

In persons with T2D, blood glucose control can improve with physical activity, largely because of decreased insulin resistance and increased insulin sensitivity, which results in increased peripheral use of glucose not only during but also after the activity [78]. This exercise-induced enhanced insulin sensitivity occurs independent of an effect on body weight. Exercise also decreases the effects of counter-regulatory hormones, reducing hepatic glucose output, and contributing to improved glucose control.

Physical Activity Recommendations

Adults with diabetes should be advised to perform at least 150 min/week of moderate-intensity aerobic physical activity (50–70% of maximum heart rate), spread over at least 3 days/week with no more than 2 consecutive days without exercise [4]. In the absence of contraindications, adults with T2D are encouraged to perform resistance training at least twice per week. All individuals should be encouraged to reduce sedentary time, particularly by breaking up extended amounts of time (>90 min) spent sitting. Children with diabetes or prediabetes should be encouraged to engage in at least 60 min of physical activity each day.

Routine screening for who should or should not begin to exercise is not recommended [4]. Providers should use clinical judgment in this area. High-risk patients should be encouraged to start with short periods of low-intensity exercise and increase the intensity and duration slowly.

Exercise Precautions for Persons with Type 2 Diabetes

Persons with T2D may have a lower maximum volume of oxygen (VO 2max) and therefore require a more gradual training program. Rest periods may be needed, but this does not impair the training effect from physical activity. Heart rates during exercise may not increase normally because of autonomic neuropathy or medications, such as for blood pressure, and individuals may need to learn to use perceived exertion as a means of determining exercise intensity.

Exercise Precautions for Persons Taking Insulin and/or Insulin Secretagogues

Variability of glucose responses to exercise contributes to difficulty in giving precise guidelines for exercising safely for persons using insulin or insulin secretagogues. Frequent blood glucose monitoring before, during, and after exercise helps individuals identify their response to physical activities. For individuals on these therapies, to prevent hypoglycemia, added carbohydrate should be ingested if pre-exercise glucose levels are <100 mg/dL (5.6 mmol/L) [4]. With unplanned activity or when insulin dosage has not been adjusted, extra carbohydrate ingestion may be necessary. In general, every 60 minutes of increased moderate-intensity exercise, such as tennis, swimming, or jogging, requires an additional 15 g of carbohydrate, either prior to or after the exercise [79]. For more strenuous exercise, such as 1–2 h of sport activities (basketball, football, soccer, etc.) or strenuous bicycling, 30 g of carbohydrate per hour may be needed. Furthermore, prebedtime blood glucose levels should be higher than 130 mg/dL (7.2 mmol/L) on days of afternoon or evening activity.

For planned activities, adjustments in insulin dosage also help prevent hypoglycemia that can occur most often with moderate to strenuous activity lasting more than 45–60 min [79]. For most persons, a modest decrease (of about 1–2 units or approximately one-half the usual dose) in the rapid-acting insulin acting during the period of the exercise is a good starting point. For prolonged vigorous exercise, a larger decrease in the total daily insulin dosage may be necessary. After exercise, insulin dosing may also need to be decreased.

Diabetes Education/Counseling and Support

DSME and DSMS are the ongoing processes of facilitating persons with diabetes to attain the knowledge, skill, and ability necessary for successful diabetes management. The overall objectives are to support the individual's informed decision making, self-care behaviors, problem solving, and active collaboration with the health care team to improve clinical outcomes, health status, and quality of life in a cost-effective manner [80]. The ADA recommends that persons with diabetes receive DSME and DSMS according to the national standards for DSME and DSMS when diabetes is first diagnosed and as needed thereafter [4]. Effective nutrition therapy interventions may be an individualized session or a component of a comprehensive group diabetes education session [6].

Monitoring

Several methods are available to assess the effectiveness of the diabetes management plan, including nutrition therapy, on glycemic control: self-monitoring of blood glucose (SMBG), continuous glucose monitoring (CGM), and A1C. SMBG and, for some individuals, CGM are used on a dayto-day basis to manage diabetes effectively and safely; however, measurement of A1C provides the best index of overall diabetes control. Lipids and blood pressure must also be monitored on a regular basis.

Pharmacological Therapy and Lifestyle Recommendations

Pharmacological Therapy for T1D

Persons with T1D should be treated with multiple-dose insulin (MDI) injections (three to four injections per day of basal and prandial insulin) or continuous subcutaneous insulin infusion (CSII), commonly referred to as insulin pump therapy [4]. They also need education on how to match prandial insulin dose to carbohydrate intake, premeal blood glucose, and anticipated activity.

Pharmacological Therapy for T2D

It is important that persons with T2D understand that T2D is a progressive disease. This will help them to understand and accept changes in medications that occur over time. The need for medications and insulin is not a "diet failure" or "medication failure" but rather a failure of the insulin secreting capacity of the beta cells.

Interventions at the time of diagnosis include healthy eating (nutrition therapy, weight control), increased physical activity, and diabetes education [81]. Metformin is the preferred initial pharmacological agent for T2D. If A1C target goals are not reached after ~3 months, a second oral agent (sulfonylurea, thiazolidinedione [TZD], dipeptidyl peptidase 4 inhibitor [DPP-4], sodium-glucose transporter 2 [SGLT-2] inhibitor), a glucagon-like peptide 1 (GLP-1) agent, or insulin is added. If A1C goals are not reached after another ~3 months, a three-drug intervention is implemented. If this combination therapy that includes a long-acting insulin does not achieve A1C goals, a more complex insulin therapy involving MDI, usually in combination with one or more noninsulin agents, is implemented.

A patient-centered approach is stressed, including patient preferences, cost, and potential side effects [4]. The overall objective is to achieve and maintain glycemic control and change interventions, including the use of insulin, in a timely progression when therapeutic goals are not being met. Circumstances that require the use of insulin in T2D, other than failure to achieve adequate control with glucose-lowering medications, include periods of acute injury, infection, extreme heat exposure, surgery, or pregnancy [4].

Pharmacological Therapy for Lipids

To improve lipid profiles, the following options are recommended: lifestyle modification focusing on the reduction of saturated fat, trans-fat, and cholesterol intake; increase of n-3 fatty acids, viscous fiber, and plant stanol/sterols, weight loss (if indicated); and increased physical activity [4]. However, evidence does not support recommending omega-3 supplements for people with diabetes [6]. Statin therapy should be added to lifestyle therapy, regardless of baseline lipid levels, for patients with diabetes and overt CVD and without CVD who are over the age of 40 years and have one or more CVD risk factors. For lower-risk patients, statin therapy is often considered as well.

Pharmacological Therapy for Blood Pressure

Lifestyle therapy for elevated blood pressure consists of weight loss, if overweight; DASH-style dietary pattern including reducing sodium and increasing potassium intake; moderation of alcohol intake; and increased physical activity [4]. Pharmacological therapy begins with a regimen that includes either an angiotensin-converting enzyme (ACE) inhibitor or an angiotensin receptor blocker (ARB). However, multiple-drug therapy (two or more agents at maximal doses) is generally required to effectively treat high and low blood pressure.

T1D and T2D Management Summary

For effective diabetes management, lifestyle recommendations must be integrated into the management plan. Clinical trials and epidemiological studies provide evidence for the beneficial impact of diabetes nutrition therapy. For persons with T1D, use of carbohydrate counting to determine insulinto-carbohydrate ratios for mealtime insulin doses is the most effective nutrition therapy strategy. Individuals on insulin who do not use this intervention do best if they are consistent in their amount and timing of carbohydrate intake. For persons with T2D, an individualized reduced energy intake is the most effective nutrition therapy strategy.

Conclusion

Lifestyle medicine for prediabetes and diabetes includes nutrition therapy, physical activity, education/counseling and support, and monitoring. Strong evidence supports the effectiveness of nutrition therapy across the continuum of diabetes management—prevention to management of diabetes and complications. For persons with prediabetes, lifestyle medicine involves a reduced energy intake leading to modest weight loss, regular physical activity, education, and support. For persons with diabetes, lifestyle medicine involves nutrition therapy based on collaboration between health professionals and persons with diabetes to determine lifestyle strategies and goals the persons with diabetes are willing and able to implement. Physical activity is encouraged, education/counseling and support are essential, monitoring must be done to determine if lifestyle and medical goals are being met, and integration of nutrition therapy interventions and medications is important.

References

- 1. IDF Diabetes Atlas 6th edition, Update 2014. Available at: www.idf.org/diabetesatlas.
- Menke A, Casagrande S, Geiss L, Cowie CC. Prevalence of and trends in diabetes among adults in the United States, 1988–2012. JAMA. 2015;314:1021–9. doi:10.1001/jama.2015.10029.
- Dabelea D, Mayer-Davis EJ, Saydah S, Imperatore G, Linder B, Divers J, et al. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. JAMA. 2014;311:1778–86. doi:10.1001/ jama.2014.3201.
- American Diabetes Association. Standards of medical care in diabetes—2016. Diabetes Care. 2016;39 Suppl 1:S6– S107. doi:10.2337/dc16-5001.
- Gregg EW, Li Y, Wang J, Burrows NR, Ali MK, Rolka D, Williams DE, Geiss L. Changes in diabetes-related complications in the United States, 1990–2010. N Engl J Med. 2014;370:1514–23. doi:10.1056/NEJMoa1310799.
- Evert AB, Boucher JL, Cypress M, Dunbar SA, Franz MJ, Mayer-Davis EJ, et al. Nutrition therapy recommendations for the management of adults with diabetes. Diabetes Care. 2013;36:3821–42. doi:10.2337/dc13-2042.
- Robbins JM, Thatcher GE, Webb DA, Valdmanis VG. Nutritionist visits, diabetes classes, and hospitalization rates and charges: the Urban Diabetes Study. Diabetes Care. 2008;31:655–60.
- Tuomilehto J, Lindström J, Eriksson JG, Valle TT, Hämäläinen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med. 2001;344:1343–50.
- The Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle modifications or metformin. N Engl J Med. 2002;346:393–403.
- Pan XR, Li GW, Hu YH, Wang WY, An ZX, Hu ZX, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. Diabetes Care. 1997;20:537–44.
- Kosaka K, Noda M, Kuzuya T. Prevention of type 2 diabetes by lifestyle intervention: a Japanese trial in IGT males. Diabetes Res Clin Pract. 2005;67:152–62.
- 12. Ramachandran A, Snehalatha C, Mary S, Mukesh B, Bhaskar AD, Vijay V, Indian Diabetes Prevention Programme (DPP). The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (DPP-1). Diabetologia. 2006;49:289–97.
- Li F, Zhang P, Wang J, Gregg EW, Yang W, Gong Q, et al. The long-term effect of lifestyle interventions to prevent diabetes in the China Da Qing Diabetes Prevention Study: a 20-year follow-up study. Lancet. 2008;371:1783–9. doi:10.1016/S0140-6736(08)60766-7.

- 8 Nutrition Therapy for the Prevention and Treatment of Prediabetes and Diabetes
- 14. Diabetes Prevention Program Research Group. Long-term effects of lifestyle intervention or metformin on diabetes development and microvascular complications over 15-year follow-up: the Diabetes Prevention Program Outcomes Study. Lancet Diabetes Endocrinol. 2015. doi:http://dx.doi.org/10.1016/S2213-8587(15)00291-0.
- Lindström J, Ilanne-Parikka P, Peltonen M, Aunola S, Eriksson JG, Hemiö K, et al. Sustained reduction in the incidence of type 2 diabetes by lifestyle intervention: follow-up of the Finnish Diabetes Prevention Study. Lancet. 2006;368:1673–9.
- Ratner R, Goldberg R, Haffner S, Marcovina S, Orchard T, Fowler S, Temprosa M, Diabetes Prevention Program Research Group. Impact of lifestyle intervention metformin therapy on cardiovascular disease risk factors in the Diabetes Prevention Program. Diabetes Care. 2005;28:888–94.
- Herman WH, Hoerger TJ, Brandle M, Hicks K, Sorensen S, Zhang P, et al. The cost-effectiveness of lifestyle modification or metformin in preventing type 2 diabetes in adults with impaired glucose tolerance. Ann Intern Med. 2005;142:323–32.
- Ackermann RT, Finch EA, Brizendine E, Zhou H, Marrero DG. Translating the Diabetes Prevention Program into the community. The DEPLOY Pilot Study. Am J Prev Med. 2008;35:357–63.
- Balk EM, Earley A, Raman G, Avendano EA, Pittas AG, Remington PL. Combined diet and physical activity promotion programs to prevent type 2 diabetes among persons at increased risk: a systematic review for the Community Prevention Services Task Force. Ann Intern Med. 2015. doi:10.7326/M15-0452.
- McClenaghan NH. Determining the relationship between dietary carbohydrate and insulin resistance. Nutr Res Rev. 2005;18:222–40.
- 21. Riserus U, Willett WC, Hu FB. Dietary fats and prevention of type 2 diabetes. Prog Lipid Res. 2009;48:45–51.
- 22. Malik VS, Popkin BM, Bray GA, Després J-P, Willett WC, Hu FB. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes. Diabetes Care. 2010;33:2477–83.
- Imamura F, O'Connor L, Ye Z, Mursu J, Hayashino Y, Bhupathiraju SN, Forouhi NG. Consumption of sugarsweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type 2 diabetes: systematic review, meta-analysis, and estimation of population attributable fraction. BMJ. 2015;351:h3575. doi:10.1136/bmj. h3576.
- Nettleton JA, McKeown NM, Kanoi S, Lemaitre RN, Hivert MF, Ngwa J, et al. Interactions of dietary whole-grain intake with fasting glucose- and insulin-related genetic loci in individuals of European descent. A meta-analysis of 14-cohort studies. Diabetes Care. 2010;33:2684–91.
- 25. The Inter Act Consortium. Dietary fibre and incidence of type 2 diabetes in eight European countries: the EPIC-InterAct Study and a meta-analysis of prospective studies. Diabetologia. 2015;58:1394–408. doi:10.1007/ s00125-015-3585-9.
- Howard AA, Amsten JH, Gourevitch MN. Effect of alcohol consumption on diabetes mellitus. A systematic review. Ann Intern Med. 2004;140:211–9.
- Baliunas DO, Taylor BJ, Irving H, Roerecke M, Patra J, Mohapatra S, Rehm J. Alcohol as a risk factor for type 2 diabetes: a systematic review and meta-analysis. Diabetes Care. 2009;32:2123–32.
- Knott C, Bell S, Britton A. Alcohol consumption and the risk of type 2 diabetes: a systematic review and doseresponse meta-analysis of more than 1.9 million individuals from 38 observational studies. Diabetes Care. 2015;38:1804–12. doi:10.2337/dc15-0710.
- 29. Mitri J, Muraru MD, Pittas AG. Vitamin D and type 2 diabetes: a systematic review. Eur J Clin Nutr. 2011;65:1005–15.
- Davidson MB, Duran P, Lee ML, Friedman TC. High-dose vitamin D supplementation in people with prediabetes and hypovitaminosis. Diabetes Care. 2013;36:260–6.
- Kastorini CM, Kastorini CM, Panagiotakos DB. Dietary patterns and prevention of type 2 diabetes; from research to clinical practice; a systematic review. Curr Diabetes Rev. 2009;5:221–7.
- Martínez-González MÁ, de la Fuente-Arrilaga C, Nunez-Cordoba JM, Basterra-Gortari FJ, Beunza JJ, Vazquez Z, et al. Adherence to Mediterranean diet and risk of developing diabetes: prospective cohort study. BMJ. 2008;336:1348–51.
- 33. Salas-Salvadó J, Bulló M, Babio N, Martinez-González MA, Ibarrola-Jurado N, Basora J, et al. Reduction in the incidence of type 2 diabetes with the Mediterranean diet. Results of the PREDIMED-Reus nutrition intervention randomized trial. Diabetes Care. 2011;34:14–9.
- Duncan GE, Perri MG, Teriaque DW, Hutson AD, Eckel RH, Stacpoolw PW. Exercise training without weight loss, increases insulin sensitivity and postheparin plasma lipase activity in previously sedentary adults. Diabetes Care. 2003;26:557–62.
- Jeon CY, Lokken RP, Hu FB, van Dam RM. Physical activity of moderate intensity and risk of type 2 diabetes. A systematic review. Diabetes Care. 2007;30:744–52.
- Perseghin G, Price TB, Petersen KF, Roden M, Cline GW, Gerow K, et al. Increased glucose transportphosphorylation and muscle glycogen synthesis after exercise training in insulin-resistant subjects. N Engl J Med. 1996;335:1357–62.

- Sjöström L, Lindroos A-K, Peltonen M, Torgerson J, Bouchard C, Carlsson B, et al. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004;351:2683–93.
- Carlsson LM, Peltonen M, Ahlin S, Anveden A, Bouchard C, Carlsson B, et al. Bariatric surgery and prevention of type 2 diabetes in Swedish obese subjects. N Engl J Med. 2012;367:695–704.
- Reis JP, Loria CM, Sorlie PD, Park Y, Hollenbeck A, Schatzkin A. Lifestyle factors and risk of new-onset diabetes: a population-based cohort study. Ann Intern Med. 2011;155:292–9.
- 40. Academy of Nutrition and Dietetics Evidence Analysis Library. Available at: http://www.andeal.org. Accessed November 2015.
- 41. Pilary J, Armstrong MJ, Butalia S, Donovan LE, Sigal RJ, Vandermeer B, et al. Behavioral programs for type 2 diabetes mellitus: a systematic review and network meta-analysis for effect moderation. Ann Intern Med. 2015. doi:10.7326/M15-1400.
- Pilary J, Armstrong MJ, Butalia S, Donovan LE, Sigal RJ, Chordiya P, et al. Behavioral programs for type 1 diabetes mellitus. A systematic review and meta-analysis. Ann Intern Med. 2015. doi:10.7326/M15-1399.
- 43. Powers MA, Bardsley J, Cypress M, Duker P, Funnell MM, Hess Fischl A, et al. Diabetes Self-management Education and Support in Type 2 Diabetes: a joint position statement of the American Diabetes Association, the American Association of Diabetes Educators, and the Academy of Nutrition and Dietetics. Diabetes Care. 2015;38:1372–82. doi:10.2337/dc15-0730.
- 44. Vitolins MZ, Anderson AM, Delanhanty L, Raynor H, Miller GD, Mobley C, et al. Look AHEAD Research Group. Action of Health in Diabetes (Look AHEAD) trial: baseline evaluation of selected nutrients and food group intake. J Am Diet Assoc. 2009;109:1367–75.
- 45. Delahanty LM, Nathan DM, Lachin JM, Hu FB, Cleary PA, Ziegler GK, et al. for the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications. Association of diet with glycated hemoglobin during intensive treatment of type 1 diabetes in the Diabetes Control and Complications Trial. Am J Clin Nutr. 2009;89:518–24.
- 46. Mayer-Davis EJ, Nichols M, Liese AD, Bell RA, Dabelea DM, Johansen JM, et al. Dietary intake among youth with diabetes: the SEARCH for Diabetes in Youth Study. J Am Diet Assoc. 2006;106:689–97.
- 47. Iqbal N, Vetter ML, Moore PH, Chittams JL, Dalton-Bakes CV, Dowd M, et al. Effects of a low-intensity intervention that prescribed a low-carbohydrate vs a low-fat diet in obese, diabetic participants. Obesity. 2010;18:1733–8.
- Van Wyk HJ, Davis E, Davies JS. A critical review of low-carbohydrate diets in people with type 2 diabetes. Diabet Med. 2015. doi:10.1111/dme.12964.
- 49. Krebs JD, Elley CR, Parry-Strong A, Lunt H, Drury PL, Bell DA, et al. The Diabetes Excess Weight Loss (DEWL) Trial: a randomized controlled trial of high-protein versus high-carbohydrate diets over 2 years in type 2 diabetes. Diabetologia. 2012;55:905–14.
- 50. Larsen RN, Mann NJ, Maclean E, Shaw JE. The effect of high-protein, low-carbohydrate diets in the treatment of type 2 diabetes: a 12 month randomized controlled trial. Diabetologia. 2011;54:731–40.
- Smart CE, Ross K, Edge JA, Collins CE, Colyvas K, King BR. Children and adolescents on intensive insulin therapy maintain postprandial glycemic control without precise carbohydrate counting. Diabet Med. 2009;26:279–85. doi:10.1111/j.1464-5491.2009.02669.x.
- 52. Smart CE, King BR, McElduff P, Collins CE. In children using intensive insulin therapy, a 20-g variation in carbohydrate amount significantly impacts on postprandial glycemia. Diabet Med. 2012;29:e21–4. doi:10.1111/j.1464-5491.2012.03595.x.
- 53. American Diabetes Association, Academy of Nutrition and Dietetics. Match your insulin to your carbs. Alexandria/ Chicago: American Diabetes Association/Academy of Nutrition and Dietetics; 2014.
- 54. Wolpert HA, Atakov-Castillo A, Smith SA, Steil GM. Dietary fat acutely increases glucose concentrations and insulin requirements in patients with type 1 diabetes: implications for carbohydrate-based bolus dose calculation and intensive diabetes management. Diabetes Care. 2013;36:810–6.
- 55. Smart CEM, Evans M, O'Connell SM, McElduff P, Lopez PE, Jones TW, et al. Both dietary protein and fat increase postprandial glucose concentrations in children with type 1 diabetes, and the effect is additive. Diabetes Care. 2013;36:3897–902.
- 56. Brand-Miller JC, Stockmann K, Atkinson F, Petocz P, Denyer G. Glycemic index, postprandial glycemia, and the shape of the curve in healthy subjects: analysis of a database of more than 1000 foods. Am J Clin Nutr. 2009;89:97–105.
- Wheeler ML, Dunbar SA, Jaacks LM, Karmally W, Mayer-Davis EJ, Wylie-Rosett J, Yancy Jr WS. Macronutrients, food groups and eating patterns in the management of diabetes: a systematic review of the literature. 2010. Diabetes Care. 2012;35:434–45.
- Estadella D, da Penha Oller do Nascimento CM, Oyama LM, Riberio EB, Damaso AR, de Piano A. Lipotoxicity: effectsof dietary saturated and transfatty acids. Mediat Inflamm. 2013:137579. http://dx.doi.org/10.1155/2013/137579.
- 59. Riserus U. Fatty acids and insulin sensitivity. Curr Opin Clin Nutr Metab Care. 2008;11:100–5.

- 60. Lee JS, Pinnamaneni SK, Eo DJ, CHO IH, Pho JH, Kim CK, et al. Saturated, but not n-6 poly-unsaturated fatty acids induce insulin resistance: role of intramuscular accumulation of lipid metabolites. J Appl Physiol. 2006;100:1467–74.
- 61. Rosenfalck AM, Almdal T, Viggers L, Madsbad S, Hilsted J. A low-fat diet improves peripheral insulin sensitivity in patients with type 1 diabetes. Diabet Med. 2006;23:384–92.
- 62. Davis NJ, Tomuta N, Schechter C, Isasi CR, Segal-Isaacson CJ, Stein D, et al. Comparative study of the effects of a 1-year dietary intervention of a low-carbohydrate diet versus a low-fat diet on weight and glycemic control in type 2 diabetes. Diabetes Care. 2009;32:1147–52.
- Feldstein AC, Nichols GA, Smith DH, Stevens VJ, Bachman K, Rosales AG, Perrin N. Weight change in diabetes and glycemic and blood pressure control. Diabetes Care. 2008;31:1960–5.
- 64. Esposito K, Maiorino MI, Ciotola M, Di Palo C, Scognamiglio P, Gicchino M, et al. Effects of a Mediterraneanstyle diet on the need for hyperglycemic drug therapy in patients with newly diagnosed type 2 diabetes: a randomized trial. Ann Intern Med. 2009;151:306–14.
- 65. Franz MJ, Boucher JL, Rutten-Ramos S, VanWormer JJ. Lifestyle weight-loss intervention outcomes in overweight and obese adults with type 2 diabetes. a systematic review and meat-analysis of randomized clinical trials. J Acad Nutr Diet. 2015;115:1447–63.
- 66. Franz MJ, VanWormer JJ, Crain AL, Boucher JL, Histon T, Caplan W, et al. Weight loss outcomes: a systematic review and meta-analysis of weight-loss clinical trials with a minimum of 1-year duration. J Am Diet Assoc. 2007;107:1755–67.
- 67. Anastasiou CA, Karfopoulou E, Yannakoulia M. Weight regaining: from statistics and behaviors to physiology and metabolism. Metabolism. 2015;64:1395–407.
- 68. Cefalu WT, Bray GA, Home PD, Garvey WT, Klein S, Pi-Sunyer FX, et al. Advances in the science, treatment, and prevention of the disease of obesity: reflections from a *Diabetes Care* editors' expert forum. Diabetes Care. 2015;38:1567–82.
- 69. Hollander PA, Elbein SC, Hirsch IB, Kelley D, MacGill J, Taylor T, et al. Role of orlistat in the treatment of obese patients with type 2 diabetes. A 1-year randomized double-blind study. Diabetes Care. 1998;21:1288–94.
- O'Neil PM, Smith SR, Weissman NJ, Fidler MC, et al. Randomized placebo-controlled clinical trial of lorcaserin for weight loss in type 2 diabetes mellitus: the BLOOM-DM Study. Obesity. 2012;20:1426–30. doi:10.1038/ oby.2012.66.
- Garvey WT, Ryan DH, Bohannon NJV, Kushner RF, Rueger M, Dvorak RV, Troupin B. Weight-loss therapy in type 2 diabetes: effects of phentermine and topiramate extended release. Diabetes Care. 2014;37:3309–316. doi:10.2337/ dc14-0930.
- 72. Hollander P, Gupta AK, Plodkowski R, Greenway F, Bays H, Burns C, et al. Effects of naltrexone sustained-release/ bupropion sustained-release combination therapy on body weight and glycemic parameters in overweight and obese patients with type 2 diabetes. Diabetes Care. 2013;36:4022–9.
- Davies MJ, Bergenstal R, Bode B, Kushner RF, Lewin A, Skjeth TV, et al. Efficacy of liraglutide for weight loss among patients with type 2 diabetes. The SCALE Diabetes Randomized Clinical Trial. JAMA. 2015;314:687–99. doi:10.1001/jama.2015.9676.
- 74. Garvey WT, Ryan DH, Henry R, Bohannon NJV, Toplak H, Schwiers M, et al. Prevention of type 2 diabetes in subjects with prediabetes and metabolic syndrome treated with phentermine and topiramate extended release. Diabetes Care. 2014;37:912–21.
- 75. Sjostrom L, Narbro K, Sjostrom CD, Karason K, Larsson B, Wedel H, et al.; for the Swedish Obese Subjects Study. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med. 2007;357:741–52.
- 76. Fox CS, Golden SH, Anderson C, Bray GA, Burke LE, de Boer IH, et al. Update on prevention of cardiovascular disease in adults with type 2 diabetes mellitus in light of recent evidence: a Scientific Statement from the American Heart Association and the American Diabetes Association. Diabetes Care. 2015;38:1777–803. doi:10.2337/dci15-0012.
- 77. Cox C. Diabetes nutrition therapy for sports and exercise. In: Franz MJ, Evert AB, editors. American diabetes association guide to nutrition therapy for diabetes. 2nd ed. Alexandria: American Diabetes Association; 2012. p. 205–27.
- American College of Sport Medicine, American Diabetes Association Joint Position Statement. Exercise and type 2 diabetes. Diabetes Care. 2010;33:2692–6.
- Rachmiel M, Buccino J, Daneman D. Exercise and type 1 diabetes mellitus in youth; review and recommendations. Ped Endocrinol Rev. 2007;5:656–65.
- Haas L, Maryniuk M, Beck J, Cox CE, Duker P, Edwards L, et al. National standards for diabetes self-management education and support. Diabetes Care. 2013;37 Suppl 1:S144–53. doi:10.2337/dc13-S100.
- Inzucchi SE, Bergenstal RM, Buse JB, Diamant M, Ferrannini E, Nauck M, et al. Management of hyperglycemia in type 2 diabetes, 2015; a patient-centered approach. Diabetes Care. 2015;38:140–9. doi:10.2337/dc14-2441.

Chapter 9 Nutrition in Weight Management and Obesity

Tracey Ledoux, Tabbetha Lopez, Craig Johnston, Elizabeth Vaughan, and John P. Foreyt

Key Points

- Obesity is caused by energy imbalance.
- Clinically significant weight loss is difficult to achieve, recidivism rates are high, and weight cycling may be harmful to health for some making sustainable behavior changes important.
- Food choice (i.e., diet) is driven by a number of complex factors that are within and beyond personal control.
- Medical nutrition therapy for treating obesity requires an interdisciplinary team and the promotion of dietary patterns using evidence based change strategies.

Keywords Medical nutrition therapy • Weight cycling • Popular diets • Food choice

Introduction

Simply put, obesity is a result of energy imbalance. Energy expenditure and energy intake are the two main factors in the energy balance equation. However, considerable disagreement occurs over the importance of energy expenditure in terms of obesity. The role that reduction in physical activity levels plays in stemming the rising obesity epidemic and the effectiveness of exercise in treating obesity are heavily debated topics. However, there is little argument that diet is a central issue in both the increasing prevalence of obesity and in the management of this disease. Although nutrition plays a straightforward role in its development and treatment, changing the diet of an individual is not so straightforward.

To further complicate this issue, considerable time and effort have been devoted to finding "the culprit" in diet that is ultimately responsible for the obesity epidemic. There are often headlines in the

T. Ledoux, PhD, RD, FAND (⊠) • T. Lopez, RD • C. Johnston, PhD Department of Health & Human Performance, University of Houston, 4800 Calhoun Road, Houston, TX 77004, USA e-mail: taledoux@Central.uh.edu

E. Vaughan, DO • J.P. Foreyt, PhD Department of Medicine, Baylor College of Medicine, Houston, TX, USA

media of a certain type of food or, even more specifically, an individual macro- or micronutrient that is touted to be "the cause" of obesity. Despite the desire for this to be the case, the keys to appropriate nutrition and healthy weight rest in variety, balance, and moderation. Although not nearly as intriguing as a singular cause, approaches that focus on these three factors are the ones that have the most evidence for long-term success. This chapter discusses factors that impact food choice, the management of obesity from a nutrition standpoint, diets that are used to reduce caloric intake, behavioral components to treatment, and myths associated with nutrition and weight loss.

Obesity

The Centers for Disease Control and Prevention (CDC) defines obesity as a condition of excess adiposity wherein weight is higher than what is considered healthy for a given height [17]. Body mass index (BMI) is the most common tool used to screen for obesity and is calculated using weight and height (weight in kilograms divided by height in meters squared) [17]. BMI is highly correlated with a person's body fat percentage making it an adequate screening tool. However, making an obesity diagnosis based on BMI alone should be done with caution as it does not distinguish between fat mass and lean muscle mass, and it does not specify adiposity. BMI should be examined within the context of other health risk indicators such as fasting glucose, blood pressure, and lipid levels.

In the general population, obesity increases the risk of chronic health conditions and all-cause mortality. When the relationship between obesity and all-cause mortality is stratified on disease status a more complicated relationship emerges [21, 96]. Paradoxically, obesity seems to have a somewhat protective effect on mortality among those with coronary heart disease [59], stroke [121], heart failure [57], end stage renal disease [80], and diabetes [21]. However, researchers hypothesize that this obesity paradox is observed when confounders are unmeasured. Several lifestyle behaviors (e.g., diet, physical activity, smoking status) and demographic variables (e.g., age, gender, race) may be confounders in the relationship between obesity and mortality and neglecting to account for only one or two can substantially skew the relationship [7].

Obesity has become a global epidemic. It is estimated that more than one-third of the US adult population is obese (78.6 million people) [18]. The 2011–2014 National Health and Nutrition Examination Survey (NHANES) found that obesity prevalence was 36.5% among adults [18, 34, 78]. Obesity prevalence in the United States is highest for non-Hispanic blacks and Mexican American women. Although obesity rates are high, there have been no significant changes in obesity prevalence for women or men between 1999 and 2010 [34].

Food Choice

Understanding the complex factors that influence food choice regarding what, when, and how much to eat will help clinicians develop and deliver effective weight loss interventions. Food choice behaviors regarding when to eat and how much are generally determined by homeostatic and nonhomeostatic pathways. Homeostatic eating begins in response to metabolic need or energy deficits experienced as physical hunger and ceases when energy needs are met and experienced as satiety. Nonhomeostatic food choice behaviors have been called "hedonic eating" and these are influenced by a variety of cognitive and environmental factors unrelated to metabolic need [11, 61, 124]. This section describes these factors and the ways they may influence food choice.

Metabolic Factors

A neural regulatory feedback system ensures that an adequate supply of energy is readily available for all necessary and critical biological functions such as brain activity and major organ function. Glucose is the primary energy source for all cells and as circulating levels of glucose decrease with prolonged time since eating, a cascade of neurohormonal processes are triggered to promote physical hunger sensations and meal initiation among other processes that release stored energy. Negative feedback signals including rising glucose levels serve to discontinue energy intake. Leptin, which is a hormone secreted by adipose tissue, enhances negative feedback signaling. Low levels of leptin or reduced leptin signaling stimulate greater food intake [11, 61, 90, 124]. Ghrelin, known as the hunger hormone, is secreted in the stomach and acts to increase feelings of hunger and food intake.

Environmental Factors

Characteristics and qualities of the environment may influence food choice through multiple mechanisms. The physical environment presents cues that initiate a desire to eat regardless of hunger level, i.e., nonhomeostatic eating. For example, the smell of popcorn at the movies may initiate a desire to eat popcorn despite having just eaten dinner. Alternatively, the physical environment provides facilitators or barriers to food choice behaviors. For example, having a pizza fast food restaurant across the street from one's home may facilitate the consumption of pizza on a more frequent basis than living several miles from a pizza restaurant.

Environmental Cues

While homeostatic eating is driven by energy stores and depletion, nonhomeostatic eating is primarily driven by reward pathways [46]. Food palatability and the neurobiologically rewarding effects of food motivate consumption beyond satiety and in the absence of hunger to varying degrees across individuals [46]. Oftentimes rewarding experiences become associated with environmental cues (e.g., sights, smells, sounds), such that exposure to the cues alone motivate behavioral activation toward fulfillment of the reward experience [10]. Several brain imaging studies show visual detection of palatable food is associated with activation of the brain's reward centers [38]. Behavioral studies have shown that manipulation of environmental conditions and cues can influence food choice behaviors regardless of hunger/satiety [115].

Facilitators and Barriers

Foods with greater availability and accessibility have fewer barriers to consumption. How strongly food availability and accessibility influence food choice may differ depending on the proximity of the environment in question. For example, the food available in one's home pantry may influence food choice to a greater extent than the food available in one's neighborhood. Of course, the food in one's neighborhood may also determine the food available in the home. The interconnectedness of food environments is something to be considered when addressing the availability of foods.

Neighborhood availability of healthy foods has been related to greater prevalence of healthy weight status of neighborhood residents [92]. However, research on the relationship between neighborhood availability of healthy foods and diet quality of neighborhood residents has been mixed [43]. Critics suggest insufficient rigor of study designs and undiscovered confounding mediators and moderators have interfered with the ability to adequately determine whether neighborhood availability of healthy and unhealthy foods influences diet quality of residents [43].

Two of the most commonly reported determinants of food choice, particularly by younger, female, and non-white populations are cost and convenience [39, 101]. Nutrient dense items like lean meats, fish, fresh vegetables, and fruit generally cost more than energy dense foods that are often highly palatable and convenient [27]. Relatively higher cost and lower convenience of healthy foods may detract from their accessibility for some populations regardless of whether these foods are available in the neighborhood environment.

Food availability and accessibility in the home are consistently related to food choice [114]. The home food environment includes our immediate surroundings—the room, eating utensils and food presentation [103]. Some factors (i.e., size and available appliances) determine the types of foods brought into the home. For example, having a broken oven may make a microwaveable food item a more viable option than one that requires baking or broiling. Another factor that directly influences food choices made in the home is visibility. Visible food items in the kitchen have a higher chance of being eaten [103, 114]. Also, more food items available at the table during meals leads to higher consumption in one sitting [103]. Plate, cup, and utensil sizes play an important role in consumption and how much we eat. Among a group of nutritionists, those who were given larger spoons consumed more ice cream than those with smaller spoons [103].

Portion size of packaged foods and foods served away from home influence food choice as well. People eat more when served more [20, 24, 122] and repeated exposure to larger portion sizes distorts consumption norms [60]. For example, participants who ate a dish of cheese pasta that had 50% more pasta in it had a 25% increase in overall meal intake [24]. In another experimental study, movie goers were randomly assigned to receive a large or medium container of stale popcorn [116]. Those given large containers of stale popcorn consumed on average 33.6% more than those given medium sized containers of stale popcorn. The moviegoers' intake was increased relative to the portion size of the popcorn regardless of the palatability. The tendency to eat more in response to larger portion sizes occurs across the population regardless of demographics [60].

Social and Media Environment

People typically eat in accordance with beliefs about what constitutes normal eating behavior [47]. Social eating norms are powerful determinants of food choice [22, 48, 89]. A meta-analysis of 15 experimental studies found a moderate effect of the provision of eating norm information (e.g., written, modeling) on food intake and food selection [89]. Also, in a review of 69 social eating studies representing diverse methodologies, nearly all of them showed food choices were influenced by social conditions [22]. Certain contextual factors may modulate the strength of the effect of social eating norms on food choice, but not substantially [22]. There is a tendency to eat more in social situations with friends and family members than when eating alone or in front of strangers [22]. People also capitalize on food choice stereotypes to portray themselves in more favorable light [111]. For example, meat consumption is generally recognized as a masculine food choice and research shows men are more likely to choose meat when their food choice is made public than if private [119]. Interestingly, there is little evidence that hunger, personality traits, age, or weight influence the effect social eating norms have on food choice [22]. The motivation to conform to

social eating norms may be rooted in basic survival mechanisms. More specifically, conforming to social eating norms promotes the consumption of safe foods, food acquisition and sharing, and social affiliation [48].

Most research regarding food advertisement and its effects on food choices is limited to samples consisting of young children and adolescents [125], but there are data to suggest that advertisement has a major influence in reinforcing and normalizing many behaviors across the rest of the population [49]. Previous research has shown that advertising can influence food preferences, purchases, and consumption behavior [125]. Evidence also suggests that increased exposure to TV food advertisements is linked to fast food consumption in adults [94].

Clinical Implications

Food choice is influenced by social and environmental factors which may be difficult to change at the individual level for a variety of reasons. First, many of these factors exert their influence innocuously. Most people are not aware that they change their eating behaviors depending on who is seated at the dinner table and what their food choice behaviors are. Further, some of these influences are not under direct control by individuals. Food prices and availability of healthy foods in one's neighborhood are not typically things that individuals can change. Nevertheless, mere awareness of the effect of these factors on behaviors may help individuals to interact with their environment in a different way.

More research is needed to determine the extent to which neighborhood availability of healthy and unhealthy foods influences food choice behaviors of residents. It is likely the association is multifaceted and sufficiently complex; therefore, policy level recommendations may be premature. However, there is enough evidence to suggest weight loss counseling should include conversations with individuals to determine their perceptions of the role of neighborhood food availability on their food choices. Clinicians should also determine the extent to which price and convenience influence food choice and whether their patients are eligible for federal and state nutrition assistance and education programs.

The home food environment has consistently been found to influence food choice behavior. This environment may be under direct control of individuals. Therefore, clinicians should assess the home food environment of their patients and make recommendations to optimize healthy food choices (e.g., make healthy foods visible). Given the importance of eating norms on food choice behaviors, weight loss counseling should correct mistaken beliefs regarding healthy portion sizes and increase awareness of the role of social and media influences on food choice.

Diet and Weight Loss

At any time, approximately 50–70% of obese Americans are actively trying to lose weight [15, 72]. According to NHANES data between 2001 and 2006, 63% of obese adults tried to lose weight in the previous year [72]. Sustained weight loss as little as 5–10% is considered clinically significant because it reduces risk of chronic conditions like diabetes and heart disease [51].

Approximately 60% of adults lose at least 5% of their body weight initially [72]. However, few sustain weight loss past the first year. Studies show that 20-50% of lost weight is regained within 1 year, and most people regain all of the weight after the first year [15, 76].

High recidivism rates may be frustrating, but, for some, weight cycling may have greater health risks than a higher but stable weight. Weight cycling is when an individual loses weight only to regain

it and repeats this process for a sustained period of time. In general, there is no consistent or conclusive association between weight cycling and mortality, diabetes risk, cardiovascular disease risk, or body composition [66]. In fact, some studies have shown weight cycling has an inverse relationship with cardiovascular disease biomarkers and diabetes risk [66]. Nonetheless, some populations may not benefit from weight loss if weight cycling occurs. For example, weight cycling was positively related to mortality among those age ≥ 65 years [4]. Older adults do not fully regain lean mass lost during weight loss and have a net gain in fat mass when weight cycling occurs [58]. Additionally, those of normal body weight may increase metabolic and cardiovascular risk factors with weight cycling [67].

Nutrition Intervention

As discussed above, many individuals attempt to lose weight on their own without much success. However, there is a strong scientific basis for assisted weight loss. Evidence-based dietary treatment of diseases is called medical nutrition therapy (MNT). It is the therapeutic approach used to treat medical conditions nutritionally. There is strong evidence that MNT improves weight, waist circumference, hip circumference, fast blood glucose, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and blood pressure [23, 84, 86, 91].

Basic MNT for adult weight management focuses on helping patients sustain a reduced calorie diet. Achieving it involves first determining calorie intake goals for weight loss and then selecting a dietary plan to sustain caloric intake reductions. Weight stability is achieved when energy intake equals energy expenditure. The first step in this process is to determine energy expenditure. The recommended energy intake at current weight can then be adjusted to create an energy deficit to facilitate weight loss. Indirect calorimetry is the gold standard approach to estimate energy expenditure by measuring oxygen consumption and carbon dioxide production. Close estimates of energy expenditure can be calculated based on age, sex, height, weight, and physical activity levels. Energy expenditure estimated from the Mifflin-St. Jeor formula (see Table 9.1) is highly correlated with energy expenditure measures from indirect calorimetry [37, 93, 99, 100, 117]. Energy expenditure in a state of energy balance. Reducing the average daily energy expenditure value for energy balance by 500 kcal per day provides an energy intake goal that will promote weight loss that is safe. Recommendations for energy intake should be no less than 1,200 calories per day for male or female adults to maintain adequate nutrient intake.

Table 9.1 Mifflin-St. Jeor equation

Men: Resting Metabolic Rate = $(9.99 \times \text{weight}) + (6.25 \times \text{height}) - (4.92 \times \text{age}) + 5$ *Women*: Resting Metabolic Rate = $(9.99 \times \text{weight}) + (6.25 \times \text{height}) - (4.92 \times \text{age}) - 161$ Equations use weight in kilograms (kg), height in centimeters (cm). Physical activity factors:

Sedentary: 1.0 or more to less than 1.4. Typical daily living activities (e.g., household tasks, walking to the bus) *Low active*: 1.4 or more to less than 1.6. Typical daily living activities plus 30–60 min of daily moderate activity (e.g., walking at 5–7 km per hour or 3 mph–4mph)

Active: 1.6 or more to less than 1.9. Typical daily living activities plus at least 60 min of daily moderate activity *Very active*: 1.9 or more to less than 2.5. Typical daily living activities plus at least 60 min of daily moderate activity

St. Jeor et al. [100]

Evidence-Based Diets

After the amount of energy intake for an individual is determined, a dietary pattern that will support the resulting energy intake goals without compromising micronutrient intake is selected. The three dietary patterns with strong evidence to be considered supportive of weight loss are the DASH diet, the Mediterranean diet, and the Healthy US Style Eating Pattern [51, 85].

The DASH diet (see Table 9.2) focuses on increasing vegetables, fruits, lean proteins, and whole grains while limiting saturated fats, sweets, sugar sweetened beverages, and sodium [12, 29, 30, 54, 71, 74, 75]. The DASH diet with caloric restriction and exercise has been shown to reduce weight and blood pressure [6, 12, 29, 75].

The Mediterranean Diet (MeDiet) emphasizes vegetables, fruits, fish, olive oil, nuts, beans, legumes, seeds, and whole grain with limited meats, dairy, and moderate wine consumption [77]. Plant-based dietary patterns, such as the MeDiet (see Table 9.3), are rich in antioxidants and low in saturated fats and are associated with lower cholesterol, lower blood pressure, improved glycemic control, reduced arterial stiffening, and reduced oxidative stress [97]. With olive oil as the primary fat, saturated fat is no more than 7-8% of caloric intake and overall fat 25-35% of intake. This pattern encourages physical activity and water consumption [97]. The MeDiet pattern has been shown to be effective in reducing weight and cardiovascular risk factors. Overweight and obese individuals following a MeDiet pattern achieved weight loss of 8.36-22.22 pounds in 12 months [64].

The Healthy US Style Eating Pattern (HUSEP) includes a healthy intake of vegetables, fruits, whole grains, fat free or low fat dairy, proteins, and mono and poly unsaturated fats, with limits on added sugars, saturated fats, trans fats, cholesterol, sodium, and alcohol [77]. It is designed to meet the recommended dietary allowances (RDA) and the adequate intakes (AI) of all micronutrients. The recommended portion size of these foods is based on caloric needs, as seen in Table 9.4. The USDA My Plate is an illustrated example of the HUSEP that focuses on variety, portion size and dietary needs [109]. The HUSEP can be used as a general healthy eating pattern for adults and children for weight loss, gain, or maintenance.

Meal Replacements and Structured Meal Plans

Meal replacements or structured meal plans are implemented within weight loss treatment programs to help overweight and obese persons decrease total calorie intake [5, 25, 26, 45, 113]. Meal replacements decrease intake by replacing one to two primary meals (breakfast, lunch, and dinner) with a

	Servings per	
Food group	day	Examples
Whole grains	6–8	Brown rice, whole grain pastas, breads, crackers
Protein	6 or less	White meat chicken, fish, lean pork, beans, nuts, cottage cheese
Vegetables	4–5	Broccoli; spinach; romaine; collard, turnip, mustard greens, tomatoes, red peppers, carrots, sweet potatoes, winter squash, and pumpkin
Fruit	4–5	Apples, oranges,
Low-fat or fat-free dairy products	2–3	1 % milk, Greek yogurt,
Fats and oils	2–3	Olive oil, canola oil, vegetable oils, avocados, nuts
Sodium	1500-2300 mg	-

Table 9.2 DASH diet - number of servings based on a 2000 calorie diet

Food group	Servings
Vegetables, fruit, olive oil, nuts, legumes, beans, seeds, herbs, and spices	Base every meal of these foods
Fish and seafood	Often, at least two times per week
Poultry, eggs, cheese, and yogurt	Moderation, daily to weekly portions
Meats and sweets	Less often
Wine	Moderation

Table 9.3 Mediterranean diet

Table 9.4 Healthy US-style eating pattern – daily amount based on a 2,000 calorie diet

Daily amount	
2 1/2 cups	
2 cups	
6 ounces	
3 cups	
5 1/2 ounces	
27 g	
14% of daily calories	
	2 1/2 cups 2 cups 6 ounces 3 cups 5 1/2 ounces 27 g

Office of Disease Prevention and Health Promotion (ODPHP) [77]

lower calorie alternative such as a bar, shake, soup, or hot chocolate [5, 25]. Structured meal plans, in combination with behavioral weight loss interventions, generally consist of a low calorie diet (1200–1500 kcal daily) with three meals (breakfast, lunch, and dinner) and two snacks per day [26]. Participants who achieve and maintain weight loss by using meal replacements or structured meal plans have shown improved biomarkers of disease risks such as insulin, triglyceride, and blood pressure [26]. Weight loss interventions that use meal replacements or structured meal plans are thought to result in greater weight loss because they reduce the need to make food choices regarding what, how much, and in some cases when to eat. Moreover, they reduce snacking and increase accessibility of low-calorie foods at home [120].

Several studies, 1 year or longer, have found significant weight loss among participants incorporating meal replacements or structured meals with a behavioral weight loss intervention compared to those who solely followed an intervention [5, 25, 26, 45, 113]. For instance, the LOOK Ahead ("Action for Health in Diabetes") trial, which is the longest running study of a lifestyle intervention to date, included 5,145 overweight and obese males and females with type 2 diabetes and found that participants who replaced one snack and one meal per day with a meal replacement (meal bar and liquid shake) lost substantially more weight than those who did not [113]. Overall, meal replacements or structured meal plans help participants to achieve greater initial weight loss compared to self-selected food plans [25].

Length of Treatment

Individuals should be monitored as they work toward their goals to help them overcome barriers to change, to maximize the likelihood changes made are sustainable, and to identify complications resulting from the prescribed nutrition intervention. Monitoring progress requires follow up appointments to assess key outcomes such as weight and laboratory data to ensure outcomes are being achieved without compromising health. Data to be collected at follow-up include lab values (e.g.,

blood glucose, blood pressure, lipids), behavior (e.g., physical activity), signs and symptoms of emergent nutrition problems (e.g., eating disorders), and quality of life. Follow-up appointments should include problem solving and relapse prevention discussions. Based on a strong research base, it is recommended that 14 consultations be conducted in 6 months for initial weight loss, and 12 consultations in 12 months for weight maintenance [51].

Multidisciplinary Approach

The Centers for Medicare and Medicaid Services [19] issued a decision memo in 2011 which indicated that obese individuals should receive 12 months of intensive behavioral therapy that includes 20 sessions of face-to-face visits. Additionally, the US Preventive Services Task Force, The American Heart Association, American College of Cardiology, The Obesity Society, and Academy of Nutrition and Dietetics recommend a multidisciplinary team approach [35, 51, 85].

A team approach produces better outcomes than treatment provided by a solitary health care provider [31, 35, 51, 83, 108]. It can be difficult for one practitioner to offer sufficient care for the obese individual due to time constraints [9, 108], lack of resources, and busy practices [83]. The interdisciplinary team may consist of registered nurses (RN), registered dietitians (RD), behavioral health specialists, exercise specialists, pharmacists, and physicians [9, 31, 51, 85, 112]. Each of these professionals must know their individual role in the treatment process and work together to achieve the most effective results. A registered dietitian (RD) is the one who is typically tasked with helping obese persons make sustainable dietary changes to support weight loss.

Behavioral Considerations to Treatment

The Academy of Nutrition and Dietetics recommends that basic MNT for adult weight management include the use of behavior change strategies to help patients sustain a reduced calorie diet and increased physical activity. The goal of behavioral techniques is to provide the individual with skills they can use to improve their overall lifestyle. By changing lifestyle patterns, the chances of short- and long-term success are increased. Although there are numerous behavioral strategies to be used in promoting an improved diet, self-monitoring, problem solving, and cognitive changes are especially important components in treatment.

Self-monitoring

Self-monitoring is an essential component of a behavioral weight loss treatment [82]. Regularly recording dietary intake, weight, and/or physical activity enables individuals to be self-aware of their behaviors and modify as needed, as well as measure progress toward lifestyle intervention goals [16, 82]. Various tools for self-monitoring include hand written diaries, digital scales, web-based programs, and personal digital assistants [16, 82]. Consistency, frequency, and comprehensiveness are all important aspects of successful self-monitoring [82]. However, a limited number of studies have shown that frequency and consistency have significantly greater impact on maintaining weight loss than comprehensiveness, which has lesser impact [82].

A systematic review of 22 studies found a positive relationship between self-monitoring of dietary intake, exercise or weight and effective weight management [16]. Further, numerous randomized

controlled trials support the use of self-monitoring as an effective behavioral weight loss strategy [13, 16, 56, 63, 106]. For instance, a randomized controlled trial of 123 postmenopausal, Caucasian overweight and obese women found those self-monitoring their energy consumption achieved greater weight loss than those who did not [56]. Using food journals may be one of the most productive weight loss strategies [56].

Problem Solving

Problem solving, a self-management technique, is a behavioral process wherein multiple solutions for coping are generated and then the pros and cons are considered before implementing one [28]. Essentially, problem solving empowers individuals by preparing them for unforeseen barriers, which may have otherwise caused them to deviate [62, 70, 81]. Possible barriers may include insufficient resources, inadequate or limited support, ambivalence, or lack of motivation. The steps of problem solving include: having motivation to problem solve, defining the problem, formulating alternate options, identifying the best option, applying the solution, and analyzing your decision afterwards [28].

Those with obesity, diabetes, or any other chronic illness may benefit from this strategy [41, 62]. In fact, various studies have assessed the validity of problem solving as a behavioral strategy, finding it to be an effective tool for weight loss, maintaining weight, and following a diabetes self-management regimen [41, 62, 70, 81]. Specifically, in a 6-month behavioral weight loss intervention study, researchers found a relationship between problem solving and weight loss. Frequent problem solvers lost significantly more weight than those less consistent. However, this method may not be successful for those with below average problem solving skills as these individuals may be deterred by the effort required or simply incapable of using that skillset. Further research identifying specific behavioral, psychological, and population characteristics affecting weight loss may be useful in improving our understanding for using it as an intervention.

Cognition

Cognitive factors such as thoughts, beliefs, and attitudes precede behavior initiation and are often developed over time in reaction to personal life experiences, the social milieu, and media. Cognitive factors interact with environmental and internal cues to influence behavior. For example, hunger pangs (i.e., internal cue) and the sight of a bag of potato chips on one's counter at home (i.e., environmental cue) may trigger the desire for them (i.e., unconscious neurobiological reaction). A series of cognitions (e.g., knowledge that potato chips are incongruent with weight loss goals) may arise and ultimately determine whether the potato chips are eaten or not.

Behavioral theories provide an organizing structure to explain how multiple cognitive factors interact to produce behavior. One model for conceptualizing cognitive determinants of food choice behaviors in both adults and children is the Theory of Planned Behavior (TPB) [65, 88]. TPB posits the primary predictor of a behavior is intention, which refers to the amount of effort one is willing to put toward a behavior [40]. Intentions are then determined by attitude toward the behavior, social normative perceptions regarding it, and perceived control in performing it [40]. Attitudes toward the behavior refer to outcome expectancies one has for performing the behavior. Social normative perceptions reflect the extent to which performing the behavior would be considered a normal behavior within one's peer group. Perceived control is similar to self-efficacy in that it refers to how confident individuals are that they can perform the behavior. A meta-analysis of 46 TPB studies showed perceived control, attitudes, and social norms were moderately to strongly related to intentions [65]. Also, perceived control and intentions were moderately related to behaviors [65].

Other cognitive factors that have been related to food choice behaviors are self-efficacy, self-regulation, and nutrition knowledge. Self-efficacy, which is very similar to perceived control from TPB, reflects a belief or confidence in the ability to overcome inherent difficulties in performing a specific task in a particular situation [1]. A number of studies have found a strong relationship between self-efficacy and fruit and vegetable intake [95]. However, results are less conclusive across a range of dietary behaviors (e.g., lowering fat, sugar) [98]. Self-regulation is the intentional use of strategies such as goal setting, self-reward, self-monitoring, and self-instruction, to control one's behaviors. A large body of research suggests that the use of self-regulation strategies has positive medium effects on health promotion food choices and small inverse effects on unhealthy food choices [2]. Results from a review of 29 studies showed the majority found a weak but significant positive relationship between nutrition knowledge and dietary intake [104].

In general, nutrition and weight loss treatment should target modifiable cognitive determinants of food choice including behavioral intentions, perceived control/self-efficacy, attitudes, perceived social norms, self-regulation, and nutrition knowledge. The strength of the association between these factors and behaviors varies depending on the specific behavior in question (e.g., fruit consumption versus avoidance of sugar), demographic characteristics of the individual (e.g., age, income), and other cognitive factors. Each cognitive factor requires different change strategies. For example, experiential learning builds self-efficacy [8], but goal setting increases self-regulation. Given the variety in strategies required to change cognitive determinants of food choice, multidisciplinary treatment teams including a registered dietitian are better positioned than individual providers to help patients change food choice behaviors.

Commercial Weight Loss Programs

Commercial and proprietary weight loss programs have grown into a multibillion dollar industry [44]. In addition, there are countless "popular" diets and trends that gain popularity for a time and then give way to another. Many obese individuals will have experience with one or more commercial weight loss programs. Thus, it is important for clinicians to know the pros and cons of these types of diet plans to be able to advise their patients adequately (see Table 9.5).

Commercial weight loss programs are monitored by the Federal Trade Commission (FTC) [107]. Without monitoring, consumers may be exposed to misleading claims, endorsements, and advertisements. When a diet or program cannot keep the promises they make, the consumer incurs substantial costs in terms of time and money [32]. This is why practice and treatment standards are so important, not just for clinicians, but for consumers. They serve as barometer by which to measure intervention options so that the consumer is protected from a circuit of false advertising.

Commercially available weight loss diets have a certain appeal. For one, they can be seen as the "medicine" needed to treat the disease, obesity. It can seem like a quicker path to a healthy weight than alternative conventional methods. Attractive endorsements, case studies, and promotions also play a role in a diet's popularity. Popular diets come in many shapes and sizes. Some may offer convenience with prepared meal deliveries or shakes. Others require special equipment and preparation demands. Some are communicated in books or on the internet while others incorporate instruction in person by varying degrees of qualified professionals. As of 2014, the \$2.5 billion commercial weight loss industry was primarily represented by Weight Watchers, Nutrisystem, and Jenny Craig. These three programs accounted for about 75 % of the market share [44].

Rankings of popular diets have been established by a panel of experts based on supporting and refuting evidence, cost, results, and practicality [110]. Along with Jenny Craig, Weight Watchers, and

Diet	Claim	Method
Weight Watchers	Drop up to 2 pounds weekly	Focuses on skill building. Uses points system to guide consumers to nutritious calorie choices. Includes fitness tracking and support meeting components. (Calories: 1300, fat 26%, carbs 49%, protein 26%)
Jenny Craig	Drop up to 2 pounds weekly	Personalized meal and activity plan as well as weekly counseling sessions with Jenny Craig certified coaches. Initially, meals and snacks are delivered and are supplemented by fruits and vegetables supplied by the consumer. Once halfway to goal weight consumers cook for themselves a couple times per week using Jenny Craig recipes. Finally, once at goal weight consumers transition off of delivered meals completely over 4 weeks. Option available for a plan geared towards customers with Type 2 Diabetes. (<i>1500 calorie plan</i> — Calories: 1469, fat: 20%, carbs: 55%, protein: 25%)
Nutrisystem	Lose 5 pounds in first week and 1–2 pounds each subsequent week	 Meals and shakes are delivered and are supplemented by fruits vegetables, protein and dairy supplied by the consumer. The grocery items are added per Nutrisystem guidance. Customizable options include gender specific, senior, diabetic, vegetarian, and DASH diet friendly. Transition off the program aided by telephone or online coaching. (<i>Basic Men's Plan</i>—Calories: 1500, fat: 21%, carbs: 58%, protein: 21% <i>Basic Women's Plan</i>—Calories: 1200, fat: 20%, carbs: 56%, protein: 24%)
HMR program	Maintain significant weight loss through medically supervised in-clinic or at home options	Two phases. Phase 1 is a regimen of delivered meals and meal replacements to be supplemented by fruits and vegetables. Phase 2 is a transition phase where meals are still provided, but no shakes. Customers add fruits, vegetables, and low calorie food to supplement meals. Includes weekly telephone coaching with dietitians and exercise physiologists. Medical supervision and in-person support group available through the in-clinic option. (Calories: 1037, fat: 14%, carbs: 67%, protein: 13%)
Biggest loser diet	6 weeks of healthy food and regular exercise can initiate weight loss, help prevent or reverse diabetes, and reduce risk for other chronic diseases.	Books released periodically laden with success stories from contestants on The Biggest Loser TV show, meal planning tips, and suggestions for burning calories (Calories: 1489, fat: 25%, carbs: 50%, protein 30%)
Raw food diet	Raw food has natural enzymes and nutrients promoting optimal health and you'll lose weight	 Participants eat raw foods exclusively. Mostly plant food, though meat and unpasteurized dairy can be included. Foods not heated above 115° F. Juicing, blending, dehydrating are common. (<i>Estimated average intake</i>—Calories: 1280, fat: 28%, carbs: 59%, protein: 13%)
Volumetrics diet	Drop 1–2 pounds per week	Participants use a guide to help steer food choices to lower overall energy density. Foods are broken up into 4 categories ranging from very low energy dense to highly energy dense. Meal plans and recipes included. (Calories: 1575, fat: 24%, carbs: 55%, protein: 23%)
Atkins diet	Lose up to 15 pounds in 2 weeks	Participants start off with 20 net carbs per day and gradually phase carbs back in over the course of the 4 phase diet. (<i>Phase 1</i> —Calories: 1527, fat: 63%, carbs: 10% protein: 29%. <i>Phase 4</i> —Calories: 1958, fat: 54%, carbs: 24%, protein: 27%)

 Table 9.5
 Commercially available weight loss programs

Table	9.5	(continued)
-------	-----	------------	---

Diet	Claim	Method
Flexitarian diet	Felxitarians weigh 15 % less than carnivores, have a lower risk of heart disease, diabetes, and cancer, and live 3.6 years longer.	Participants build their meals from a set of 5 food groups unique to this diet. They are 'new meat' or plant based protein, fruit and vegetable, grains, dairy, and sugar and spice which include herbs, spices, and sweeteners. A 5 week 1500 calorie meal plan (breakfast, lunch, dinner and snacks) is included. Participants can be flexible with this plan and swap recipes per preference. (Calories: 1543, fat: 27%, carbs: 57%, protein: 15%)
Slim fast diet	Drop 1–2 pounds weekly	Participants drink or consume 3 meals and 3 snacks per day. 2 meals are slim fast meal replacement bar or shakes. The other meal is a 500 calorie meal prepared by participant. Snacks are either fruit or a slim fast snack bar. (Calories: 1200, fat: 24%, carbs: 58%, protein: 22%)
Vegan diet	Following a vegan diet can help participants lose weight and reduce risk of chronic disease	Food sourced from animals and animal products are not consumed on this diet. This plant based diet can present a challenge in meeting micro and macronutrient needs but it can be done. Supplements and fortification are used to boost intake of hard to source nutrients like B12. (US Dietary guidelines recommendation for vegan diet— Calories: 2000, fat: 33 %, carbs: 57 % protein: 13 %) [53]
Mediterranean diet	Lose weight and improve overall health	To follow a Mediterranean diet is to build meals that focus on some food groups with others added in moderation. At it's a core, a Mediterranean diet draws from fruits, vegetables, nuts, seeds, grains, olive oil, herbs and spices. Seafood, eggs, and dairy are scattered in, while red meat, kcal dense desserts and alcohol are limited to the rare special occasion. (<i>Nutrition breakdown per Oldways interpretation of this</i> <i>highly varied diet</i> —Calories: 1527, fat: 29%, carbs: 50%, protein: 18%) [79]

Nutrisystem, the list includes the HMR Program, Biggest Loser Diet, Raw Food Diet, Volumetrics Diet, Atkins Diet, Flexitarian Diet, Slim Fast Diet, Vegan Diet, and the Mediterranean Diet. The rankings are presented with an important acknowledgement that "no diet is ideal for everybody." Therefore the *best* diet for an individual would be one that is proven safe, effective, and can be tolerated and sustained in the long run [110].

When determining the possible dietary paths to weight loss in overweight and obese adults, the AHA/ACC/TOS practice guidelines thoroughly examined the available evidence [51]. The task force evaluated articles using strict inclusion and exclusion criteria and rigorous quality standards. The most reliable existing research was considered to address the comparative effectiveness of dietary patterns, structures, strategies and/or nutrient breakdowns in achieving weight loss. The panel concluded that a reduction in overall energy intake (typically 1200–1500 kcal/day for women and 1500–1800 kcal/day for men) is required to achieve weight loss while the macronutrient composition may vary [51]. In a recent meta-analysis of commercial weight loss program comparison studies little to no difference was seen between programs that were effective in achieving significant weight loss at 6 and 12 month follow-up [52].

An increased protein, calorie-restricted approach such as Weight Watchers, Jenny Craig, and The Biggest Loser received a high strength of evidence rating by the task force. The low carbohydrate approach (<20 g/day carbohydrates for 3 months followed by increased intake) was effective if energy deficit was realized. High and low glycemic index meals did not appear to have a bearing on the diet's effectiveness to achieve weight loss. There was no difference seen between low carbohydrate and low

fat diets at 6 months. Lower fat calorie-restricted diets (<30% kcal/day from fat) did not outperform similarly restricted higher fat diets ($\geq 40\%$ kcal/day from fat). The meta-analysis did not find a substantial difference between low carb (<40% kcal) and low fat diets (<20% kcal) though the two held an edge over moderate macronutrient distributions [52].

Diets that focus on eating pattern such as Flexitarian, Vegan, Raw Food, Volumetrics, and Mediterranean can yield weight loss. However, the results may be comparable to a typical calorie restricted lower fat diet. With that in mind, it is vital for individuals to consider the development, resources, time, palatability, and personal preference associated with any of these systems.

Very low calorie approaches (800–1000 kcal/d) such as the HMR Program are not considered differently by the AHA/ACC/TOS guidelines in that they meet the requirement of supporting a calorie deficit. However, a review of commercial weight loss programs found interventions like this one to be initially effective, but lack evidence of sustained weight loss [44]. The moderate calorie meal replacement approach like Slim Fast has been shown to be more effective for overweight and obese women over balanced calorie-deficit food based diets up to 6 months [51]. With limited and low strength of evidence, there is a need for more research to determine the strength of the meal replacement method versus a similar calorie restricted food approach in overweight and obese adults. For a consumer, there is a degree of convenience of food provision interventions, like Slim Fast, HMR, and Nutrisystem. Prepackaged food has been shown to improve compliance and weight loss [33].

The guidelines along with other reviews and studies suggest that a variety of dietary approaches can be effective in helping overweight and obese adults achieve weight loss [14, 44, 51, 52]. Weight management should focus on more than diet alone. Multifactorial treatments spanning dietary, behavioral, and activity interventions delivered by trained professionals (i.e., registered dietitians) have been shown to be more effective than usual care alone [14, 51]. Therefore, consumers would be best served in finding a diet program that has demonstrated safety and efficacy, that is supported by clinical evidence, and that has the highest likelihood of long-term adherence. In addition to the diet program selected, participants should consider physical activity and behavioral interventions as part of a comprehensive lifestyle intervention to managing obesity [14, 44, 51, 52].

Nutrition Weight Loss Myths

It is typical for individuals to want to know "the secret" to weight loss. Because of this, many myths about diet are formed. Additionally, the media often picks up on these myths and can further promote these popular opinions about weight loss.

"Drinking More Water Can Aid in Weight Loss"

Undecided. In a review of the impact of water consumption on weight loss and maintenance, two proposed mechanisms for the association of increased water intake and weight loss are identified as short-term suppression of hunger and increased thermogenesis (energy consuming) effect of consuming water (though very minor) [69]. These mechanisms may be observed with other beverages, though weight loss may not be achieved as well with sugar-sweetened beverages, which may cause excess energy intake [102]. In general, the recommended dietary allowance (RDA) of water is 1 mL of water for every calorie consumed.

"Skipping Breakfast Causes Weight Gain"

Myth. Results of some observational studies indicate that those who eat breakfast are less likely to be overweight and obese. Unfortunately, these studies do not encompass causation as to why those who eat breakfast are less likely to have weight problems. In a randomized control study [55], eight male subjects were measured for 24-h periods in a room-sized respiratory chamber, meals were varied between three (breakfast, lunch, dinner) and two (lunch, dinner) but overall caloric intake remained the same. Results concluded that between the two dietary patterns there was no statistical difference between energy expenditure, thermic effect of food or energy expenditure during the inactive state. While overall caloric intake affects weight gain more than the timing of the intake, there is a body of evidence that indicates eating breakfast may aid in weight loss. For example, in a 12-week study [50], one group of women consumed a high calorie breakfast while a second group consumed a high calorie dinner. Both consumed 1400 calories per day with identical micronutrients. At the completion of the study, both groups lost weight but the high calorie breakfast group lost significantly more weight. This suggests that eating breakfast may have benefits that support weight loss. Therefore, while skipping breakfast does not lead to weight gain, the addition or continuation of eating breakfast may support weight loss.

"Eat Small Meals More Frequently"

Myth. Several studies have examined the relationship between food timing and frequency. Currently there is not enough research to support increased eating frequency on energy intake or body weight [73, 118, 123]. The Academy of Nutrition and Dietetics examined 31 studies on the relationship between eating frequency and weight change. The studies reported inconsistent results including decreased weight, increased weight and no association.

"Eating After 10 pm Will Cause Weight Gain"

Undecided. Several studies have found an association between higher evening energy intake and increased body weight [3, 36, 42, 68, 105]. It is not known whether evening meals or snacks cause weight gain or whether these two phenomena co-occur together. Ultimately, positive energy balance causes weight gain, but there is potential of later evening meals to increase overall caloric intake. A recent study found that eating later in the day could lead to weight gain due to more meals consumed throughout the day [87].

Conclusion

A firm foundation has been established in terms of the treatment of obesity through nutrition. Understanding that food choices are determined by a myriad of factors and that social learning with regards to food begins at a young age, it is no surprise that obesity treatment through diet is a slow process. Following a structured plan that allows individuals to be in a caloric deficit is critical. Additionally, a multidisciplinary team that can address the complications of obesity provides the support needed for individuals who are suffering from this disease. Finding the right diet for the person

is a critical step. Overall, the best diet for individuals is one that is safe and effective, and one they can adhere to for an extended period of time.

Acknowledgment We would like to express our thanks for the exemplary work on this project provided by Denny Dao, Seemab Jamil, Moe Schlachter, Julia Jarrell, Christina Garaghty, Meredith Price, and Sara Falk. Their participation in writing, revising, and providing feedback has significantly contributed to this manuscript. Their dedication to promoting the health of individuals through the dissemination of sound science is greatly appreciated.

References

- 1. AbuSabha R, Achterberg C. Review of self-efficacy and locus of control for nutrition and health related behavior. J Am Diet Assoc. 1997;97(10):1122–32.
- Adriaanse MA, Vinkers CD, Ridder DT, Hox JJ, De Wit JB. Do implementation intentions help to eat a healthy diet? A systematic review and meta-analysis of the empirical evidence. Appetite. 2011;56(1):183–93. doi:10.1016/j. appet.2010.10.012.
- 3. Anderson I, Rossner S. Meal patterns in obese and normal weight men: the 'Gustaf' study. Eur J Clin Nutr. 1996;50(10):639–46.
- Arnold AM, Newman AB, Cushman M, Ding J, Kritchevsky S. Body Weight Dynamics and their association with physical function and mortality in older adults: The Cardiovascular Health Study. J Gerontol A Biol Sci Med Sci. 2009;65(1):63–70. doi:10.1093/gerona/glp050.
- 5. Ashley JM, St Jeor ST, Schrage JP, Perumean-Chaney SE, Gilbertson MC, McCall NL, Bovee V. Weight control in the physician's office. Arch Intern Med. 2001;161(13):1599–604.
- 6. Azadbakht L, Mirmiran P, Esmaillzadeh A, Azizi T, Azizi F. Beneficial effects of a Dietary Approaches to Stop Hypertension eating plan on features of the metabolic syndrome. Diabetes Care. 2005;28(12):2823–31.
- Banack MA, Kaufman JS. Does selection bias explain the obesity paradox among individuals with cardiovascular disease? Ann Epidemiol. 2015;25(5):342–9. doi:10.1016/j.annepidem.2015.02.008.
- 8. Bandura A. Health promotion by social cognitive means. Health Educ Behav. 2004;31(2):143-64.
- Bernstein KM, Manning DA, Julian RM. Multidisciplinary teams and obesity: role of the modern patient-centered medical home. Prim Care Clin Off Pract. 2016;43(1):53–9. doi:10.1016/j.pop.2015.08.010.
- 10. Berridge KC, Robinson TE. Parsing reward. Trends Neurosci. 2003;26(9):507-13.
- Berthoud HR. Homeostatic and non-homeostatic pathways involved in the control of food intake and energy balance. Obesity. 2006;14(S5):197S–200.
- Blumenthal JA, Babyak JA, Sherwood A, Craighead L, Pao-HWa L, Johnson J, Watkins LL, Wang JT, Kuhn C, Feinglos M, Hinderliter A. The effects of the dash diet alone and in combination with exercise and caloric restriction on insulin sensitivity and lipids. Hypertension. 2010;55(5):1199–205.
- Boutelle K, Kirschenbaum D, Baker R, Mitchell M, Krantz DS. How Can obese weight controllers minimize weight gain during the high risk holiday season? By self-monitoring very consistently. Health Psychol. 1999;18(4):364–8. doi:10.1037/0278-6133.18.4.364.
- Bray GA, Fruhbech G, Ryan DH, Wilding JPH. Management of obesity. Lancet. 2016;387:1947–56. doi:10.1016/ S0140-6736(16)00271-3.
- Brown RE, Kuk JL. Consequences of obesity and weight loss: a devil's advocate position. Obes Rev. 2014;16(1):77– 87. doi:10.1111/obr.12232.
- Burke LE, Wang J, Sevick MA. Self-monitoring in weight loss: a systematic review of the literature. J Am Diet Assoc. 2011;111(1):92–102. doi:10.1016/j.jada.2010.10.008.
- Centers for Disease Control and Prevention. Defining adult overweight and obesity. 2012. Retrieved from http:// www.cdc.gov/obesity/adult/defining.html.
- Centers for Disease Control and Prevention. Prevalence of obesity among adults and youth: United States, 2011– 2014. 2015. Retrieved from http://www.cdc.gov/nchs/products/databriefs/db219.htm.
- Centers for Medicare and Medicaid Services. Decision memo for intensive behavioral therapy for obesity. 2011. Retrieved from https://www.cms.gov/medicare-coverage-database/details/nca-decision-memo.aspx?&NcaName= Intensive.Behavioral Therapy for Obesity&bc=ACAAAAAAIAAA&NCAId=253&.
- 20. Chandon P, Wansink B. Does food marketing need to make us fat? A review and solutions. Nutr Rev. 2012;70(10):571–93. doi:10.1111/j.1753-4887.2012.00518.x.
- Chang HW, Li YH, Hsieh CH, Liu PY, Lin GM. Association of body mass index with all-cause mortality in patients with diabetes: a systematic review and meta-analysis. Cardiovasc Diagn Ther. 2016;6(2):109–19. doi:10.21037/cdt.2015.12.06.

- Cruwys T, Bevelander KE, Hermans RC. Social modeling of eating: a review of when and why social influence affects food intake and choice. Appetite. 2015;86:3–18. doi:10.1016/j.appet.2014.08.035.
- 23. Digenio AG, Mancuso JP, Gerber RA, Dvorak RV. Comparison of methods for delivering a lifestyle modification program for obese patients: a randomized trial. Ann Int Med. 2009;150(4):255–62.
- Diliberti N, Bordi PL, Conklin MT, Roe LS, Rolls BJ. Increased portion size leads to increased energy intake in a restaurant meal. Obes Res. 2004;12(3):562–8.
- Ditschuneit HH, Flechtner-Mors M, Johnson TD, Adler G. Metabolic and weight-loss effects of a long-term dietary intervention in obese patients. Am J Clin Nutr. 1999;69(2):198–204.
- 26. Ditschuneit HH, Flechtner-Mors M. Value of structured meals for weight management: risk factors and long-term weight maintenance. Obes Res. 2001;9(S4):284S–9S. doi:http://dx.doi.org/10.1038/oby.2001.132.
- Drewnowski A, Darmon N. The economics of obesity: dietary energy density and energy cost. Am J Clin Nutr. 2005;82(1):265S–73.
- D'Zurilla T, Goldfried M, Peterson D. Problem solving and behavior modification. J Abnorm Psychol. 1971;78(1):107–26.
- Elmer PJ, Obarzanek E, Vollmer WM, Simons-Morton D, Stevens VJ, Young DR, Pao-Hwa L, Champagne C, Harsha DW, Svetkey LP, Ard J, Brantley PJ, Proschan MA, Erlinger TP, Appel LJ. Effects of comprehensive lifestyle modification on diet, weight, physical fitness, and blood pressure control: eighteen-month results of a randomized trial. Ann Intern Med. 2006;144:485–95.
- 30. Epstein DE, Sherwood A, Smith PJ, Craighead L, Caccia C, Lin PH, Babyak MA, Johnson JJ, Hinderliter A, Blumenthal JA. Determinants and consequences of adherence to the dietary approaches to stop hypertension diet in African-American and white adults with high blood pressure: results from the ENCORE trial. J Acad Nutr Diet. 2012;112(11):1763–73.
- 31. Fakih S, Marriott JL, Hussainy SY. Employing the nominal group technique to explore the views of pharmacists, pharmacy assistants and women on community pharmacy weight management services and educational resources. Int J Pharm Pract. 2015;24(2):86–96. doi:10.1111/jjpp.12218.
- Federal Trade Commission (FTC). FTC releases report on weight-loss advertising. 2002. Retrieved at https:// www.ftc.gov/news-events/press-releases/2002/09/ftc-releases-report-weight-loss-advertising.
- 33. Flechtner-Mors M, Herwig H, Ditschuneit M, Johnson TD, Suchard MA, Adler G. Metabolic and weight loss effects of long-term dietary intervention in obese patients: four-year results. Obesity. 2000;8(5):399–402. doi:10.1038/oby.2000.48.
- Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. JAMA. 2012;307(5):491–7. doi:10.1001/jama.2012.39.
- Fitzpatrick SL, Wischenka D, Appelhans BM, Pbert L, Wang M, Wilson DK, Pagoto SL. An evidence-based guide for obesity treatment in primary care. Am J Med. 2016;129(1):115. doi:10.1016/j.amjmed.2015.07.015.
- Forslund HB, Lindroos AK, Sjostrom L, Lissner L. Meal patterns and obesity in Swedish women a simple instrument describing usual meal types. Eur J Clin Nutr. 2002;56:740–7.
- Frankenfield DC, Rowe WA, Smith JS, Cooney RN. Validation of several established equations for resting metabolic rate in obese and non-obese people. J Am Diet Assoc. 2003;103:1152–9.
- Garcia-Garcia I, Narberhaus A, Marques-Iturria I, Garolera M, Radoi A, Segura B, Pueyo R, Ariza M, Jurado MA. Neural responses to visual food cues: Insights from functional magnetic imaging. Eur Eat Disord Rev. 2013;21(2):89–98. doi:10.1002/erv.2216.
- 39. Glanz K, Basil M, Maibach E, Goldberg J, Snyder D. Why Americans eat what they do: taste, nutrition, cost, convenience, and weight control concerns as influences on food consumption. J Am Diet Assoc. 1998;98(10):1118–26.
- 40. Glanz K, Rimer BK, Viswanath K. Health behavior and health education: theory, research, and practice. 2008. Retrieved 29 Feb 2016, from http://fhc.sums.ac.ir/files/salamat/health_education.pdf.
- Glasgow R, Toobert E, Barrera D, Strycker J. Assessment of problem-solving: a key to successful diabetes selfmanagement. J Behav Med. 2004;27(5):477–90. doi:10.1023/B:JOBM.0000047611.81027.71.
- Gluck ME, Venti CA, Salbe AD, Krakoff J. Nighttime eating: Commonly observed and related to weight gain in an inpatient food intake study. Am J Clin Nutr. 2008;88(4):900–5.
- Gordon-Larsen P. Food availability/convenience and obesity. Adv Nutr Int Rev J. 2014;5(6):809–17. doi:10.3945/ an.114.007070.
- 44. Gudzune KA, Doshi RS, Mehta AK, Chaudhry ZW, Jacobs DK, Vakil RM, Lee CJ, Bleich SN, Clark JM. Efficacy of commercial weight-loss programs: an updated systematic review. Ann Int Med. 2015;162(7):501–12. doi:10.7326/M14-2238.
- 45. Hamdy O, Zwiefelhofer D. Weight management using a meal replacement strategy in type 2 diabetes. Curr Diab Rep. 2010;10(2):159–64. doi:10.1007/s11892-010-0103-9.
- Harrold JA, Dovey TM, Blundell JE, Halford JC. CNS regulation of appetite. Neuropharmacology. 2012;63(1):3– 17. doi:10.1016/j.neuropharm.2012.01.007.
- 47. Herman CP, Polivy J. Normative influences on food intake. Physiol Behav. 2005;86(5):762-72.

- Higgs S. Social norms and their influence on eating behaviours. Appetite. 2015;86:38–44. doi:10.1016/j. appet.2014.10.021.
- 49. Hoek J, Gendall P. Advertising and obesity: a behavioral perspective. J Health Commun. 2006;11(4):409-23.
- Jakubowicz D, Barnea M, Wainstain J, Froy O. High caloric intake at breakfast vs. dinner differentially influences weight loss of overweight and obese women. Obesity (Silver Spring). 2013;21(12):2504–12. doi:10.1002/oby.20460.
- Jensen MD, Ryan DH, Donato KA, Apovian CM, Ard JD, Comuzzie AG, Hu FB, et al. Executive summary: guidelines (2013) for the management of overweight and obesity in adults. Obesity. 2014;22(S2):S5–39. doi:10.1002/oby.20821.
- Johnston BC, Kanters S, Bandayrel K, et al. Comparison of weight loss among named diet programs in overweight and obese adults: a meta-analysis. JAMA. 2014;312(9):923–33. doi:10.1001/jama.2014.10397.
- Key TJ, Appleby PN, Rosell MS. Healthy effects of vegetarian and vegan diets. Proc Nutr Soc. 2006;65(1):35–41. doi:10.1079/PNS2005481.
- 54. Kirpizidis H, Stavrati A, Geleris P. Assessment of quality of life in a randomized clinical trial of candesartan only or in combination with DASH diet for hypertensive patients. J Cardiol. 2005;46(5):177–82.
- 55. Kobayashi F, Ogata H, Omi N, Nagasaka S, Yamaguchi S, Hibi M, Tokuyama K. Effect of breakfast skipping on diurnal variation of energy metabolism and blood glucose. Obes Res Clin Pract. 2014;8:e201–98.
- 56. Kong A, Beresford SAA, Alfano CM, Foster-Schubert KE, Neuhouser ML, Johnson DB, Duggan C, Wang CY, Xiao L, Jeffery RW, Bain CE, McTiernan A. Self-monitoring and eating-related behaviors associated with 12-month weight loss in postmenopausal overweight-to-obese women. J Acad Nutr Diet. 2012;112(9):1428–35. http://doi.org/10.1016/j.jand.2012.05.014.
- Lavie CJ, Alpert MA, Arena R, Mehra MR, Milani RV, Ventura HO, et al. Impact of obesity and the obesity paradox on prevalence and prognosis in heart failure. JACC Heart Fail. 2013;1(2):93–102. doi:10.1016/j. jchf.2013.01.006.
- Lee JS, Visser M, Tylavsky FA, Kritchevsky SB, Schwartz AV, Sahyoun N, Harris TB, Newman AB. Weight loss and regain and effects on body composition: the Health, Aging, and Body Composition Study. J Gerontol A Biol Sci Med Sci. 2009;65(1):78–83. doi:10.1093/gerona/glp042.
- 59. Lin GM, Li YH, Lin CL, Wang JH, Han CL. Relation of body mass index to mortality among Asian patients with obstructive coronary artery disease during a 10-year follow-up: a report from the ET-CHD registry. Int J Cardiol. 2013;168(1):616–20. doi:10.1016/j.ijcard.2013.01.204.
- 60. Livingstone MB, Pourshahidi LK. Portion size and obesity. Adv Nutr Int Rev J. 2014;5(6):829–34. doi:10.3945/ an.114.007104.
- 61. Lowe MR, Butryn ML. Hedonic hunger: a new dimension of appetite? Physiol Behav. 2007;91(4):432-9.
- Luszczynska A, Sobczyk A, Abraham C, Kaplan RM. Planning to lose weight: randomized controlled trial of an implementation intention prompt to enhance weight reduction among overweight and obese women. Health Psychol. 2007;26(4):507–12. doi:10.1037/0278-6133.26.4.507.
- Mattfeldt-Beman MK, Corrigan SA, Stevens VJ, Sugars CP, Dalcin AT, Givi MJ, Copeland KC. Participants' evaluation of a weight-loss program. J Am Diet Assoc. 1999;99(1):66–71. doi:10.1016/S0002-8223(99)00018-8.
- 64. Mancini JG, Filion KB, Atallah R, Eisenberg MJ. Systematic review of the Mediterranean diet for long-term weight loss. Am J Med. 2015. doi:10.1016/j.amjmed.2015.11.028.
- 65. McDermott MS, Oliver M, Svenson A, Simnadis T, Beck EJ, Coltman T, Iverson D, Caputi P, Sharma R. The theory of planned behaviour and discrete food choices: a systematic review and meta-analysis. Int J Behav Nutr Phys Act. 2015;12:162. doi:10.1186/s12966-015-0324-z.
- Mehta T, Smith DL, Muhammad J, Casazza K. Impact of weight cycling on risk of morbidity and mortality. Obes Rev. 2014;15(11):870–81. doi:10.1111/obr.12222.
- 67. Montani JP, Schutz Y, Dulloo AG. Dieting and weight cycling as risk factors for cardiometabolic diseases: who is really at risk? Obes Rev. 2015;16(S1):7–18. doi:10.1111/obr.12251.
- 68. Morse SA, Ciechanowski PS, Katon WJ, Hirsch IB. Isn't this just bedtime snacking? The potential adverse effects of night-eating symptoms on treatment adherence and outcomes in patients with diabetes. Diabetes Care. 2006;29(8):1800–804.
- Muckelbauer R, Sarganas G, Gruneis A, Muller-Nordhorn J. Association between water consumption and body weight outcomes: a systematic review. Am J Clin Nutr. 2013;98(2):282–99.
- Murawski ME, Milsom VA, Ross KM, Rickel KA, DeBraganza N, Gibbons LM, Perri MG. Problem solving, treatment adherence, and weight-loss outcome among women participating in lifestyle treatment for obesity. Eat Behav. 2009;10(3):146–51. doi:10.1016/j.eatbeh.2009.03.005.
- 71. National Institutes of Health (NIH). DASH eating plan. 2015. Retrieved from https://www.nhlbi.nih.gov/health/ health-topics/topics/dash.
- Nicklas JM, Huskey KW, Davis RB, Wee CC. Successful weight loss among obese U.S. adults. Am J Prev Med. 2012;42(5):481–5. doi:10.1016/j.amepre.2012.01.005.
- Nonino-Borges CB, Martins-Borges R, Bavaresco M, Suen VM, Moreira AC, Marchini JS. Influence of meal time on salivary circadian cortisol rhythms and weight loss in obese women. Nutrition. 2007;23(5):385–91.

- Nowson CA, Wattanapenpaiboon N, Pachett A. Low-sodium Dietary Approaches to Stop Hypertension-type diet including lean red meat lowers blood pressure in postmenopausal women. Nutr Res. 2009;29(1):8–18.
- Nowson CA, Worsley A, Margerison C, Jorna MK, Godfrey SJ, Booth A. Blood pressure change with weight loss is affected by diet type in men. Am J Clin Nutr. 2005;81:983–9.
- Ochner CN, Barrios DM, Lee CD, Pi-Sunyer FX. Biological mechanisms that promote weight regain following weight loss in obese humans. Physiol Behav. 2013;120:106–13.
- Office of Disease Prevention and Health Promotion (ODPHP). 2015–2020 Dietary Guidelines for Americans. 2015. Retrieved from http://health.gov/dietaryguidelines/2015/.
- Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity among adults: United States, 2011–2012. NCHS Data Brief. 2013;131:1–8.
- 79. Oldways Health through Heritage (Oldways). Mediterranean diet pyramid. 2016. Retrieved at http://oldwayspt. org/resources/heritage-pyramids/mediterranean-pyramid/overview.
- Park J, Ahmadi SF, Streja E, Molnar MZ, Flegal KM, Gillen D, et al. Obesity paradox in end-stage kidney disease patients. Prog Cardiovasc Dis. 2014;56(4):415–25. doi:10.1016/j.pcad.2013.10.005.
- Perri M, Nezu A, McKelvey W, Shermer R, Renjilian D, Viegener B, Kendall PC. Relapse prevention training and problem-solving therapy in the long-term management of obesity. J Consult Clin Psychol. 2001;69(4):722–6. doi:10.1037/0022-006X.69.4.722.
- Peterson N, Middleton K, Nackers L, Medina K, Milsom V, Perri M. Dietary self-monitoring and long-term success with weight management. Obesity. 2014;22(9):1962–7. doi:10.1002/oby.20807.
- Phillips K, Wood F, Kinnersley P. Tackling obesity: the challenge of obesity management for practice nurses in primary care. Fam Pract. 2014;31(1):51–9. doi:10.1093/fampra/cmt054.
- Raatz SK, Wimmer JK, Kwong CA, Shalamar DS. Intensive diet instruction by registered dietitians improves weight-loss success. J Am Diet Assoc. 2008;108(1):110–3.
- Raynor HA, Champagne CM. Position of the academy of nutrition and dietetics: interventions for the treatment of overweight and obesity in adults. J Acad Nutr Diet. 2016;116(1):129–47. doi:10.1016/j.jand.2015.10.031.
- Redmon JB, Reck KP, Raatz SK, Swanso JE, Kwong CA, Ji H, Thomas W, Bantle JP. Two-year outcome of a combination of weight loss therapies for type 2 diabetes. Diabetes Care. 2005;28:1311–5.
- 87. Reid KJ, Baron KG, Zee PC. Meal timing influences daily caloric intake in healthy adults. Nutr Res. 2014;34(11):930–5.
- Riebl SK, Estabrooks PA, Dunsmore JC, Savla J, Frisard MI, Dietrich AM, Dietrich AM, Peng Y, Zhang X, Davy BM. A systematic literature review and meta-analysis: The Theory of Planned Behavior's application to understand and predict nutrition-related behaviors in youth. Eat Behav. 2015;18:160–78. doi:10.1016/j.eatbeh.2015.05.016.
- Robinson E, Fleming A, Higgs S. Prompting healthier eating: testing the use of health and social norm based messages. Health Psychol. 2014;33(9):1057. doi:10.1037/a0034213.
- Rogers PJ, Brunstrom JM. Appetite and energy balancing. Physiol Behav. 2016. http://dx.doi.org/10.1016/j. physbeh.2016.03.038.
- 91. Sacks FM, Bray GA, Carey VJ, Smith SR, Ryan DH, Anton SD, McManus K, Champagne CM, Bishop LM, Laranjo N, Leboff MS, Rood JC, de Jonge L, Greenway FL, Loria CM, Obarzanek E, Williamson DA. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. N Engl J Med. 2009;360(9):859–73.
- 92. Sallis JF, Glanz K. Physical activity and food environments: solutions to the obesity epidemic. Milbank Q. 2009;87(1):123–54. doi:10.1111/j.1468-0009.2009.00550.x.
- Scalfi L, Coltorti A, Sapio C, DiBiase G, Borrelli R, Contaldo F. Predicted and measured resting energy expenditure in healthy young women. Clin Nutr. 1993;12:1–7.
- Scully M, Dixon H, Wakefield M. Association between commercial television exposure and fast-food consumption among adults. Public Health Nutr. 2009;12(1):105–10. doi:10.1017/S1368980008002012.
- Shaikh AR, Yaroch AL, Nebeling L, Yeh MC, Resnicow K. Psychosocial predictors of fruit and vegetable consumption in adults. Am J Prev Med. 2008;34(6):535–43. doi:10.1016/j.amepre.2007.12.028.
- 96. Sharma A, Lavie CJ, Borer JS, Vallakati A, Goel S, Lopez-Jimenez F, Arbab-Zadeh A, Mukherjee D, Lazar JM. Meta-analysis of the relation of body mass index to all-cause and cardiovascular mortality and hospitalization in patients with chronic heart failure. Am J Cardiol. 2015;115(10):1428–34. doi:10.1016/j.amjcard.2015.02.024.
- Shen J, Wilmot KA, Ghasemzadeh N, Molloy DL, Burkman G, Mekonnen G, Gongora MC, Quyyumi AA, Sperling LS. Mediterranean dietary patterns and cardiovascular health. Annu Rev Nutr. 2015;35:425–49. doi:10.1146/annurev-nutr-011215-025104.
- Sleddens EF, Kroeze W, Kohl LF, Bolten LM, Velema E, Kaspers P, Kremers SP, Brug J. Correlates of dietary behavior in adults: an umbrella review. Nutr Rev. 2015;73(8):477–99. doi:10.1093/nutrit/nuv007.
- Skouroliakou M, Giannopoulou I, Kostara C, Vasilopoulou M. Comparison of predictive equations for resting metabolic rate in obese psychiatric patients taking olanzapine. Nutrition. 2009;25(2):188–93.
- St. Jeor ST, Cutter GR, Perumean-Chaney SE, Hall SJ, Herzog H, Bovee V. The practical use of charts to estimate resting energy expenditure in adults. Topics Clin Nutr. 2003;19:51–6.

- Steenhuis IH, Waterlander WE, de Mul A. Consumer food choices: the role of price and pricing strategies. Public Health Nutr. 2011;14(12):2220–6. doi:10.1017/S1368980011001637.
- 102. Stookey JD, Constant F, Gardner CD, Popkin BM. Replacing sweetened caloric beverages with drinking water is associated with lower energy intake. Obesity. 2007;15(12):3013–22.
- Sobal J, Wansink B. Kitchenscapes, tablescapes, platescapes, and foodscapes influences of microscale built environments on food intake. Environ Behav. 2007;39(1):124–42.
- 104. Spronk I, Kullen C, Burdon C, O'Connor H. Relationship between nutrition knowledge and dietary intake. Br J Nutr. 2014;111(10):1713–26. doi:10.1017/s000711451400008743.
- 105. Summerbell CD, Moody RC, Shanks J, Stock MJ, Geissler C. Relationship between feeding pattern and body mass index in 220 free-living people in four age groups. Eur J Clin Nutr. 1996;50:513–9.
- 106. Tate D, Jackvony E, Wing R. Effects of internet behavioral counseling on weight loss in adults at risk for type 2 diabetes: a randomized trial. JAMA. 2003;289(14):1833–6.
- 107. Tsai AG, Wadden TA. Systematic review: an evaluation of major commercial weight loss programs in the United States. Ann Intern Med. 2005;142(1):56–66.
- 108. Tsai AG, Wadden TA. Treatment of obesity in primary care practice in the United States: a systematic review. J Gen Intern Med. 2009;24(9):1073–9. doi:10.1007/s11606-009-1042-5.
- United States Department of Agriculture (USDA). Choose my plate. 2016. Retrieved from http://www.choosemyplate.gov.
- 110. US News and World Report (US News). Best weight loss diets. 2016. Retrieved from http://health.usnews.com/ best-diet/best-weight-loss-diets.
- Vartanian LR. Impression management and food intake. Current directions in research. Appetite. 2015;86:74–80. doi:10.1016/j.appet.2014.08.021.
- 112. Wadden TA, Butryn ML, Hong PS, Tsai AG. Behavioral treatment of obesity in patients encountered in primary care settings: a systematic review. JAMA. 2014;312(17):1779–91. doi:10.1001/jama.2014.14173.
- 113. Wadden TA, Neiberg RH, Wing RR, Clark JM, Delahanty LM, Hill JO, Krakoff J, Otto A, Ryan DH, Vitolins MZ. Four-year weight losses in the Look AHEAD study: factors associated with long-term success. Obesity (Silver Spring, Md.). 2011;19(10):1987–98. doi:10.1038/oby.2011.230.
- 114. Wadhera D, Capaldi-Philips ED. A review of visual cues associated with food on food acceptance and consumption. Eating Behav. 2014;15(1):132–43. doi:10.1016/j.eatbeh.2013.11.003.
- 115. Wansink B. Environmental factors that increase the food intake and consumption volume of unknowing consumers. Annu Rev Nutr. 2004;24:455–79.
- 116. Wansink B, Kim J. Bad popcorn in big buckets: portion size can influence intake as much as taste. J Nutr Educ Behav. 2005;37(5):242–5.
- 117. Weijs PJ. Validity of predictive equations for resting energy expenditure in US and Dutch overweight and obese class I and II adults aged 18–65 years. Am J Clin Nutr. 2008;88(4):959–70.
- 118. Whybrow S, Mayer C, Kirk TR, Mazlan N, Stubbs RJ. Effect of two weeks' mandatory snack consumption on energy intake and energy balance. Obesity (Silver Spring). 2007;15(3):673–85.
- 119. White K, Dahl DW. To be or not be? The influence of dissociative reference groups on consumer preferences. J Consumer Psychol. 2006;16(4):404–14.
- 120. Wing RR, Jeffery RW, Burton LR, Thorson C, Nissinoff KS, Baxter JE. Food provision vs. structured meal plans in the behavioral treatment of obesity. Int J Obes Relat Metabol Dis. 1996;20(1):56–62.
- 121. Wohlfahrt P, Lopez-Jimenez F, Krajcoviechova A, Jozifova M, Mayer O, Vanek J, et al. The obesity paradox and survivors of ischemic stroke. J Stroke Cerebrovasc Dis. 2015;24(6):1443–50. doi:10.1016/j. jstrokecerebrovasdis.2015.03.008.
- 122. Young LR, Nestle M. The contribution of expanding portion sizes to the US obesity epidemic. Am J Public Health. 2002;92(2):246–9.
- 123. Zaveri S, Drummond S. The effect of including a conventional snack (cereal bar) and a nonconventional snack (almonds) on hunger, eating frequency, dietary intake and body weight. J Hum Nutr Diet. 2009;22:461–8.
- 124. Zheng H, Lenard NR, Shin AC, Berthoud HR. Appetite control and energy balance regulation in the modern world: reward-driven brain overrides repletion signals. Int J Obes (Lond). 2009;33(S2):S8–13. doi:10.1038/ ijo.2009.65.
- 125. Zolfani SH, Rezaeiniya N, Pourhossein M, Zavadskas K. Decision making on advertisement strategy selection based on life cycle of products by applying FAHP and TOPSIS GREY: growth stage perspective; a case about food industry in IRAN. Engineer Econ. 2012;23(5):471–84.

Chapter 10 Nutrition in Oral Health

Atheer Yacoub and Wahida Karmally

Key Points

- Prevention of oral diseases such as dental caries and periodontitis begins with early nutrition intervention.
- Poor oral health is associated with a myriad of systemic diseases.
- Maintaining oral hygiene and a healthy eating pattern and lifestyle can help prevent oral diseases.
- The use of multiple medications increases risk of oral problems by compromising nutrient bioavailability.
- Functional foods, polyphenols, and probiotics may contribute to a healthy oral mucosa.

Keywords Nutrition • Oral health • Macro and micronutrients • Dental caries • Periodontal disease

Introduction

"Diet and Nutrition are major multifactorial environmental factors in the etiology and pathogenesis of craniofacial diseases and disorders."

Surgeon General's Report on "Oral Health in America" 2000 [1].

Oral health is essential to general health and well-being. Diet and nutrition play a key role in tooth development, gingival and oral tissue integrity, bone strength, and prevention and management of diseases of the oral cavity. In June 2003, Centers for Disease Control and Prevention (CDC) issued a national "Call to Action to Promote Oral Health" to address the country's oral health needs in the twenty-first century [2].

The goals of the Call to Action are to

- Promote oral health
- Improve quality of life
- Eliminate oral health disparities

A. Yacoub, MS, RDN • W. Karmally, Dr PH, RD, CDE, CLS, FNLA (⊠) Irving Institute for Clinical and Translational Research, Columbia University Medical Center, 622 West 168th St, PH10-310, New York, NY 10032, USA e-mail: aay2107@cumc.columbia.edu; wk2@cumc.columbia.edu

Dental disease is a worldwide problem, both in developed and developing countries, resulting in a global economic burden due to direct treatment costs and indirect costs related to decreased work productivity. Estimates from the 2010 Global Burden of Disease study found that the yearly global indirect and direct costs for oral diseases amounted to \$144 billion and \$298 billion, respectively [3]. Two of the most common and prevalent infectious oral diseases include dental caries and periodontal diseases; however, poor oral health is associated with a myriad of other diseases such as diabetes, autoimmune disorders, human immunodeficiency virus (HIV), eating disorders, cardiovascular disease, and oropharyngeal cancer [4]. Medication regimens can also increase risk of oral problems [5]. Medical nutrition therapy can reduce the risk of oral infections and improve the outcome of treatment of patients with oral manifestations of acute and chronic diseases.

Nutrition and oral health are interdependent and influence individuals' overall health status in numerous ways throughout the lifecycle. Oral health extends beyond teeth. Good health begins in the mouth for a very simple reason. The mouth is where nutrition begins; digestion begins and is a mirror of systemic health. The mouth is the beginning of the gastrointestinal (GI) tract.

Scientific and epidemiologic data suggest a lifelong synergy between nutrition and oral health status in health and disease. Nutrition and dietary patterns can affect the development and integrity of the mouth and progression of oral diseases. The soft tissues of the mouth are among the first to develop clinical manifestations of nutrient deficiencies. The links between oral health and nutrition are emerging and complex. Diet and nutrition may affect the development and progression of diseases of the oral cavity which, in turn can affect nutritional status. Nutritional deficiencies may cause dental problems in that they affect bony tissue synthesis and modify the resistance of the gingival tissues to plaque microorganisms [4].

Nutrition plays protective and preventive roles in oral health. The protective role is in promoting healthy development and maintenance of the mouth's tissues and their natural protective mechanisms. The role of nutrition is also to prevent oral disease through the influence of the foods' properties on plaque development and saliva flow as well as the management of chronic diseases such as diabetes mellitus. A healthy eating pattern that includes a variety of foods to optimize nutrient consumption is of importance to maintain oral health. Specific foods no longer are singled out as major risk factors for dental caries.

The direct relationship between diet and dental caries is clearly established. The primary factors to consider in determining the cariogenic, cariostatic and anticariogenic properties of the diet are the food form (liquid, solid and sticky and long lasting), frequency of consumption of sugar and other fermentable carbohydrates, nutrient composition, sequence of food intake, and combination of foods. Major components of a preventive dental regimen include nutrition counseling for a healthy eating pattern, fluoride therapy, and use of sealants and control of cariogenic bacteria.

Periodontal disease increases risk for nutritional deficiencies because the infection can alter tissue capacity to utilize nutrients needed for healing and repair. Optimizing nutritional status and eating patterns combined with removal of the stimuli of inflammatory periodontal lesions are important in the treatment of periodontal disease.

Dental Caries

Dental caries is an infectious disease of teeth in which organic acid metabolites lead to gradual demineralization of enamel and destruction of tooth structure. Although dental caries is largely preventable, it is the most common oral disease and the most common chronic disease in children, and it also affects older adults. Maintaining oral hygiene such as brushing and flossing regularly is important to remove bacterial plaque and prevent tooth decay. Oil pulling (swishing oil in the mouth) is a method from ancient India also used to prevent tooth decay and protect the oral cavity [6]. A randomized study in 20 adolescent boys with plaque-induced gingivitis compared the effects of oil pulling (using sesame oil) and chlorhexidine mouthwash, on plaque. The study participants were divided into either group. A significant decrease in plaque and modified gingival index scores were found in both groups. There was not a statistical difference in effectiveness on plaque in either method. Oil pulling can be used as a cost-effective, preventive home method for oral hygiene [7].

Dental caries development depend on specific bacteria such as Streptococcus mutans, salivary constituents with dietary fermentable carbohydrates, and host factors that include genetics, behavior, race and age and oral hygiene, use of fluoride and dietary patterns. Development of teeth usually begins between the 6th and 8th weeks of gestation and continues to develop throughout the pregnancy. Taking the appropriate measures to prevent dental caries should begin very early on, as teeth become vulnerable shortly after they erupt.

Children put to bed with a bottle of milk or sweetened liquid or sip on sweetened drinks throughout the day, increase their risk of Early Childhood Caries (ECC) (formerly known as baby bottle tooth decay). This condition affects 10% of 2 year olds, putting them at greater risk of permanent teeth decay. Low intake of energy and protein can also inhibit the proper development of teeth by delaying eruption, and affecting the size and solubility of the enamel and can also lead to salivary gland dysfunction [8].

Sugar-sweetened beverages (SSB) such as sodas and 100% fruit juice, along with certain eating patterns, may lead to severe-ECC (S-ECC). If left untreated, caries can have severe consequences related to pain which can interfere with eating and result in failure to thrive. Children living in poverty are at greater risk for S-ECC and untreated tooth decay. In a cross-sectional study, the relationship between SSB and severe-ECC in children from low-income households was assessed. Dietary data were collected through 24-h recalls and food frequency questionnaires (FFQ) from 454 children with S-ECC and 428 caries-free children. Children with S-ECC consumed statistically more added sugar, on average, and 3.2–4.8 fluid ounces more of SSB per day than those without S-ECC. The study also found that children were 4.6 times more likely to have S-ECC if they consumed 5 oz. of SSB per day compared to children who consumed 1 oz. or less per day. Soda was found to have a more harmful effect than 100% fruit juice, even when sugar content was similar. This is possibly due to the higher acid content in soda, which leads to erosion and demineralization of tooth enamel [9].

The World Health Organization (WHO) conducted a systematic review to establish an updated guideline on the restriction of sugar intake. The review measured the effect of restricting sugar intake to <10% of energy and <5% of energy on dental caries incidence. The review showed moderate quality of evidence for restricting sugar to <10% of energy to reduce risk of dental caries. Reducing sugar intake to <5% of energy may also help further reduce dental caries risk [10].

Breastfeeding should be encouraged and children should not be left to fall asleep with a bottle containing milk or beverages other than water. Frequency of sugar consumption is more of a risk factor for caries than the amount of sugar; therefore, children should be discouraged from sipping any beverages other than water throughout the day. Juices should be diluted with water to reduce sugar exposure. It is important to teach children healthful eating habits at a young age to prevent childhood caries or other oral diseases during adulthood [11].

Cariogenic and Anticariogenic Foods

The common culprits that increase risk of dental caries include simple sugars such as sweetened carbonated beverages, sweetened tea and coffees, fruit drinks, and energy drinks. In addition to beverages, other simple sugars that increase risk include sucrose and molasses [4]. A study included a sample of 60 healthy, primary teeth donated by children ages 6–7 and the teeth were divided into three groups, with 20 samples in each. Group 1 was kept in 20 ml of honey for 10 min, Group 2 was kept in 20 ml of molasses for 10 min, and Group 3 was kept in 20 ml of orange juice for 10 min. The samples were then kept in 20 ml of saliva for 1 h. In order to mimic the oral environment, this cycle was repeated 180 times. The samples were measured for enamel surface roughness at baseline and after the procedure. Results indicated that molasses and orange juice had a statistically significant impact on dental erosion, whereas honey did not cause a significant change [12]. Antibacterial properties have been attributed to honey [13].

Healthful eating patterns to reduce risk include spacing food and beverage intake by at least 2 h, chewing sugarless gum briefly after eating, and consuming whole, unprocessed foods to stimulate salivation [4]. Saliva helps prevent collection of bacteria on teeth, while clearing food particles, sugars, and bacteria from the oral cavity, and plays an important role in remineralization of teeth and protection of tooth enamel as a buffer to prevent bacterial growth [8, 12]. Calcium and phosphate in saliva may prevent formation of bacteria on teeth enamel [14]. Foods high in protein such as meats, eggs, cheese, fish, beans, and legumes are also protective dietary components, along with whole-grain, low-sugar breads and cereals in the prevention of dental caries. Oral soft tissues are one of the first tissues in the bodies to experience nutrient deficiency, especially with water-soluble vitamins (B vitamins and vitamin C), protein and iron [4].

Sugar Alcohols and Dental Caries

While sugar and fermentable carbohydrates contribute to enamel erosion, certain sugar alcohols, such as xylitol, have the opposite effect. This white crystalline five-carbon sugar polyol is found naturally in fruit, vegetables, and berries and can be artificially made from xylan-containing plants like birch and beechwood. Xylitol is a commonly used nonnutritive sweetener in sugar-free gum and its role in reducing risk of dental caries has been well established. In addition to promoting saliva, which mineralizes teeth and neutralizes acid, xylitol prevents harmful microorganisms from attaching to teeth and producing harmful acids. It also helps fight cariogenic bacteria, plaque, xerostomia, gingival inflammation, and teeth erosion.

In order to provide protection against dental caries, a dose of 6–10 g/day of xylitol is recommended. While an amount of 40 g/day is tolerated by most adults, it can cause diarrhea in doses of 100 g/day for adults and 45 g/day for children. The recommended time for chewing xylitol-containing gum is 20 min after eating [15].

Several studies have shown regular use of chewing gum with xylitol significantly decreased risk of dental caries. The effect of chewing sugar-free gum on dental plaque was studied in 20 participants randomized to either the sugar-free or sugar-containing gum, which they were required to chew for 30 min, three times a day after meals. They were also instructed to refrain from brushing teeth or using mouth rinses during the study period. The average plaque scores were significantly higher in the sugar-containing gum group than the sugar-free one. The sugar-free gum consisted of various alternative sweeteners and other ingredients such as mannitol, xylitol soya lecithin, aspartame, acesulfame K, glazing agent, and a freshening agent. The decrease in plaque seen in this group is likely a result of increased saliva production from chewing as well as anticariogenic ingredients of sugar alcohols [16].

Diet Quality and Dental Caries

The Healthy Eating Index-2005 (HEI-2005) is a measure of diet quality, which includes all of the major food groups: total fruit, total vegetables, total grains, milk, meat and beans, and other components such as whole fruit, dark green and orange vegetables, legumes, whole grains, oils, saturated fat,

sodium, solid fat, alcohol, and added sugar, and is used to assess diet quality. In a cross-sectional study, the HEI-2005 was used to determine the risk of ECC in 60 healthy children aged 2–6 years. The participants were divided into three groups: 20 caries-free children, 20 with ECC, and 20 with S-ECC. After averaging HEI-2005 scores, the only components that were statistically different among the three groups were whole fruit, milk, and sodium. The average score of these components was statistically higher in the noncaries group than the other two groups. Though not statistically significant, scores for meat and beans were highest in the caries-free group. Whole-fruit, dairy, and protein-rich foods have been shown to have anticariogenic effects in various studies. Meeting dietary recommendations and consuming adequate amounts of anticariogenic foods, while limiting cariogenic foods can reduce the risk of dental caries [17].

Eating Disorders and Oral Health

Unlike dental caries, dental erosion from eating disorders is not caused solely by bacteria. Bulimia nervosa may cause dental erosion and demineralization from acidic contents of gastric reflux or self-induced vomiting. Additionally, binging on citrus fruit, softs drinks, or sports drinks may also cause dental erosion. Starvation related to anorexia nervosa and self-induced vomiting may cause dry mouth and hyposalivation, which are both risk factors for dental caries. Individuals with anorexia nervosa are also at risk for many nutrient deficiencies, which can lead to other oral problems (Table 10.1) that can impact consumption of nutrient-rich foods. In a meta-analysis, individuals with eating disorders had five times greater risk of dental erosion than those without disordered eating. Self-induced vomiting posed the greatest risk for erosion. Even individuals with eating disorders without vomiting had a significantly higher risk than those without eating disorders [25].

Tooth Loss and Nutrition

Tooth loss can negatively impact nutritional status by making it more difficult to consume adequate energy and meet nutrient needs, especially in older populations. This can also increase risk of developing other chronic diseases and decrease overall quality of life. The relationship between the total number of natural teeth and diet was studied, using data from the National Health and Nutrition Examination Survey (NHANES). The study included data from 9140 participants who were divided into three groups, according to dentition: full dentition (28 teeth), moderate dentition (21–27 teeth), and poor dentition (20 teeth or less). Energy intake, nutrient intake, and HEI-2005 scores were assessed to determine diet quality. There was a significant inverse relationship between energy intake and number of natural teeth and a significant positive correlation between total number of natural teeth and diet quality, as measured by the HEI-2005 score. Poor dentition was also

Oral condition	Associated nutritional deficiencies	
Stomatitis	Niacin, folic acid, vitamin B12	
Glossitis	Niacin, folic acid, B6, and vitamin B12	
Xerostomia	Vitamins A and B 12	
Cheilosis	Iron, B vitamins	
Gingival bleeding	Vitamins C and K	
Glossodynia	B vitamins, zinc, iron	

Table 10.1 Nutritional deficiencies associated with oral health [18–24]

associated with a lower intake of protein and most micronutrients, with a higher intake of carbohydrates. The full dentition group had a significantly lower intake of sodium than the other two groups [26].

A systematic review also showed that having fewer than 20 teeth with nine to 10 pairs of contacting units was related to a decrease in chewing ability and efficiency. Beyond just chewing ability, tooth loss further leads to alveolar bone loss which weakens mandibular muscles. This makes chewing more difficult even for denture wearers, which can negatively affect diet and food choices. As a consequence, individuals with tooth loss tend to have lower intakes of fruits, vegetables, and fiber, with higher intakes of saturated fat and cholesterol. This can further increase risk for dental-related diseases such as heart disease, obesity, and diabetes [27].

Periodontal Disease

Periodontal disease is an inflammation of periodontal issue and the second most common type of oral disease, which can vary in severity from gingivitis, to the more severe periodontitis. Risk factors associated with periodontal disease include gender, smoking, alcohol intake, diabetes, obesity and metabolic syndrome, osteoporosis, dietary calcium, vitamin D levels, stress, and genetics [28]. Periodontitis affects about 47.2% of adults \geq 30 years old in the United States and gingivitis is even more common, affecting about half of the population. Gingivitis can be reversible and does not always lead to periodontitis, whereas periodontitis can lead to tooth loss and destruction [29]. Tooth loss is a result of gingival bleeding and recession, deep pockets between the gingiva and tooth, and deterioration of supportive tissue [4, 29].

Some of these chronic diseases can be explained by nutrigenomics, which studies the relationship between nutrition and the genome, while providing scientific reasoning for the role of diet in improving public health. The risk factors for many diseases, including periodontal disease, may be determined by the interaction between nutrition and genes. While there is an interrelationship between type 2 diabetes and periodontal disease, it may be explained by genetic components that are affected by diet. For example, in type 2 diabetes, insulin storage and release may be influenced by a zinctransporter gene. Identifying zinc's role in risk for type 2 diabetes may help treat or prevent this disease and in turn, affect periodontal disease through changes in gene expression [30].

Diabetes and Periodontitis

Periodontitis is linked to other systemic diseases such as diabetes, obesity, and heart disease.

This is due to the shared metabolic response between these diseases, such as the spread of infection, toxins, and inflammation originating from bacteria present in the oral cavity [31]. Studies have shown a two-way relationship between diabetes and periodontitis, where patients with diabetes and periodontal disease exhibit poorer glycemic control and conversely, increased severity of periodontal disease and destruction of tissue is seen in individuals with diabetes. Patients with diabetes are three times more likely to get periodontitis than those without diabetes, especially those with higher glycated hemoglobin (HbA1c) levels [29, 32]. Periodontitis is a major risk factor for diabetes.

The National Health and Nutrition Examination Survey 2009–2010 found that 12.5% of patients with periodontal disease had diabetes, compared to 6.3% of the population without periodontitis. Though it has been shown that periodontitis is linked to a slight increase in HbA1c, the exact causal

relationship between periodontitis and diabetes is not certain; however, it is presumed that periodontitis contributes to poorer glycemic control by increasing insulin resistance due to inflammation and triggering an immune response [29].

Obesity and Periodontitis

Inflammation resulting from oral bacteria may contribute to obesity and metabolic syndrome by triggering oxidative stress and disruption of the mitochondria. Obesity further exacerbates insulin resistance and contributes to uncontrolled glucose levels [33]. Obesity, defined as having a body mass index (BMI) of \geq 30 kg/m², is not only a risk factor for diabetes and cardiovascular disease but it was also found to be an independent risk factor for periodontal disease. There is a positive association between BMI and attachment loss. Adipose tissue, which is present in greater amounts in obese individuals, produces proinflammatory cytokines (interleukin 6 [IL-6] and tumor necrosis factor alpha [TNF- α]) and increase circulation of reactive oxygen species (ROS). This results in increased risk of periodontitis and exacerbation of existing periodontitis. An analysis from the US Third National Health and Nutrition Examination Survey (NHANES III) showed that abdominal obesity may be a greater risk factor for periodontitis than subcutaneous fat. Obesity also increases insulin resistance, which can lead to type 2 diabetes and further increases the risk of periodontitis [34].

In a meta-analysis, the relationship between chronic periodontal disease and obesity was assessed. Multiple studies across the world, in diverse populations, showed a positive association between periodontal disease and obesity. This association was particularly greater in women, non-smokers, and a younger population, compared to adults. A two-way relationship between periodontal disease and obesity may occur, due to inflammation related to periodontal disease, which may worsen metabolic syndrome. Some older adults with periodontal disease may have lower BMIs related to decreased energy intake from tooth loss [35].

Cardiovascular Disease and Periodontitis

While there is an association between cardiovascular disease and periodontal disease, which is supported by several observational studies, the American Heart Association (AHA) reviewed the evidence to determine whether there is a causative relationship between periodontal disease and atherosclerotic vascular disease (ASVD). The two diseases have common risk factors, such as type 2 diabetes, smoking, and age. Although short-term studies have shown that treatment of periodontal disease results in reduced systemic inflammation and improved endothelial function, a causative relationship cannot be established [36].

More recently, a study investigated whether there is an independent relationship between periodontal disease and the risk of a first myocardial infarction (MI) in the PAROKRANK study. This large case-control trial included 805 patients under the age of 75 who had experienced an MI and they were matched with controls (age 62 ± 8) without MI. Standardized dental examinations, including panoramic X-ray, were conducted on both groups to determine their periodontal status, defined as either healthy (\geq 80%), moderate (79–66%), or severe (<66%). Results showed that 43% of patients who had a history of a MI also had periodontal disease, compared to 33% in the control group. Subjects with moderate to severe periodontal disease had a significantly higher risk of MI, as measured by radiographic bone loss. This further supports an independent relationship between periodontal disease and MI, but does not prove a causative effect [37].

Dietary Omega-3 Fatty Acids and Periodontitis

The anti-inflammatory effect of omega-3 fatty acids and its role in lowering heart disease risk has been widely researched. Food sources of the longer chain omega-3 fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are fatty fish such as salmon, sardines, trout, tuna, and herring. Alpha-linolenic acid (ALA) is an essential fatty acid that is found in plant-based foods such as walnuts, flax, chia, hemp, and soybean and canola oil. The US Dietary Reference Intake recommends 1.1–1.6 g/day or 0.6–1.2% of ALA per day. The Academy of Nutrition and Dietetics recommend an intake of 0.6–1.2% of ALA and 500 mg of EPA and DHA per day. The American Heart Association recommends consuming fatty fish at least twice a week [38].

In addition to having a protective effect on cardiovascular disease, omega-3 fatty acids may also have a direct benefit on periodontal disease. A review of data including 9,182 adults over the age of 20 years from the National Health and Nutrition Examination Survey (NHANES) between 1999 and 2004 assessed the relationship between omega-3 fatty acids intake and prevalence of periodontitis. Intakes of DHA, EPA, and ALA, measured through 24-h recalls and self-reported supplement use, showed an inverse relationship between DHA intake and periodontal disease. A similar association was seen with EPA intake, but to a lesser degree than with DHA [39].

The emerging treatment for periodontal disease that focuses on the host's response to infection is known as host modulatory therapy (HMT). This therapy aims to reduce inflammation and tissue destruction, while promoting wound healing and regeneration of the periodontium. This treatment was examined in a study that included 60 patients with moderate and severe periodontitis, without systemic diseases or infection, between the ages of 30 and 60 years. Participants were randomized to either the treatment group (TG), who received 300 mg of omega-3 (DHA and EPA) and nonsurgical periodontal therapy (scaling and root planning) or the control group (CG), who received 300 mg of a laxative containing liquid and the same nonsurgical periodontal therapy. All subjects were followed up with at 6 and 12 weeks and were assessed for periodontal parameters and C-reactive protein (CRP), an inflammatory biomarker. Results showed that the treatment group had significantly better periodontal outcomes compared with the control at 12 weeks, but there was not a significant difference between CRP levels in either group. Omega-3 fatty acids may have a protective effect on periodontal disease, while reducing inflammation that leads to tissue damage [40].

Dietary Fiber and Oral Health

Similar to omega-3 fatty acids, consumption of dietary fiber may reduce inflammation related to risk factors of periodontal disease such as cardiovascular disease, obesity, and diabetes. Fiber intake also plays a positive role in these diseases by improving lipids, blood pressure, and BMI. The effect of a high-fiber, low-fat diet on periodontal disease in slightly obese and/or individuals with prediabetes, was studied in 21 subjects who either had a BMI of ≥ 25 kg/m² or elevated glucose measured by an oral glucose tolerance test (OGTT). Subjects were given high-fiber, low-fat test meals three times per day, followed by an OGTT, for 8 weeks. Calorie needs were based on 30 kcal/kg/day. Results of the study showed improved periodontal disease markers at the end of the follow-up period; however, mild reductions in body weight and improved glucose tolerance, returned to baseline at the end of follow-up [41]. Dietary fiber may play a protective role in reducing inflammatory markers in periodontal disease.

Unlike simple sugars, fiber is an indigestible carbohydrate that does not have a cariogenic effect on teeth. Foods that are high in dietary fiber may provide a direct benefit in preventing oral health diseases by helping remove plaque from teeth, while also stimulating more chewing which increases

salivary flow [42, 43]. A study including 625 men aged 65 years and older, observed the effect of total fiber intake on periodontal disease by measuring alveolar bone loss (ABL). Sources of fiber were considered good or excellent if they provided 2.5 g or more per serving. Results showed a higher fiber intake, especially from fruits, was associated with less ABL and tooth loss [43].

The Academy of Nutrition and Dietetics recommends consuming fiber from various plant food sources. Adequate intake of fiber is linked to lower risk of chronic diseases. The recommended intake of fiber is based on 14 g of total fiber per 1,000 kcal, or 25 g for adult females and 38 g for adult males [44]. Only 5% of the population in the United States meets these recommendations, with the average intake being 17 g/day. Good sources of fiber include whole grains, legumes, vegetables, fruits, and nuts [45].

Fluoride and Oral Health

Fluoride is found in soil, water, plants, and animals and is a regular part of all diets. This natural element is important to maintaining the health of teeth and bone and 99% of its presence in the body is in hard tissues. Fluoride is also one of the most effective ways to prevent dental caries for children and adults. It helps promote oral health by contributing to tooth mineralization, reducing teeth sensitivity, and preventing acid-producing bacteria that causes dental caries. Fluoride's action against acid demineralization begins during teeth development before eruption. Once teeth erupt, fluoride continues to mineralize teeth and neutralize acid via its presence in saliva. Primary protection against dental caries occurs after teeth have erupted and by using topical fluoride agents. During the posteruptive phase, fluoride helps block bacteria which produces acid onto teeth and causes breakdown of enamel. Fluoride can be found in drinking water, tea, topical gels, oral rinses and toothpaste, and dietary supplements. Tap and bottled water, including beverages made with fluoridated water (i.e., tea, coffee, and juice) make up 80% of dietary fluoride [46]. While the majority of homes in the United States have fluoridated water, many children rely on intake from other sources such as processed foods, beverages, and toothpaste. Earlier recommendations from the Centers for Disease Control (CDC) and American Dental Association (ADA) for fluoride in drinking water ranged from 0.7 to 1.2 ppm; however, upon later review, this was lowered to 0.7 ppm to avoid risk of dental fluorosis [47]. This condition occurs when too much fluoride is ingested during tooth development causing mild small white spots on the teeth to more severe discoloration [46].

Micronutrients and Oral Health

Calcium and Vitamin D

Calcium is an important mineral for skeletal health and it makes up over 99% of bones and teeth. While the Recommended Dietary Allowance (RDA) for calcium is 1000–1200 mg/day, which varies with age and gender, most Americans are not meeting their needs. Vitamin D helps regulate calcium and enhances its absorption, and it also plays an important role in maintaining musculo-skeletal and bone health. Vitamin D does not occur naturally in many foods. The main dietary sources of calcium for several people include foods fortified with vitamin D, such as dairy products, milk substitutes, and cereal. Sun exposure and supplements are also major sources [48]. Vitamin D's anti-inflammatory properties along with its role in bone and mineral metabolism may have a positive effect on dental health. This is particularly important in periodontitis, where tooth loss may

occur as a result of alveolar bone reabsorption. While the relationship between osteoporosis and vitamin D has been established, there may be evidence that osteoporosis is an early sign of periodontitis and vice versa [49].

Periodontal health was assessed by alveolar bone loss, pocket depth, and attachment loss in 562 men with an average age of 62.9 years. Participants in the Department of Veterans Affairs Dental Longitudinal Study between 1986 and 1998 were examined one to four times to study the effect of recommended intake of \geq 800 IU of vitamin D on periodontal health. The dietary intakes of subjects were assessed using a Harvard University food frequency questionnaire to capture total vitamin D intake from food, and supplements. The study showed an inverse relationship between total vitamin D intake of \geq 800 IU and risk of periodontal disease. Subjects who took vitamin D supplements were found to have less periodontal disease than those who did not use supplements, but the difference was not statistically significant [50].

The effect of various doses of vitamin D on gingivitis during a 3-month period was examined in 88 participants who were divided into one of four groups: 2000 IU for group A, 1000 IU for group B, 500 IU for group C, and group D received a placebo. Clinical examination during follow-up was conducted on days 30 and 60 to assess gingivitis severity as well as serum calcium to monitor vitamin D toxicity. Serum vitamin D level increased in each group in proportion to the dosage received. Gingivitis scores were significantly lower at follow up compared to baseline. The anti-inflammatory effects of all three groups receiving vitamin D were significantly higher compared to the placebo group. Improvements in gingivitis were seen earliest in group A, possibly due to serum vitamin D intakes at higher dosages revealed an earlier anti-inflammatory effect, there was not a significant difference between groups A and B or between B and C at the end of the study. It is hypothesized that anti-inflammatory levels may have stabilized after reaching serum vitamin D levels >32 ng/ml. This study indicated that an anti-inflammatory effect of vitamin D on gingivitis can be seen in doses of 500–2000 IU with serum vitamin D levels reaching >30 ng/ml. The beneficial effects from vitamin D supplementation can be seen in 2–3 months for patients suffering from gingivitis [51].

The relationship between calcium and vitamin D intakes and dental disease was studied in 106 women. Their diets were assessed for calcium and vitamin D adequacy in relation to their oral health status. They were divided into three groups: adequacy, insufficiency, and sufficiency. Results showed that higher calcium intakes were associated with lower risk of dental caries. The group with low calcium intake was mostly due to low consumption of milk and dairy products. All of the women had gingivitis and 71% had significantly low levels of vitamin D. Soft drink consumption may have also affected their oral health status, as 72% of women were found to consume them daily [52]. Consuming low-fat dairy can help maintain calcium and vitamin D levels, which have a protective effect on teeth.

Calcium may also aid in remineralization of teeth and prevent alveolar bone loss. The diets of 606 older Danish adults were assessed for calcium, vitamin D, and dairy intake using a diet history questionnaire. After adjusting for age, gender, vitamin D intake, and other numerous factors, results showed that that higher calcium and within recommendations (\geq 3 servings/day) of dairy intake were linked to lower plaque, but only in the higher (\geq 6.8 µg/day) vitamin D intake group. This is likely due to the increased calcium absorption in the presence of vitamin D [14].

Magnesium

Consumption of adequate magnesium is important in maintaining overall health. Magnesium deficiency may play a role in pathological diseases such as cardiovascular disease, diabetes, pre-eclampsia, sickle cell disease, and chronic alcoholism. Magnesium intake may also be associated with periodontal disease, particularly in relation to calcium intake [53]. A cross-sectional study found that a higher magnesium/calcium ratio was inversely related to level of periodontitis. A matched-pair analysis of the subjects showed those who were taking magnesium-containing medications had better periodontal outcomes compared to subjects not taking magnesium [54].

Vitamin C

Ascorbic acid, or vitamin C, is an antioxidant with a beneficial effect on oral health. In addition to having anti-inflammatory properties, vitamin C is also necessary for the formation of collagen, connective and osteoid tissues, and dentine [55]. It also stimulates periodontal ligament cells and helps strengthen connective tissue, responsible for attaching teeth to bone. Fruits and vegetables are two of the major sources of vitamin C. These foods include peppers, strawberries, broccoli, Brussels sprouts, sweet potatoes, oranges, grapefruits, and kiwi. It is important to consume a variety of foods rich in vitamin C, as inadequate intake can lead to deficiency. Low serum ascorbic acid has been shown to have a significant association with a higher prevalence of periodontal disease in a study with older Japanese men [56]. Vitamin C deficiency can also lead to gingivitis, bleeding gums, and attachment loss. The Recommended Dietary Allowance (RDA) for men and women \geq 19 years is 90 mg and 75 mg [57], respectively; however, various studies show that a higher intake of 200 mg may be warranted for optimal plasma concentrations [53].

Probiotics and Oral Health

While the effect of harmful bacteria on periodontitis and dental caries has been well established, probiotics which are living microorganism may play a beneficial role in human health, particularly in prevention of oral diseases. Probiotics use natural bacteria such as ones found in healthy mouths to protect teeth and gum against harmful bacteria. Prebiotics are made up of non-digestible oligosaccharides, which include inulin-type fructans, maltodextrin, fructooligosaccharides and galactooligosaccharides, and can alter the gut microbiome to benefit the host and improve health. Prebiotics and probiotics work together. The term 'synbiotics' is used to describe when they are both combined in the same product, which may increase the longevity of probiotic bacteria as well as their growth in the intestinal tract [58].

There are several different mechanisms of probiotics, depending on various conditions such as the strain or combinations of strains used, when they are administered, and the severity of the disease state. Certain strains of probiotics, such as L. reuteri, may improve gingivitis by inhibiting pathogenic bacteria and reducing plaque. Yogurt with active cultures can be a rich source of probiotics [58]. Probiotics may also be found in other dairy products such as cheese, kefir, milk, as well as dietary supplements. Various studies have indicated the potential role of probiotics in reducing harmful bacteria that contribute to oral diseases [59].

Polyphenols and Oral Health

Polyphenols are antibacterial and antioxidant metabolites which may have a protective effect on oral health. Dietary sources are mainly plant foods such as fruits, vegetables, seeds, and legumes. Other sources include whole grain cereals, chocolate, coffee, tea, curcumin, and red wine. In addition to their anti-inflammatory properties, polyphenols may improve the oral microflora by preventing growth

of harmful organisms, while promoting beneficial ones [60]. In vitro studies have indicated that polyphenols have antibacterial properties in the biofilm; however, human studies need to be conducted to confirm beneficial effects in the prevention and treatment of periodontal disease [61]. A more standardized method of assessing polyphenol intake is also needed, as different sources vary in the amount of phenolic compounds [60].

Functional Foods and Oral Health

Functional foods are a term for foods that have a beneficial effect beyond their nutritional content, which may also have a protective effect on oral health. Foods such as whole grains, garlic (allyl compound), soybeans, carrots (carotenoids), tomatoes (lycopene), tea (polyphenols), fiber, vegetables, and fruits are considered functional foods when consumed regularly. These food groups may contribute to healthy aging, prevention of chronic diseases, including chronic periodontitis and caries. Green tea is the most commonly consumed functional beverage worldwide and it is one of the richest sources of polyphenols amongst teas, with a concentration of 30-40% as opposed to only 3-10% in black tea [62].

The epidemiologic relationship between the daily intake of green tea and periodontal disease was observed through a comprehensive health examination in middle-aged Japanese men. The intake of green tea had a modest inverse correlation with attachment loss and probing depth [63]. Cross-sectional data from a baseline survey conducted for the Ohsaki Cohort 2006 study indicated an association of green tea consumption with decreased odds for tooth loss [64].

Tea is a good source of fluoride and with its polyphenol component gives it anticariogenic properties [65]. Green tea reduces the activity of salivary and bacterial enzymes involved in carbohydrate metabolism and fermentation. It also helps prevent the attachment of bacteria to the tooth enamel and protects it against oxidative stress. The polyphenol responsible for green tea's functional properties is called epigallocatechin-3-gallate (EGCG). One cup of green tea, containing 2.5 g of leaves in 200 mL of water, is reported to have about 90 mg of EGCG. Studies have also been carried out using green tea gels, mouth wash, and green tea chips [62].

Nutrition Counseling in Caries Prevention

Along with regular dental visits, nutrition counseling can play a beneficial role in preventing dental caries. Good oral health practices should begin early, before the eruption of teeth. Feeding practices such as using a nursing bottle or "sippy" cup as a pacifier can increase the risk of dental caries. After exclusive breastfeeding for the first 6 months, it is recommended to incorporate other food to increase micronutrient intake, especially iron, in the following 6–12 months. While the American Association of Pediatric Dentistry encourages breastfeeding, at-will breastfeeding for long periods of time or bottle feeding during sleep should be avoided [66]. Parents should be instructed on healthy dietary patterns and strategies to promote healthful eating and oral habits early on.

Nutrition Counseling for Periodontal Health

When counseling individuals on good oral health, it is important to emphasize a wholesome and balanced diet that incorporates all of the food groups and meets recommended intakes. Nutrients that are associated with maintaining periodontal health include folate, vitamin A, and vitamin C for connective tissue integrity, protein, calcium, and phosphorus for collagen, and omega-3 fatty acids and vitamin D for immune function. While consuming these individual nutrients has been shown to have improved outcomes in periodontal disease, more research data are needed to establish dietary recommendations. In addition to individual nutrients, a variety of food groups are also associated with improved periodontal health. Whole grains are good sources of fiber, B vitamins, and magnesium, and are associated with a lower risk of periodontal disease. On the other hand, refined grains are associated with a higher risk of periodontal disease [67]. The 2015–2020 Dietary Guidelines for Americans recommends 6 oz-equivalents of whole grains per day, with at least half coming from whole grain sources such as brown rice, quinoa, and oats. For example, a slice of whole wheat bread or ½ cup of cooked brown rice is equivalent to a 1 oz portion [66]. Dairy products are also good sources of nutrients such as calcium, vitamin D, phosphorus, magnesium, and B vitamins associated with lower risk of periodontal disease [68]. For adults, three servings of low-fat or non-fat dairy such as milk, yogurt, cheese, or fortified soy beverages should be consumed daily [66]. Adequate consumption of fruits and vegetables should also be encouraged, as they are good sources of dietary fiber, carotenoids, vitamin C, and folate, which are also associated with periodontal health [67].

The daily recommended intake of fruit is four servings (two cups) per day and five servings (2.5 cups) of vegetables per day. A variety of different colored vegetables are recommended to maximize nutrient intake. Protein foods such as fish, meats, poultry, eggs, soy products, and legumes (beans and peas) can also be a good source of nutrients that can lower risk of periodontal disease. The 2015–2020 Dietary Guidelines for Americans recommend 5.5 oz of protein foods per day, based on a 2000 kcal diet. Individuals should also be counseled on limiting intakes of added sugars, saturated fat and cholesterol, sodium, and alcohol. Calories from added sugars should be limited to 10% or less per day. A high intake of saturated fat and cholesterol can increase low density lipoprotein cholesterol, a risk factor for cardiovascular disease. Foods high in saturated fats and cholesterol such as butter, high fat dairy and high fat meats, should be replaced with nonfat/low-fat dairy, lean meats and unsaturated fats. Healthful sources of fat include olive oil, canola oil, avocados, and nuts. Excess sodium intake can increase blood pressure and should be limited to 2,300 mg/day for healthy individuals and 1,500 mg/day for individuals with hypertension [68]. Eating a plant-based balanced diet and meeting dietary recommendations is not only important to oral health, but it also beneficial for overall health and disease prevention. By reducing risk of periodontal disease, the risk of its related diseases such as heart disease, diabetes, and obesity may also be reduced.

Lifestyle Factors and Oral Health

Tobacco use and alcohol overconsumption are risk factors for oral health diseases as well as many other chronic diseases. Smoking, in particular, can have many damaging effects on the body, which may lead to cardiovascular diseases, lung cancer, and respiratory diseases. Smoking lowers the immune system, making the body more susceptible to oral infection, while prolonging healing of tissue from surgery. This can expose the body to a host of diseases and infections, therefore increasing the likelihood of periodontal disease, while decreasing the likelihood of successful treatment and recovery. The risk of developing oral cancer is six times higher in smokers than in nonsmokers. The combination of alcohol with tobacco use further increases the chances of developing oral and pharyngeal cancer [69]. The 2015–2020 Dietary Guidelines recommend limiting alcohol intake to one drink per day for women and up to two drinks per day for men. One drink is equivalent to 5 oz of wine, 1.5 oz of distilled spirits, or 12 oz of beer [68].

Physical activity is another lifestyle factor recommended in tandem with dietary guidelines for disease prevention and improved quality of life. Maintaining a healthy weight and being physically active can help lower the risk of obesity, cardiovascular disease, and diabetes. The Physical Activity

Guidelines for Americans 2008, issued by US Department of Health and Human services (HHS) recommend at least 150 min of aerobic exercise per week for adults, in order to achieve long-term health benefits. Strength training for working all of the major muscle groups is also recommended at least twice a week. Older adults who are not able to do 150 min of aerobic exercise per week due to chronic conditions or other immobility, should aim to be as physically as possible, within the limits of their conditions [70].

Polypharmacy and Oral Health

A significant number of elderly individuals suffer from several chronic diseases and ailments, many requiring treatment with medication or multiple medications; however, side effects from multiple medication use, or polypharmacy, may increase the risk of oral diseases. These side effects include diminished salivary flow, difficulty in swallowing, and dry mouth, also known as xerostomia. The risk of developing side effects from medication increases with the more medications used [5, 71].

Dry mouth and decreased saliva are serious consequences of taking multiple medications, since saliva is responsible for demineralization and remineralization of teeth. Decreased salivary flow results in less exposure to calcium, phosphate, and fluoride. The absence of saliva's antimicrobial properties creates an environment for cariogenic microorganisms and *Candida*. Without saliva debriding teeth, there is an increased production of bacteria and acid along with a higher risk of returning or new carious lesions. Older individuals suffering from xerostomia should have regular dental visits, every 3 months. They should also be encouraged to chew sugar-less gum to promote salivary flow. Commercially available salivary substitutes such as gels, sprays, and mouth rinses are also available, but may not be as effective in caries prevention [71].

Polypharmacy can also affect nutritional status in the elderly by interfering with nutrient absorption. Some drugs and nutrients have the same metabolic pathway, which could affect their respective absorption and excretion. Nutritional status may decline due to reduced bioavailability of nutrients. Antihypertensive medications such as angiotensin-converting enzyme inhibitors increase serum potassium levels and sodium excretion. Acid blockers interfere with calcium, phosphorus, and iron absorption. Proton pump inhibitors (PPIs) decrease calcium, iron, magnesium, and B12 absorption [72]. Metformin can also lead to a B12 and vitamin D deficiency in individuals with type 2 diabetes [73]. Medications may affect nutritional status by reducing food intake due to decreased appetite or changes in taste perception. Certain medications such as diuretics may lead to a deficiency in zinc, an important mineral affecting taste and appetite [22]. A study in adults 65 years or older taking multiple drugs was associated with a lower intake of fiber, fat-soluble vitamins, water-soluble vitamins (thiamine, niacin, biotin) and a higher intake of glucose, sodium, and dietary cholesterol [72]. Older adults, in particular, should be educated on any potential drug-nutrient interactions and how to avoid nutrient deficiencies through a healthy eating pattern.

Summary

Dental diseases such as caries and periodontitis are global health issues which require early prevention and intervention. Left untreated, these diseases can lead to serious health consequences in both children, adults, and the elderly. The elderly are at particular risk for oral diseases due to side effects of medications and poor diet quality as a consequence of tooth loss [71]. Individuals with eating disorders are also vulnerable to oral diseases due to dental erosion secondary to self-induced vomiting and nutrient deficiencies from starvation [25]. Periodontitis can cause systemic inflammation which may increase risk of chronic diseases such as obesity, diabetes, and heart disease [31]. Along with good oral hygiene practice, avoiding cariogenic foods, increasing intake of anticariogenic foods, and eating a healthful and balanced diet, can help prevent development of dental diseases [4]. Nutrients found in fruits, vegetables, and low-fat dairy have been shown to have a protective effect on teeth. Diets rich in omega-3 fatty acids, are also associated with lower risk of dental disease, due to their anti-inflammatory properties [17]. Polyphenols, probiotics, and certain functional foods, may add further protective benefit to teeth [59–64]. Chewing xylitol-containing gum after meals [15] and using topical fluoride help fight acid-producing bacteria that lead to dental caries and periodontitis [47].

Following recommendations from the 2015–2020 Dietary Guidelines can help meet individual nutrient needs and establish a healthy eating pattern. Maintaining an overall healthful diet and lifestyle, with regular physical activity, is necessary in reducing risk of dental diseases and their associated diseases [68]. Other important lifestyle factors include avoiding excessive alcohol use and smoking, which are major contributors to poor oral health [69].

References

- US Department of Health and Human Services. Oral Health in America Oral Health in America: a report of the surgeon general. Executive summary. Rockville: US Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health; 2000.
- A National Call to Action to Promote Oral Health. Division of Oral Health, National Center for Chronic Disease Prevention and Health Promotion. 2013. http://www.cdc.gov/OralHealth/publications/factsheets/adult_oral_health/ call_to_action.htm.
- Listl S, Galloway J, Mossey PA, Marcenes W. Global economic impact of dental diseases. J Dent Res. 2015;94:1355– 61. doi:10.1177/0022034515602879.
- 4. Touger-Decker R, Mobley C, Academy of Nutrition and Dietetics. Position of the Academy of Nutrition and Dietetics: oral health and nutrition. J Acad Nutr Diet. 2013;113:693–701. doi:10.1016/j.jand.2013.03.001.
- 5. Dagli RJ, Sharma A. Polypharmacy: a global risk factor for elderly people. J Int Oral Health. 2014;6(6):i-ii.
- Singh A, Purohit B. Tooth brushing, oil pulling and tissue regeneration: a review of holistic approaches to oral health. J Ayurveda Integr Med. 2011;2:64–8. doi:10.4103/0975-9476.82525.
- Asokan S, Emmadi P, Chamundeswari R. Effect of oil pulling on plaque induced gingivitis: a randomized, controlled, triple-blind study. Indian J Dent Res. 2009;20:47–51.
- Viswanath D, Jayasimha VL, Prabhuji MLV, Vasudevan V. A critical appraisal of diet and nutrition on oral health in children – a review. Int J Health Sci Res. 2014;4:165–73.
- Evans EW, Hayes C, Palmer CA, Bermudez OI, Cohen SA, Must A. Dietary intake and severe early childhood caries in low-income, young children. J Acad Nutr Diet. 2013;113:1057–61. doi:10.1016/j.jand.2013.03.014.
- Moynihan PJ, Kelly SA. Effect on caries of restricting sugars intake: systematic review to inform WHO guidelines. J Dent Res. 2014;93(1):8–18. doi:10.1177/0022034513508954.
- Ramos-Gomez F, Crystal YO, Ng MW, Tinanoff N, Featherstone JD. Caries risk assessment, prevention, and management in pediatric dental care. Gen Dent. 2010;58:505–17.
- 12. Altun C, Maden EA, Maden EA, Uçar BD, Polat GG. The erosive effects of honey, molasses and orange juice on the primary teeth of children. Pediatr Dent J. 2015;25:50–3.
- 13. Atwa AD, AbuShahba RY, Mostafa M, Hashem MI. Effect of honey in preventing gingivitis and dental caries in patients undergoing orthodontic treatment. Saudi Dent J. 2014;26:108–14. doi:10.1016/j.sdentj.2014.03.001.
- Adegboye AR, Christensen LB, Holm-Pedersen P, Avlund K, Boucher BJ, Heitmann BL. Intakes of calcium, vitamin D, and dairy servings and dental plaque in older Danish adults. Nutr J. 2013;12:61.
- Nayak PA, Nayak UA, Khandelwal V. The effect of xylitol on dental caries and oral flora. Clin Cosmet Investig Dent. 2014;6:89–94. doi:10.2147/CCIDE.S55761.
- Ingle NA, Dubey HV, Kaur N, Gupta R. Effect of sugared and sugar free chewing gums on dental plaque: a clinical study. J Adv Oral Research. 2013;4:7.
- Zaki NA, Dowidar KM, Abdelaziz WE. Assessment of the Healthy Eating Index-2005 as a predictor of early childhood caries. Int J Paediatr Dent. 2015;25:436–43. doi:10.1111/ipd.12150.
- Bhatia G, Gupta S, Arora A, Saxena S, Sikka N. Nutrition in oral health and disease. J Adv Med Dent Sci. 2014;2:74–85.

- Lalla RV, Choquette LE, Feinn RS, Zawistowski H, Latortue MC, Kelly ET, et al. Multivitamin therapy for recurrent aphthous stomatitis: a randomized, double-masked, placebo-controlled trial. J Am Dent Assoc. 2012;143:370–6.
- George JP, Shobha R, Lazarus FJ. Folic acid: a positive influence on periodontal tissues during health and disease. Int J Health Allied Sci. 2013;2:145–52.
- Sheetal A, Hiremath VK, Patil AG, Sajjansetty S, Kumar SR. Malnutrition and its oral outcome a review. J Clin Diagn Res. 2013;7:178–80. doi:10.7860/JCDR/2012/5104.2702.
- Thomas DM, Mirowski GW. Nutrition and oral mucosal diseases. Clin Dermatol. 2010;28:426–31. doi:10.1016/j. clindermatol.2010.03.025.
- Graells J, Ojeda RM, Muniesa C, Gonzalez J, Saavedra J. Glossitis with linear lesions: an early sign of vitamin B12 deficiency. J Am Acad Dermatol. 2009;60(3):498–500.
- Coculescu EC, Ţovaru Ş, Coculescu BI. Epidemiological and etiological aspects of burning mouth syndrome. J Med Life. 2014;7:305–9.
- Kisely S, Baghaie H, Lalloo R, Johnson NW. Association between poor oral health and eating disorders: systematic review and meta-analysis. Br J Psychiatry. 2015;207:299–305. doi:10.1192/bjp.bp.114.156323.
- 26. Zhu Y, Hollis JH. Tooth loss and its association with dietary intake and diet quality in American adults. J Dent. 2014;42:1428–35. doi:10.1016/j.jdent.2014.08.012.
- 27. Emami E, de Souza RF, Kabawat M, Feine JS. The impact of edentulism on oral and general health. Int J Dent. 2013;2013:498305. doi:10.1155/2013/498305.
- 28. Genco RJ, Borgnakke WS. Risk factors for periodontal disease. Periodontol 2000. 2013;62:59–94. doi:10.1111/j.1600-0757.2012.00457.x.
- Llambés F, Arias-Herrera S, Caffesse R. Relationship between diabetes and periodontal infection. World J Diabetes. 2015;6:927–35. doi:10.4239/wjd.v6.i7.927.
- Singh-Dang T, Walker M, Ford D, Valentine RA. Nutrigenomics: the role of nutrients in gene expression. Periodontol 2000. 2014;64:154–60.
- Nagpal R, Yamashiro Y, Izumi Y. The two-way association of periodontal infection with systemic disorders: an overview. Mediators Inflamm. 2015;2015:793898. doi:10.1155/2015/793898.
- Casanova L, Hughes FJ, Preshaw PM. Diabetes and periodontal disease: a two-way relationship. Br Dent J. 2014;217:433–7. doi:10.1038/sj.bdj.2014.907.
- 33. Bullon P, Newman HN, Battino M. Obesity, diabetes mellitus, atherosclerosis and chronic periodontitis: a shared pathology via oxidative stress and mitochondrial dysfunction? Periodontol 2000. 2014;64:139–53. doi:10.1111/j.1600-0757.2012.00455.x.
- 34. Ryan ME, Raja V. Diet, obesity, diabetes, and periodontitis: a syndemic approach to management. Curr Oral Health Rep. 2016;3:14–27. doi:10.1007/s40496-016-0075-1.
- 35. Chaffee BW, Weston SJ. Association between chronic periodontal disease and obesity: a systematic review and meta-analysis. J Periodontol. 2010;81(12):1708–24. doi:10.1902/jop.2010.100321.
- 36. Lockhart PB, Bolger AF, Papapanou PN, Osinbowale O, Trevisan M, Levison ME, et al. Periodontal disease and atherosclerotic vascular disease: does the evidence support an independent association?: a scientific statement from the American Heart Association. Circulation. 2012;125:2520–44. doi:10.1161/CIR.0b013e31825719f3.
- 37. Rydén L, Buhlin K, Ekstrand E, de Faire U, Gustafsson A, Holmer J, et al. Periodontitis increases the risk of a first myocardial infarction: a report from the PAROKRANK Study. Circulation. 2016;133:576–83. doi:10.1161/ CIRCULATIONAHA.115.020324.
- Vannice G, Rasmussen H. Position of the academy of nutrition and dietetics: dietary fatty acids for healthy adults. J Acad Nutr Diet. 2014;114:136–53. doi:10.1016/j.jand.2013.11.001.
- Naqvi AZ, Buettner C, Phillips RS, Davis RB, Mukamal KJ. Omega 3 fatty acids and periodontitis in U.S. adults. J Am Diet Assoc. 2010;110:1669–75. doi:10.1016/j.jada.2010.08.009.
- 40. Deore GD, Gurav AN, Patil R, Shete AR, Naiktari RS, Inamdar SP. Omega 3 fatty acids as a host modulator in chronic periodontitis patients: a randomised, double-blind, palcebo-controlled, clinical trial. J Periodontal Implant Sci. 2014;44:25–32. doi:10.5051/jpis.2014.44.1.25.
- Kondo K, Ishikado A, Morino K, Nishio Y, Ugi S, Kajiwara S. A high-fiber, low-fat diet improves periodontal disease markers in high-risk subjects: a pilot study. Nutr Res. 2014;34(6):491–8. doi:10.1016/j.nutres.2014.06.001.
- Bradshaw DJ, Lynch RJ. Diet and the microbial aetiology of dental caries: new paradigms. Int Dent J. 2013;2:64– 72. doi:10.1111/idj.12082.
- 43. Schwartz N, Kaye EK, Nunn ME, Spiro 3rd A, Garcia RI. High-fiber foods reduce periodontal disease progression in men aged 65 and older: the Veterans Affairs normative aging study/Dental Longitudinal Study. J Am Geriatr Soc. 2012;60:676–83. doi:10.1111/j.1532-5415.2011.03866.x.
- 44. Trumbo P, Schlicker S, Yates AA, Poos M, Food and Nutrition Board of the Institute of Medicine, The National Academies. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. J Am Diet Assoc. 2002;102:1621–30.

10 Nutrition in Oral Health

- Dahl WJ, Stewart ML. Position of the Academy of Nutrition and Dietetics: health implications of dietary fiber. J Acad Nutr Diet. 2015;115:1861–70. doi:10.1016/j.jand.2015.09.003.
- Palmer CA, Gilbert JA. Position of the Academy of Nutrition and Dietetics: the impact of fluoride on health. J Acad Nutr Diet. 2012;112:1443–53. doi:10.1016/j.jand.2012.07.012.
- Carey CM. Focus on fluorides: update on the use of fluoride for the prevention of dental caries. J Evid Based Dent Pract. 2014. doi:10.1016/j.jebdp.2014.02.004.
- Levis S, Lagari V. The role of diet in osteoporosis prevention and management. Curr Osteoporos Rep. 2012;10:296– 302. doi:10.1007/s11914-012-0119-y.
- 49. Martelli FS, Martelli M, Rosati C, Fanti E. Vitamin D: relevance in dental practice. Clin Cases Miner Bone Metab. 2014;11:15–9.
- Alshouibi EN, Kaye EK, Cabral HJ, Leone CW, Garcia RI. Vitamin D and periodontal health in older men. J Dent Res. 2013;92:689–93. doi:10.1177/0022034513495239.
- Hiremath VP, Rao CB, Naiak V, Prasad KV. Anti-inflammatory effect of vitamin D on gingivitis: a dose response randomised controlled trial. Indian J Public Health. 2013;57:29–32. doi:10.4103/0019-557X.111365.
- 52. Antonenko O, Bryk G, Brito G, Pellegrini G, Zeni SN. Oral health in young women having a low calcium and vitamin D nutritional status. Clin Oral Investig. 2015;19:1199–206. doi:10.1007/s00784-014-1343-x.
- Van der Velden U, Kuzmanova D, Chapple IL. Micronutritional approaches to periodontal therapy. J Clin Periodontol. 2011;38 Suppl 11:142–58. doi:10.1111/j.1600-051X.2010.01663.x.
- Meisel P, Schwahn C, Luedemann J, John U, Kroemer HK, Kocher T. Magnesium deficiency is associated with periodontal disease. J Dent Res. 2005;84:937–41.
- Willershausen B, Ross A, Försch M, Willershausen I, Mohaupt P, Callaway A. The influence of micronutrients on oral and general health. Eur J Med Res. 2011;16:514–8.
- 56. Iwasaki M, Manz MC, Taylor GW, Yoshihara A, Miyazaki H. Relations of serum ascorbic acid and α-tocopherol to periodontal disease. J Dent Res. 2012;91:167–72. doi:10.1177/0022034511431702.
- 57. Institute of Medicine, Food and Nutrition Board. Dietary reference intakes for vitamin C, vitamin E, selenium, and carotenoids. Washington, DC: National Academy Press; 2000.
- 58. Gupta G. Probiotics and periodontal health. J Med Life. 2011;4(4):387-94.
- 59. Kalra M, Bhaskar DJ, Punia H, Singh V, Jinghala V. Probiotics and oral health. J Contemp Dent. 2015;5(2):104-6.
- 60. Lolayekar N, Shanbhag C. Polyphenols and oral health. RSBO. 2012;9(1):74-84.
- Shahzad M, Millhouse E, Culshaw S, Edwards CA, Ramage G, Combet E. Selected dietary (poly)phenols inhibit periodontal pathogen growth and biofilm formation. Food Funct. 2015;6:719–29. doi:10.1039/c4fo01087f.
- 62. Gaur S, Agnihotri R. Green tea: a novel functional food for the oral health of older adults. Geriatr Gerontol Int. 2014;14:238–50. doi:10.1111/ggi/12194.
- Kushiyama M, Shimazaki Y, Murakami M, Yamashita Y. Relationship between intake of green tea and periodontal disease. J Periodontol. 2009;80:372–7. doi:10.1902/jop.2009.080510.
- 64. Koyama Y, Kuriyama S, Aida J, Sone T, Nakaya N, Ohmori-Matsuda K, et al. Association between green tea consumption and tooth loss: cross-sectional results from the Ohsaki Cohort 2006 Study. Prev Med. 2010;50:173–9. doi:10.1016/j.ypmed.2010.01.010.
- Gazzani G, Daglia M, Papetti A. Food components with anticaries activity. Curr Opin Biotechnol. 2012;23:153–9. doi:10.1016/j.copbio.2011.09.003.
- Datta A, Datta G. Nutritional counseling in prevention of caries a team approach. Int J Dental Sci Res. 2014;2:31– 3. doi:10.12691/ijdsr-2-6B-9.
- 67. Kaye EK. Nutrition, dietary guidelines and optimal periodontal health. Periodontol 2000. 2012;58(1):93–111. doi:10.1111/j.1600-0757.2011.00418.x.
- U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015 2020 Dietary Guidelines for Americans. 8th ed. December 2015. http://health.gov/dietaryguidelines/2015/guidelines/.
- Berkey DB, Scannapieco FA. Medical considerations relating to the oral health of older adults. Spec Care Dentist. 2013;33:164–76. doi:10.1111/scd.12027.
- US Department of Health and Human Services. 2008 physical activity guidelines for Americans. 2008. http://www. health.gov/paguidelines/guidelines/summary.aspx.
- Singh ML, Papas A. Oral implications of polypharmacy in the elderly. Dent Clin North Am. 2014;58:783–96. doi:10.1016/j.cden.2014.07.004.
- Ortolani E, Landi F, Martone AM, Onder G, Bernabei R. Nutritional status and drug therapy in older adults. J Gerontol Geriat Res. 2013;2:123. doi:10.4172/2167-7182.1000123.
- 73. Kos E, Liszek MJ, Emanuele MA, Durazo-Arvizu R, Camacho P. Effect of metformin therapy on vitamin D and vitamin B₁₂ levels in patients with type 2 diabetes mellitus. Endocr Pract. 2012;18:179–84. doi:10.4158/EP11009.

Part III Nutrition in Childhood

Chapter 11 Childhood Obesity

Craig A. Johnston, Daphne C. Hernandez, and Abdullah Shuaib

Key Points

- Childhood obesity is measured in many ways including body mass index (BMI) percentile and BMI standardized score. In addition to these measures of height and weight, direct measures of adiposity are encouraged in order to obtain a full-body composition profile.
- Obesity in childhood can lead to a host of comorbid conditions, but perhaps the most important risk is that these children are likely to become obese adults.
- The greatest likelihood of success in treating childhood obesity is through family-based lifestyle interventions.
- In addition to primary (population-wide strategies) and secondary (lifestyle interventions), tertiary prevention strategies (drug therapy or weight-loss surgery) have been developed to treat obesity in youth.

Keywords Adiposity • BMI • Family-based behavioral approach • Type 2 diabetes • Weight-loss surgery

Abbreviations

AGB	Adjustable gastric banding		
BIA	Bioelectrical impedance analysis		
BMI	Body mass index		
BMI z-score	Standardized BMI		
DEXA	Dual-energy x-ray absorptiometry		
LSG	Laparoscopic sleeve gastrectomy		
RYBG	Roux en Y gastric bypass		
TLP	Traffic Light Program		

C.A. Johnston, PhD (🖂) • D.C. Hernandez, PhD, MSEd • A. Shuaib, BS

Department of Health & Human Performance, University of Houston,

³⁸⁷⁵ Holman Street, Garrison Gymnasium, Room 104, Houston, TX 77204-6015, USA e-mail: cajohn25@Central.UH.EDU

Introduction

The US childhood obesity prevalence rate has increased from 5.0% in 1976–1980 to 8.4% in 2011– 2012 for preschool-aged children (ages 2-5 years) [1, 2]. More dramatic increases have been observed among children in middle childhood (ages 6-11) from 6.5 to 17.7% and adolescents (ages 12-19 years) from 5.0 to 20.5 % within the same time period [1, 2]. Despite the sharp increases in obesity prevalence rates in the past 40 years, a review of the literature indicates the prevalence rates among children and adolescents are stabilizing and consequently plateauing [3], with a couple of exceptions. Among preschool-aged children, obesity prevalence rates have decreased since 2003–2004 when it was at 13.9% [1]. On the other hand, obesity prevalence rates have not leveled off among (a) race/ ethnic minority children and children from lower socio-economic status groups and (b) children classified with severe obesity: class 2 obesity category (>120% of 95th percentile) and class 3 obesity category (>140% of 95th percentile) [4, 5]. For instance, in 2011–2012, 16.7% of Hispanic preschoolaged children and 11.3% of black preschool-aged children were obese compared to 3.5% of white preschool-aged children [1]. Further, the prevalence rates of class 2 obesity has increased from 3.8 % in 1999–2000 to 5.9% in 2011–2012, and class 3 obesity prevalence rates has risen from 0.9 to 2.1%within the same time frame [4]. These trends are particularly concerning because the groups that have historically been most impacted by obesity appear to be consistently increasing in terms of obesity prevalence.

Aside from the population-level trends, there are development trends that cause concern. Early childhood obesity tracks into adolescence and adulthood. Overweight 5-year-olds have been shown to be four times more likely to become obese by age 14 compared to normal weight peers [6]. Further, as much as 82% of obese children become obese adults [7]. Thus, the developmental trends, along with the population trends and corresponding disparities, suggest that childhood obesity is an epidemic that all health professionals will face. This chapter provides an explanation of how measurement guidelines and various methodologies can be used to identify children at-risk for obesity as well as those that are obese. The chapter also briefly describes comorbidities associated with childhood obesity and strategies for treating childhood obesity.

Measurement Guidelines and Methodologies

Childhood overweight and obesity are weight status terms that are measured using body mass index (BMI). Most of the childhood obesity research has used BMI as an outcome measure. BMI is calculated by dividing the child's weight in kilograms by the square of child's height in meters [8]. The reference value is then plotted on Centers for Disease Control (CDC) growth curve charts that are gender- and age-specific to determine the BMI percentile [9]. The BMI percentiles are divided into four categories which are used to describe a child's weight status. Children with a BMI percentile less than 5th percentile are categorized as underweight, while children with a BMI percentile between the 5th percentile and less than the 85th percentile and less than the 95th percentile. Obese children have a BMI percentile at or above the 95th percentile [8]. BMI provides a measure of excess body weight (e.g., overweight status); yet, it is not a measure of direct body fat. However, it is considered to be correlated with adiposity.

An alternate measure to BMI that is also commonly used is standardized BMI, also referred to as a BMI z-score. BMI z-score is the transformation of the child's BMI into the number of standard deviations (SD) above and below the population mean BMI based on gender and age [10]. Similar to BMI percentiles that are divided into categories to describe a child's weight status, the same has been

done with BMI z-score. Weight status criteria using BMI z-scores are normal weight 0 SD (50th percentile), overweight \geq 1.04 SD (85th percentile), obesity \geq 1.64 SD (95th percentile), and severe obesity \geq 2.33 SD [11]. For weight loss to be clinically meaningful (i.e., reduce health concerns), a reduction in BMI z-score of 0.5 SD over 6 months or at least 0.6 SD over 6–12 months [12] needs to occur. However, reduction in BMI z-score of 0.25 SD over 12 months is considered clinically relevant in improving cardiovascular risk factors (i.e., insulin sensitivity, total cholesterol/high-density lipoprotein ratio, and blood pressure) in adolescents [13].

Although BMI z-scores provide the opportunity to compare changes in weight status, BMI z-score compared to BMI is more sensitive to changes in weight status [12]. For instance, among obese children, any weight change that does occur is observed as less change because there is less variability at the upper end of the weight distribution. This is a problem when comparing the effects of obesity interventions because the heavier children will show less change in their BMI z-scores, despite having similar changes in BMI as the less heavy children [14].

Aside from weight status, there are various methods to measure adiposity, which include skinfold thickness, waist circumference, bioelectrical impedance analysis (BIA), and dual-energy x-ray absorptiometry (DEXA). Skinfold thickness, waist circumference, and BIA are fairly inexpensive, portable, and quick methods of collecting adiposity information from children. Skinfold thickness is measured using standardized calipers to capture a double layer of skin and subcutaneous fat lifted as a fold at various areas of the body, including but not limited to triceps, biceps, abdominal, thigh, and calf. Measurement reliability is low for skinfold thickness, and reference percentiles have not been published [15]. A tape measure around the abdominal region determines the circumference of the waist, which is suggestive of the amount of body fat in that particular area of the body. Previous research has used a waist circumference measurement \geq 90th percentile for age and sex to be considered to be elevated percent body fat [16, 17], and this has been linked to increased risk of diabetes and other cardiovascular diseases [18]. Waist circumference establishes fat distribution in the abdomen region, but it does not provide a measurement for total body fatness [15]. BIA involves the child stepping on a scale that uses an electric current to differentiate between fat mass and lean tissue mass. The electrical current is unnoticeable to the child [15]. BIA measurements are considered to be reliable [15, 19], and the Tanita BF-689 (Tanita Corporation, Arlington Heights, IL) [20], which is designed for children, is highly specific for classifying children as over fat and obese [19]. However, it has also demonstrated to have a low sensitivity and specificity for healthy percent body fat classification [19].

Unlike the other methods, DEXA provides the most information and is considered the gold standard. DEXA scans the entire body in about 7 min using an extremely low amount of radiation to calculate fat mass, lean tissue mass, and bone mineral content for the whole body and for specific regions of the body [21]. DEXA is the most accepted method to calculate percent body fat; however, DEXA fails to be useful in community outreach settings as the machinery is expensive and not portable [19]. Despite their limitations, all of these methods are important for research, especially when wanting to create a comprehensive body composition profile.

Obesity-Related Comorbidities

The additional weight that overweight and obese children and adolescents carry places them at increased risk for physical and physiological consequences. Comorbidities include increased risk for worse overall health [22], functional limitations [22], orthopedic complications (e.g., spinal complications, slipped capital femoral epiphysis, and acute fractures) [23], earlier puberty and menarche in girls [24], obstructive sleep apnea [25], prediabetes and type 2 diabetes [26, 27], metabolic syndrome [26], and elevated blood pressure, blood lipids, insulin, and insulin resistance [28]. A majority of these health problems have traditionally only been observed in adults. Additionally, individuals that

experience either childhood or adolescent obesity are at risk for experiencing obesity in adulthood [7, 29] and experiencing further health complications, including colorectal cancer and gout among adult men, arthritis among adult women, coronary heart disease and atherosclerosis among both adult men and women [30], and consequently increased risk for mortality [26].

Psychological and social consequences may be even more common than physical and physiological comorbidities. At the core of emotional well-being is depression and self-esteem. Overweight/ obese adolescents are at more risk of experiencing depression symptomatology and poor self-esteem compared to normal weight adolescents their age [22]. While depression may result in behaviors that promote obesity [31], negative social outcomes that result from obesity may increase symptoms of depression. For example, obese children are more likely to be bullied due to their deviation from ideal appearances [32], and being bullied is associated with depression and anxiety [33, 34]. In fact, it has been suggested that a reciprocal relationship exists between obesity and depression [35]. Binge eating, impulsivity, eating-disordered cognitions and behaviors, and body dissatisfaction have been observed among overweight, obese, and severely obese children [36–39]. Overall, the quality life of overweight/obese children has been described to be similar to that of children who have cancer [40].

The academics of overweight and obese children also suffer which has socio-economic consequences. Pediatric overweight and obesity have been linked to lower cognitive functioning [41] and standardized test scores [42], although some of the findings can be explained by socio-economic status, including parental education [43]. The decreased performance in the academic realm may result in harmful social consequences that persist into adulthood. For example, heavy women are less likely to attain post-secondary schooling compared to their non-heavy peers, which can negatively influence their occupational standing [44, 45]. Further, obese women are at risk for lower wages and consequently lower family income [45–47]. Last, heavy women are more likely to delay marriage and have lower odds of marrying compared to their thinner peers [45, 46].

Due to its diverse and widespread comorbidities, child and adolescent overweight and obesity have become a public health concern. If left unabated, more preventable deaths will be associated with obesity than with cigarette smoking, and deaths related to obesity could replace those prevented by improved treatment for cancer, heart disease, and diabetes [48]. Fortunately, receiving treatment for overweight/obesity may attenuate some of the negative physical, physiological, and psychological consequences.

History of Treatment Strategies

The general consensus in medicine throughout the nineteenth century was that carrying an extra 20–30 lb or more of fat was a health advantage that enhanced resistance when a person became ill. Unless obesity was extreme to the extent that it made it difficult to carry out normal activities, it was not identified as a problem. In this era, a slender figure was a sign of poor health in association with diseases like tuberculosis [49, 50]. It was not until the twentieth century that the position on overweight changed. American insurance enterprises began selling an increased amount of life insurance policies. The companies started to study the mortality rates of different groups of people and conditions in order to remain profitable. This resulted in the collection of comprehensive, longstanding statistics on health and mortality for thousands of people [49, 51].

A pioneer in this data collection was Oscar H. Rogers, a physician with the New York Life Insurance Company. Rogers conducted a systematic study of insured lives that included parameters such as height, weight, occupation, personal history, and family history. His findings in 1901 showed that a group of 1,500 policyholders who were 30% or more overweight had a 35% higher mortality rate [50, 52].

This evidence was critical for American insurance companies and drove the industry to further collect and analyze information on body weight. Insurance companies desired to insure policyholders of "normal weight," but lacked statistical support for their judgment. Louis I. Dublin, a statistician of the Metropolitan Life Insurance Company, calculated "average weights" from a comprehensive retrospective survey of New York Life Insurance policyholders. In 1908, Dublin compiled the *Dublin Standard Table of Heights and Weights* that became the primary reference of average weight. Dublin became one of the leading voices that spread the concerns of overweight throughout the medical community. Health professionals now had weight averages that were based on evidence [50, 53].

Although the statistics of insurance enterprises could not validate that excess weight directly caused increased mortality rates, the correlation was significant enough for insurers to adjust rates accordingly. The correlation, though debated, was also compelling enough for many health professionals to include it as part of their medical advice. Dublin's charts were published in popular newspapers and magazines like *The New York Times* and *McClure's Magazine*, which created an initial public awareness. Other life insurance studies confirmed that as excess bodyweight increased, life expectancy decreased. From 1900 to 1910, an increasing number of Americans were interested in knowing their exact weight at the doctor's office or with home scales. Weighing spring scales with weight charts were sold in increasing quantities throughout the following decades. In the years following 1910, it was common for middle-class families to pay close attention to standardized weights for children [51, 52].

By the 1930s, the medical profession no longer debated the health implications of increased fat mostly due to the insurance industry's continuing studies. In 1930, Louis Dublin published a paper that detailed the specific problems that occurred more frequently among individuals who were obese such as heart disease, diabetes, kidney disease, atherosclerosis, and brain hemorrhages. This paper was a historic turning point that resulted in increased research to investigate the link between excess weight and the large array of health problems. This paper, while partially flawed and not representative of the whole population, was viewed as compelling evidence of the risks associated with being overweight. Within 10 years, excess weight went from a topic that concerned only a few in the medical profession to being seen as a significant medical issue [49].

In the following years, it became evident that obesity was rarely caused by medical abnormalities within the body as it was previously thought. It also became evident that people generally underestimated what they consumed. After the 1930s, it was no longer unknown why people gained excess weight. Obesity appeared to be an accessible problem to solve. Education was the main intervention as doctors taught the calorie equation to their patients. While some individuals who were overweight successfully and permanently lost weight by reducing intake and increasing activity, most failed to do so. From the 1930s onward, a variety of methods were used to assist in weight loss. Drugs and hormones were prescribed that decreased appetite or increased metabolic rate such as dinitrophenyl and amphetamines. Diuretics and formula meal replacements were also commonly taken. It became apparent that the brain played a considerable role in weight gain, and some treatments took a psychological approach such as group therapies to target food addiction, loneliness, anxiety, and depression. As a whole, no method had a moderate or high success rate in reducing weight in the long run [49, 51].

In 1952, the director of the National Institutes of Health declared that obesity was a primary national health concern [49, 51]. At the same time, studies were being conducted on the behavioral management of obesity [54, 55]. These studies on "self-control of overeating" were the first to describe behavioral principles (e.g., stimulus control, shaping) that are still used in obesity management today to improve diet and physical activity. In 1973, a conference was held at the National Institutes of Health to bring experts studying obesity together and discuss "the need for heightened research on psychological, physiological, and biochemical determinants of this important health derangement" [56].

By 1980, there was significant and dramatic increase in the rates of adult obesity [57]. The prevalence of adult obesity had increased from 14.5 to 22.5 % over the course of about 15 years [57]. It was at this time that some of the first behavioral treatments for obesity in children were being published [58, 59]. Epstein's pioneering work in this field provided healthcare professionals with a model for how to promote healthy lifestyles in children and adolescents [59].

By early 1990, the attention shifted to the increase in childhood obesity. An article in *Science* discussed obesity alongside pressing conditions facing US adolescents: suicide, pregnancy, and decline in college aptitude test scores [60]. By the early turn of the century, considerable attention was being given to the comorbidities associated with childhood obesity. For instance, studies were beginning to demonstrate that obese children were experiencing hyperlipidemia and hypertension [61], type 2 diabetes [62], and atherosclerotic lesions [63]. Consequently, programs were developed in numerous settings to both prevent and treat obesity. In the traditional healthcare sense, these settings included but were not limited to hospitals, primary care, and specialty clinics. The need to deliver these services into the community also became apparent as many children that were most impacted were also less likely to seek medical care or be insured [64, 65]. Because children spend a majority of their day at school, interventions taking place at schools also became a priority.

Childhood Obesity Treatment Strategies

The numerous comorbidities associated with pediatric obesity make it clear that beginning the prevention and treatment of this condition in childhood is necessary. Overall, the most successful interventions use a multidimensional, family-based approach by focusing on modifications to diet, physical activity, and other lifestyle behaviors [66]. In a family-based obesity treatment program, the behaviors of both the parent and child are targeted with the ultimately goal to facilitate new healthy behaviors and a home environment that supports healthy eating and activity [66]. Since the home/family environment is a strong predictor of adolescent obesity [67], parents are viewed as the agents of change and their involvement and corresponding weight-loss success is critical to their child being successful in losing weight [68, 69]. Last, strategies used to modify behaviors include self-monitoring, stimulus control/cue control, and contingency management (e.g., positive reinforcement, social praise, rewards, and contracting) [66].

Several national committees have been formed to help provide childhood obesity treatment guidelines and recommendations. The US Preventive Services Task Force recommends pediatricians to screen for obesity in all children between 6 and 18 years of age and refer overweight and obese children to comprehensive behavioral treatment programs that focus on diet and physical activity, and provide behavioral counseling. Treatment should be moderate- (26–75 h) to high-intensity (>75 h). A treatment duration of 6 months and family involvement are also preferred [70]. Short-term benefits have been observed among obese children and adolescents that engage in moderate- to high-intensity behavioral interventions [71].

The Academy of Nutrition and Dietetics provides guidelines and recommendations for interventions targeting childhood obesity [72]. In addition, an expert committee comprised of representatives from 15 professional organizations, appointed experienced scientists and clinicians to develop recommendations regarding the prevention and treatment of childhood obesity [73]. The Academy discusses obesity prevention in terms of primary, secondary, and tertiary, while the expert committee uses a four-stage approach [72, 73]. The two approaches are discussed below, along with a brief example of primary, secondary, and tertiary prevention programs.

Primary Prevention

Primary prevention uses diet and physical activity messages or programs intended to prevent overweight or obesity or provide support to maintain weight. Primary prevention is targeted to all youth (i.e., population-wide intervention), without a specific focus on a particular weight status. Populationwide prevention, as is early intervention, is considered to be ideal in establishing healthy eating and physical activity habits before poor habits become ingrained. There is not a corresponding stage approach to primary prevention [72].

Primary prevention has taken place in childcare centers, schools, and community-based settings. Several primary prevention interventions have targeted preschool-aged children as young children are more responsive to behavior change compared to older children [74]. A number of these interventions have focused on improving nutrition and/or increasing physical activity. A recent trend in nutrition education programs is the use of garden-based nutrition education programs in childcare centers, schools, and community settings as a mechanism to learn about fruits and vegetables [75]. These primary prevention programs have shown to increase preference and consumption of fruits and vegetables, along with physical activity and science knowledge [76–79]. However, there is a dearth of studies that have focused on the use of garden-based nutrition education programs as a mechanism to prevent obesity or maintain weight status. Among Latino fourth- and fifth-grade students, Davis and colleagues [80] found that a 12-week, after-school gardening, nutrition, and cooking program not only increased fiber intake and decreased diastolic blood pressure, but it also reduced BMI and promoted less weight gain compared to a control group. More studies are needed to understand the association between nutrition education and obesity prevention and weight loss.

Secondary Prevention

Secondary prevention involves more structured intervention and strategies involving diet and physical activity intended to assist overweight or obese children with no weight-related comorbidities obtain a healthy weight. Secondary prevention corresponds to Stages 1–3 of the staged approach [72]. Stage 1 is referred to the Prevention Plus stage. In Stage 1, children who are overweight are encouraged to focus on these healthy habits and gradually improve BMI status. Families and care providers are encouraged to work together to identify health behaviors that most impact the child's energy balance. Once agreement has been reached on specific health behaviors, small and measurable steps should be taken to make changes. If the child has not made improvements in 3–6 months, Stage 2, Structured Weight Management, is encouraged. In this stage, children and families receive more structure and support for their targeted behavior change. Some of the main differences for this stage are providing the family with a planned diet, incorporating structured meals, and planned physical activity. Stages 3, Comprehensive Multidisciplinary Intervention, is suggested after Stages 1 and 2 have been attempted and do not appear intensive enough to curb the velocity of the child's weight gain. Some of the primary components of Stage 3 include a structured behavior modification program, systematic evaluation of body measurements and behavior change, and weekly appointments [73].

An example of a secondary prevention program is Epstein's Traffic Light Program (TLP) [81]. The TLP is a family-based behavioral treatment designed for overweight or obese children that focuses on modifying diet and physical activity. The purpose of the diet component of the TLP is used to decrease high energy density foods and increase low energy density foods [82]. Foods are categorized into the three colors of a traffic light based on their calorie and nutrient content.

- *Red* foods are high in calories and low in nutrient content. Anything above 5 g of fat and/or 15 g of sugar is considered a "red" food. Examples of these foods include potato chips, soda, and candy. It is recommended to eat these food sparingly and to eat seven or fewer *red* foods per week.
- *Yellow* foods are moderate in calories but include nutrients necessary for a balanced diet. Examples of these foods include skim milk, lean meats, and grains. It is recommended to eat these foods in moderation.
- *Green* foods are low in calories and high in nutrient content. These foods are primarily vegetables. It is recommended to eat these foods often.

Trials that use this program with children initially instructed parents and children to consume a restricted amount of calories (typically ranging from 900 to 1,200 kcal). This was later changed to 1,000 to 1,500 kcal/day [83].

In the physical activity component of the TLP, children and their parents are provided written manuals focused on advantages of increased physical activity and disadvantages of sedentary behaviors [84]. Physical activity is defined in terms of caloric expenditure and translated to activity points using number of calories burned in a 10-min period of the activity according to weight. Children and parents are taught to calculate the caloric expenditure of each activity in which they participate. Sedentary behaviors are defined as behaviors that compete for being active (e.g., watching television, playing videogames, talking on the phone). Sedentary behaviors, such as listening to music or completing homework, are not specifically targeted for reduction. However, participants are rewarded for a gradual increase in physical activity and a gradual reduction in sedentary behaviors. Once the participant reaches the goal of 15 or fewer hours of sedentary behavior a week or 150 activity points, they are expected to maintain this change.

Multiple investigations have found the TLP to be superior to control conditions in randomized studies. For example, Epstein and colleagues demonstrated that receiving the TLP is superior to no treatment [85] and to an attention placebo control condition [86, 87]. Perhaps the most impressive finding is that children who received the TLP demonstrated maintenance of effects in 10-year follow-up studies [88, 89].

Tertiary Prevention

Because behavioral interventions are not successful for all obese children, and the long-term success rate at 2 years post intervention is low [90], tertiary prevention was developed. Tertiary prevention is the most intensive and comprehensive treatment for overweight and obese children with comorbidities and the severely obese youth. The strategy is to resolve weight-related comorbidities or at least reduce their severity. Tertiary prevention corresponds to Stage 4, Tertiary Care Intervention [72]. Stage 4 is the final stage offered to some severely obese youth after Stages 1–3 do not appear to influence weight change. In other words, when behavioral and lifestyle changes are deemed unsuccessful among the severely obese, medical treatment is prescribed in the form of drug therapy or weight-loss surgery [73]. Compared to behavioral interventions, less is known about using medical treatment to alleviate childhood obesity. Medical treatment is seldom performed and is not the preferred choice of treatment [90].

Orlistat (Xenical; Roche Products), which is a gastrointestinal lipase inhibitor, is the only form of prescription weight-loss medication that is approved by the US Food and Drug Administration (FDA) for obesity treatment with adolescents 12 years of age and older [91]. Orlistat blocks the absorption of fat in the intestine by inhibiting lipase activity. Because Orlistat may reduce the absorption of fat-soluble vitamins, the FDA recommends children on Orlistat treatment take a daily multivitamin supplement that contains vitamins A, D, E, and K [91]. In addition, every meal should have no more than 30 % calories from fat. A non-prescription, reduced-strength version of Orlistat (Alli;

GlaxoSmithKline) was FDA-approved in 2007 for adult use, but not adolescents. Several studies have demonstrated the safety and efficacy of Orlistat on weight loss in obese adults [92–94]. Among children and adolescents, Orlistat is effective; yet, side effects (e.g., fatty/oily stool, fecal urgency) are common [95–99].

Sibutramine was briefly FDA-approved in 2009 for adolescents 16 years and older and considered to be effective in decreasing BMI [100–102]. Among adults, this drug treatment was associated with weight loss and maintenance [103, 104]. However, in 2010, this medication was withdrawn because a large clinical trial reported increased risk of non-fatal myocardial infarction and non-fatal stroke after long-term use (i.e., average 3 years) among older adults with pre-existing history of cardiovascular disease, type 2 diabetes, or both [103]. Similar, but less severe side effects (e.g., increased heart rate and blood pressure), had been observed among adolescents [100, 102].

Although weight-loss surgery for adolescents was first reported in 1980, it was not until 2004 that surgical and pediatric experts recommended this strategy as an appropriate treatment for severely obese adolescents [105]. There are several criteria that adolescents must meet to be eligible for the surgery [106]. Adolescents must have failed at least 6 months of a structured weight-loss program. A BMI in the 99th percentile with either serious comorbidities (e.g., type 2 diabetes, moderate, or severe obstructive sleep apnea) or other comorbidities (e.g., hypertension, insulin resistance, impaired quality of life or activities) is required. A BMI in the 99th percentile is equivalent to having an adult BMI greater than 35 (with serious comorbidities) or greater than 40 (with other comorbidities), which are the BMI criteria for adults to be eligible for weight-loss surgery [107]. Adolescents must also have reached 95% of skeletal maturity based on radiography and reached a puberty maturity of a Tanner stage IV or V. Psychologically, adolescents must exhibit that they understand the dietary and physical activity changes that will be required of them for optimal post-operative success and the potential risks and benefits of surgery. Last, a supportive network needs to be in place during pre- and post-operative procedures [106].

The most frequent weight-loss surgery procedures conducted on adolescents include the roux en Y gastric bypass (RYBG), adjustable gastric banding (AGB), and the laparoscopic sleeve gastrectomy (LSG), with RYBG being the most common of the three procedures. RYBG procedure reorganizes the gastrointestinal tract to bypass the stomach and duodenum and creates an egg-size pouch. This new pouch is limited by the amount of food that it can hold. The concern with this surgery is that nutrients will not be absorbed properly due to the reconfiguration of the gastrointestinal tract. Specifically, deficiencies in thiamine, iron, calcium, and vitamins B12 and D have been observed [108, 109]. RYBG has been successful in helping adolescents lose weight and decrease cardiovascular risk factors, including elevated glucose (i.e., fasting glucose and HbA1c) and lipids (i.e., LDL-cholesterol and triglycerides) [110–112].

AGB and LSG are appealing to adolescents because they avoid intestinal bypass. AGB is a laparoscopic surgical procedure where a saline-filled band is placed around the stomach, just below the junction of the esophagus. This restrictive procedure creates a small gastric pouch. A saline solution is injected through a port surgically implanted on the abdominal wall under the skin when there is a need to adjust the band [72, 113]. AGB has been associated with helping adolescents lose significant amounts of weight and improve quality of life and depressive symptoms [114–116]. However, AGB is not FDA-approved for adolescents, and the information made available is through clinical trials designed to investigate the safety and effectiveness of the procedure [72]. Complications that occur among some recipients of AGB are band slippage (or band dislocation) which can lead to band erosion or pouch dilatation and require surgery to correct [117]. Over the years, this procedure has become the least popular of the three [118].

LSG is a procedure where a significant portion of the stomach is surgical removed. Specifically, the stomach is stapled into a smaller pouch that is the shape of a sleeve, banana, or half-moon tube. This results in individuals feeling fuller sooner and increases satiety [119]. It is hypothesized that greater satiety occurs because less ghrelin (i.e., hormone that stimulates hunger) is secreted by the lining of the fundus as a consequence of the fundus being resected in the gastrectomy [120]. Short-term data

suggest that it is safe and effective in helping adolescents lose weight and improve quality of life [121–123]. Trends indicate that this procedure is increasing in popularity [118]. Popularity is not only related to the positive outcomes of the surgery, but also because LSG is (a) surgically less technically challenging compared to RYBG and (b) does not involve implantation of an artificial device like AGB [124]. However, the stapling does place individuals at risk for post-surgery complications, and the procedure is irreversible [119]. Data that follow these adolescents into adulthood are needed to understand the sustainability of their weight loss and the reduction of their comorbidities. In addition, clinical trials that compare all three surgical methods are needed.

Future Directions

While current treatment strategies to reduce childhood obesity have shown some progress, childhood obesity remains a serious concern due to its high prevalence rate [1]. In order to further decrease the prevalence of obesity in children and adolescence in the future, more advanced strategies need to be implemented and newer observational and experimental research must be conducted.

Critical Periods for Weight Gain

An important aspect in attempting to reduce the prevalence of childhood obesity in the future is to identify periods of critical weight gain. Weight gain may not necessarily follow a smooth, continuous pattern. If there are instances where weight gain is disproportionally accelerated, it is essential to attempt to understand the fundamental causes and address them. A suspected period of significant weight gain among children has been during summer vacation and winter holiday break. During the holiday season, children from third to fifth grade gain an average 1.3 lb in weight and grew an average 0.3 inches in height; yet, this resulted in no significant difference in BMI [125]. In contrast, summer weight gain is an order of magnitude greater than over the general school year [126]. Fitness progress from school-based interventions is also lost during the summer vacation [126, 127]. An area for future development could include analyzing dietary assessments, along with physical activity, in children throughout an entire year in attempt to map changes in food consumption and physical activity. Data should also be collected on the environmental changes that may occur. Since informal childcare is linked with increased risk of obesity, summer weight gain could be accounted for by the different lifestyles under day-care centers, babysitters, and relative care [128].

Use of Social Media Marketing to Target Adolescents for Nutrition and Physical Activity Information

Social marketing is a method used to alter or maintain people's behavior and ideas for the benefit of society. It proved to be successful at targeting public health issues such as vaccination, breast-feeding, and tobacco usage. Social marketing has made notable progress in increasing exercise and improving diet among children [129]. Most methods of social marketing concerning childhood obesity that targets children include integrated school programs, advertisements, and books.

According to PEW Research Center 2015 data, 73 % of adolescents have smartphones, while 71 % use more than one social network site [130]. This indicates a potential setting to use as social marketing to decrease the prevalence of childhood and adolescent obesity [131]. While many programs use

social media for social marketing, there is little research on the effectiveness on targeting adolescents [132]. This may be an area for future development. In conjunction with artists, athletes, and entertainment figures that are popular among adolescents, information may be used to alter the behavior of overweight or obese children and adolescents. More research is necessary to present data on obese adolescents to understand whether variances in ethnicity, gender, socioeconomic status, and geographic status affect the level of participation with social media and technology-driven interventions. Further research is also needed to recognize what elements can be merged into social media that will increase the effectiveness of interventions to decrease the prevalence of childhood obesity. Even though social media marketing is not likely to be successful at reducing rates of obesity independently, it may be significant in combination with other interventions.

Conclusion

Although childhood obesity rates for the most part are stabilizing or plateauing, overall the prevalence rates remain high, and obesity-related comorbidities are influencing the physical, physiological, and psychological health of children. Several measurement guidelines and methodologies have been developed to assist with identifying children at-risk for obesity as well as those that are obese. The methodologies used to measure weight and adiposity each have their own sets of limitations; yet, when used together, they can build a comprehensive body composition profile. Guidelines by national organizations and committees, along with structured interventions and corresponding strategies, have been created to assist with lowering the weight status of overweight and obese children and related comorbidities. A multidimensional family-based behavioral approach that focuses on diet, physical activity, and behavior modifications has gained the most traction. For severely obese children that do not respond to behavioral interventions, medical therapy is available. Although medical therapy is considered safe and effective, it is not the preferred choice of treatment. Overall, significant gains have been made in understanding and treating pediatric obesity. Despite these gains, many children and families remain significantly impacted by this disease. Innovation from both researchers and healthcare providers is needed to further stem the tide of this epidemic.

References

- Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA. 2014;311(8):806–14.
- Ogden CL, Carroll M. Prevalence of obesity among children and adolescents: United States, Trends 1963–1965 through 2007–2008 2010 [cited 2016 January 2]. Available from: http://www.cdc.gov/nchs/data/hestat/obesity_ child_07_08/obesity_child_07_08.htm.
- 3. Rokholm B, Baker JL, Sørensen TIA. The levelling off of the obesity epidemic since the year 1999–a review of evidence and perspectives. Obes Rev. 2010;11(12):835–46.
- Skinner AC, Skelton JA. Prevalence and trends in obesity and severe obesity among children in the United States, 1999–2012. JAMA Pediatr. 2014;168(6):561–6.
- Wang Y, Beydoun MA. The obesity epidemic in the United States–gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. Epidemiol Rev. 2007;59:6–28.
- Cunningham SA, Kramer MR, Narayan KV. Incidence of childhood obesity in the United States. N Engl J Med. 2014;370(5):403–11.
- Juonala M, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, et al. Childhood adiposity, adult adiposity, and cardiovascular risk factors. N Engl J Med. 2011;365(20):1876–85.
- Centers for Disease Control and Prevention. Assessing your weight about BMI for children and teens. Atlanta: Center for Disease Control and Prevention. 2011 [cited 2016 January 2]. Available from: http://www.cdc.gov/ healthyweight/assessing/bmi/childrens_bmi/about_childrens_bmi.html.

- Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, Mei Z, et al. 2000 CDC growth charts for the United States: methods and development. National Center for Health Statistics. Vital Health Stat. 2002;246:1– 190, Contract No.: 246.
- 10. National Center for Health Statistics. Z-score data files. 2009 [cited 2016 January 6]. Available from: http://www. cdc.gov/growthcharts/zscore.htm.
- Bundred P, Kitchiner D, Buchan I. Prevalence of overweight and obese children between 1989 and 1998: population based series of cross sectional studies. BMJ. 2001;322(7282):326.
- 12. Hunt LP, Ford A, Sabin MA, Crowne EC, Shield JP. Clinical measures of adiposity and percentage fat loss: which measure most accurately reflects fat loss and what should we aim for? Arch Dis Child. 2007;92(5):399–403.
- Ford AL, Hunt LP, Cooper A, Shield JPH. What reduction in BMI SDS is required in obese adolescents to improve body composition and cardiometabolic health? Arch Dis Child. 2010;95(4):256–61.
- Paluch RA, Epstein LH, Roemmich JN. Comparison of methods to evaluate changes in relative body mass index in pediatric weight control. Am J Hum Biol. 2007;19(4):487–94.
- 15. Himes JH. Challenges of accurately measuring and using BMI and other indicators of obesity in children. Pediatrics. 2009;124(Suppl 1):S3–22.
- Fernández JR, Redden DT, Pietrobelli A, Allison DB. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. J Pediatr. 2004;145(4):439–44.
- 17. Hernandez DC. Latino mothers' cumulative food insecurity exposure and child body composition. Am J Health Behav. 2016;40(1):92–9.
- 18. Bassali R, Waller JL, Gower B, Allison J, Davis CL. Utility of waist circumference percentile for risk evaluation in obese children. Int J Pediatr Obes. 2010;5(1):97–101.
- Kabiri LS, Hernandez DC, Mitchell K. Reliability, validity, and diagnostic value of a pediatric bioelectrical impedance analysis scale. Child Obes. 2015;11(5):650–5.
- Tanita Corporation. BF-689 children's body fat monitor. 2014 [cited 2015 December 13]. Available from: http:// www.tanita.com/en/bf-689/.
- Helba M, Binkovitz LA. Pediatric body composition analysis with dual-energy X-ray absorptiometry. Pediatr Radiol. 2009;39(7):647–56.
- Swallen KC, Reither EN, Haas SA, Meier AM. Overweight, obesity, and health-related quality of life among adolescents: the National Longitudinal Study of Adolescent Health. Pediatrics. 2005;115(2):340–7.
- 23. Wills M. Orthopedic complications of childhood obesity. Pediatr Phys Ther. 2004;16(4):230-5.
- Kaplowitz PB, Slora EJ, Wasserman RC, Pedlow SE, Herman-Giddens ME. Earlier onset of puberty in girls: relation to increased body mass index and race. Pediatrics. 2001;108(2):347–53.
- Blechner M, Williamson AA. Consequences of obstructive sleep apnea in children. Curr Probl Pediatr Adolesc Health Care. 2015;46(1):19–26.
- 26. Biro FM, Wien M. Childhood obesity and adult morbidities. Am J Clin Nutr. 2010;91(5):1499S-505.
- May AL, Kuklina EV, Yoon PW. Prevalence of cardiovascular disease risk factors among US adolescents, 1999–2008. Pediatrics. 2012;129(6):1035–41.
- Friedemann C, Heneghan C, Mahtani K, Thompson M, Perera R, Ward AM. Cardiovascular disease risk in healthy children and its association with body mass index: systematic review and meta-analysis. BMJ. 2012;345:e4759.
- 29. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med. 1997;337(13):869–73.
- 30. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents: a follow-up of the Harvard Growth Study of 1922 to 1935. N Engl J Med. 1992;327(19):1350–5.
- Goodman E, Whitaker RC. A prospective study of the role of depression in the development and persistence of adolescent obesity. Pediatrics. 2002;110(3):497–504.
- 32. Lumeng JC, Forrest P, Appugliese DP, Kaciroti N, Corwyn RF, Bradley RH. Weight status as a predictor of being bullied in third through sixth grades. Pediatrics. 2010;125(6):e1301–7.
- Klomek AB, Marrocco F, Kleinman M, Schonfeld IS, Gould MS. Bullying, depression, and suicidality in adolescents. J Am Acad Child Adolesc Psychiatry. 2007;46(1):40–9.
- 34. Gladstone GL, Parker GB, Malhi GS. Do bullied children become anxious and depressed adults?: a cross-sectional investigation of the correlates of bullying and anxious depression. J Nerv Ment Dis. 2006;194(3):201–8.
- 35. Luppino FS, de Wit LM, Bouvy PF, Stijnen T, Cuijpers P, Penninx BW, et al. Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. Arch Gen Psychiatry. 2010;67(3):220–9.
- Tanofsky-Kraff M, Yanovski SZ, Wilfley DE, Marmarosh C, Morgan CM, Yanovski JA. Eating-disordered behaviors, body fat, and psychopathology in overweight and normal-weight children. J Consult Clin Psychol. 2004;72(1):53–61.
- 37. Wildes JE, Marcus MD, Kalarchian MA, Levine MD, Houck PR, Cheng Y. Self-reported binge eating in severe pediatric obesity: impact on weight change in a randomized controlled trial of family-based treatment. Int J Obes. 2010;34(7):1143–8.

- Mond J, Van den Berg P, Boutelle K, Hannan P, Neumark-Sztainer D. Obesity, body dissatisfaction, and emotional well-being in early and late adolescence: findings from the project EAT study. J Adolesc Health. 2011;48(4):373–8.
- Nederkoorn C, Braet C, Van Eijs Y, Tanghe A, Jansen A. Why obese children cannot resist food: the role of impulsivity. Eat Behav. 2006;7(4):315–22.
- Schwimmer JB, Burwinkle TM, Varni JW. Health-related quality of life of severely obese children and adolescents. JAMA. 2003;289(14):1813–9.
- Li Y, Dai Q, Jackson JC, Zhang J. Overweight is associated with decreased cognitive functioning among schoolage children and adolescents. Obesity. 2008;16(8):1809–15.
- Roberts CK, Freed B, McCarthy WJ. Low aerobic fitness and obesity are associated with lower standardized test scores in children. J Pediatr. 2010;156(5):711–8.
- Datar A, Sturm R, Magnabosco JL. Childhood overweight and academic performance: national study of kindergartners and first-graders. Obes Res. 2004;12(1):58–68.
- Glass CM, Haas SA, Reither EN. The skinny on success: body mass, gender and occupational standing across the life course. Soc Forces. 2010;88(4):1777–806.
- Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. N Engl J Med. 1993;329(14):1008–12.
- 46. Conley D, Glauber R. Gender, body mass, and socioeconomic status: new evidence from the PSID. Adv Health Econ Health Serv Res. 2007;17:253–75.
- 47. Averett S, Korenman S. The economic reality of the beauty myth. J Hum Resour. 1996;31(2):304–30.
- 48. U.S. Department of Health and Human Services. The Surgeon General's call to action to prevent and decrease overweight and obesity. Rockville: U.S. Department of Health and Human Services, Public Health Service, Office the Surgeon General; 2001.
- 49. Pool R. Fat: fighting the obesity epidemic: fighting the obesity epidemic. New York: Oxford University Press, USA; 2000.
- 50. Brumberg JJ. Fasting girls: the history of anorexia nervosa. New York: Vintage Books; 2000.
- 51. Schwartz H. Never satisfied: a cultural history of diets, fantasies, and fat. New York: Free Press; 1986.
- 52. Bouk D. How our days became numbered: risk and the rise of the statistical individual. Chicago: University of Chicago Press; 2015.
- 53. Foxcroft L. Calories and Corsets: a history of dieting over two thousand years. London: Profile Books; 2012.
- 54. Ferster CB, Nurnberger JI, Levitt EB. The control of eating. J Math. 1962;1:87-109.
- 55. Stuart RB. Behavioral control of overeating. Behav Res Ther. 1967;5(4):357-65.
- Leavitt MD. Preface. In: Obesity in perspective: Fogarty International Center Series on Preventive Medicine [Internet]. Washington, DC: US Government Printing Office; 1976.
- Flegal KM, Carroll MD, Kuczmarski RJ, Johnson CL. Overweight and obesity in the United States: prevalence and trends, 1960–1994. Int J Obes Relat Metab Disord. 1998;22(1):39–47.
- Aragona J, Cassady J, Drabman RS. Treating overweight children through parental training and contingency contracting. J Appl Behav Anal. 1975;8(3):269–78.
- Epstein LH, Wing RR, Steranchak L, Dickson B, Michelson J. Comparison of family-based behavior modification and nutrition education for childhood obesity. J Pediatr Psychol. 1980;5(1):26–36.
- 60. Fuchs VR, Reklis DM. America's children: economic perspectives and policy options. Science. 1992;255(5040):41-6.
- Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. Pediatrics. 1999;103(6):1175–82.
- Pinhas-Hamiel O, Dolan LM, Daniels SR, Standiford D, Khoury PR, Zeitler P. Increased incidence of non-insulindependent diabetes mellitus among adolescents. J Pediatr. 1996;128(5):608–15.
- 63. Berenson G, Srinivasan S, Bao W, Newman W, Tracy R, Wattigney W. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. New Engl J Med. 1998;338(23):1650.
- 64. Haas JS, Lee LB, Kaplan CP, Sonneborn D, Phillips KA, Liang S-Y. The association of race, socioeconomic status, and health insurance status with the prevalence of overweight among children and adolescents. Am J Public Health. 2003;93(12):2105–10.
- Lau M, Lin H, Flores G. Racial/ethnic disparities in health and health care among US adolescents. Health Serv Res. 2012;47(5):2031–59.
- 66. Wilfley DE, Vannucci A, White EK. Family-based behavioral interventions. In: Freemark M, editor. Contemporary endocrinology: pediatric obesity: etiology, pathogenesis, and treatment. New York: Humana Press; 2010. p. 281–301.
- Larson N, Wall M, Story M, Neumark-Sztainer D. Home/family, peer, school, and neighborhood correlates of obesity in adolescents. Obesity. 2013;21(9):1858–69.

- Boutelle KN, Cafri G, Crow SJ. Parent predictors of child weight change in family based behavioral obesity treatment. Obesity. 2012;20(7):1539–43.
- Watson PM, Dugdill L, Pickering K, Bostock S, Hargreaves J, Staniford L, et al. A whole family approach to childhood obesity management (GOALS): relationship between adult and child BMI change. Ann Hum Biol. 2011;38(4):445–52.
- US Preventive Services Task Force. Screening for obesity in children and adolescents: US Preventive Services Task Force recommendation statement. Pediatrics. 2010;125(2):361–7.
- Whitlock EP, O'Connor EA, Williams SB, Beil TL, Lutz KW. Effectiveness of weight management interventions in children: a targeted systematic review for the USPSTF. Pediatrics. 2010;125:e396–418.
- Hoelscher DM, Kirk S, Ritchie L, Cunningham-Sabo L, Academy Positions Committee. Position of the Academy of Nutrition and Dietetics: interventions for the prevention and treatment of pediatric overweight and obesity. J Acad Nutr Diet. 2013;113(10):1375–94.
- Barlow SE, Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007;120 Suppl 4:S164–92.
- 74. Wofford LG. Systematic review of childhood obesity prevention. J Pediatr Nurs. 2008;23(1):5-19.
- Robinson-O'Brien R, Story M, Heim S. Impact of garden-based youth nutrition intervention programs: a review. J Am Diet Assoc. 2009;109(2):273–80.
- 76. Blair D. The child in the garden: an evaluative review of the benefits of school gardening. J Environ Educ. 2009;40(2):15–38.
- McAleese JD, Rankin LL. Garden-based nutrition education affects fruit and vegetable consumption in sixthgrade adolescents. J Am Diet Assoc. 2007;107(4):662–5.
- Parmer SM, Salisbury-Glennon J, Shannon D, Struempler B. School gardens: an experiential learning approach for a nutrition education program to increase fruit and vegetable knowledge, preference, and consumption among second-grade students. J Nutr Educ Behav. 2009;41(3):212–7.
- Graham H, Zidenberg-Cherr S. California teachers perceive school gardens as an effective nutritional tool to promote healthful eating habits. J Am Diet Assoc. 2005;105(11):1797–800.
- Davis JN, Ventura EE, Cook LT, Gyllenhammer LE, Gatto NM. LA Sprouts: a gardening, nutrition, and cooking intervention for Latino youth improves diet and reduces obesity. J Am Diet Assoc. 2011;111(8):1224–30.
- Epstein LH. Family-based behavioural intervention for obese children. Int J Obes Relat Metab Disord. 1996;20(1):S14–21.
- Epstein LH, Wing RR, Koeske R, Valoski A. A comparison of lifestyle exercise, aerobic exercise, and calisthenics on weight loss in obese children. Behav Ther. 1985;16(4):345–56.
- Epstein LH, Paluch RA, Raynor HA. Sex differences in obese children and siblings in family-based obesity treatment. Obes Res. 2001;9(12):746–53.
- Epstein LH, Valoski AM, Vara LS, McCurley J, Wisniewski L, Kalarchian MA, et al. Effects of decreasing sedentary behavior and increasing activity on weight change in obese children. Health Psychol. 1995;14(2):109–15.
- Epstein LH, Wing RR, Koeske R, Valoski A. Effects of diet plus exercise on weight change in parents and children. J Consult Clin Psychol. 1984;52(3):429–37.
- Epstein LH, Wing RR, Steranchak L, Dickson B, Michelson J. Comparison of family-based behavior modification and nutrition education for childhood obesity. J Pediatr Psychol. 1980;5(1):25–36.
- Epstein LH, Wing RR, Woodall K, Penner BC, Kress MJ, Koeske R. Effects of family-based behavioral treatment on obese 5-to-8-year-old children. Behav Ther. 1985;16(2):205–12.
- Epstein LH, Valoski A, Wing RR, McCurley J. Ten-year follow-up of behavioral, family-based treatment for obese children. JAMA. 1990;264(19):2519–23.
- Epstein LH, Valoski A, Wing RR, McCurley J. Ten-year outcomes of behavioral family-based treatment for childhood obesity. Health Psychol. 1994;13(5):373.
- 90. Reinehr T. Lifestyle intervention in childhood obesity: changes and challenges. Nat Rev Endocrinol. 2013;9(10):607–14.
- Food and Drug Adminstration. Division of Metabolic and Endocrine Drug Products (HFD-510) Clinical reviews for NDA 20-766/S018. 2003 [cited 2016 January 6]. Available from: http://www.fda.gov/downloads/Drugs/ DevelopmentApprovalProcess/DevelopmentResources/UCM163348.pdf.
- Finer N, James W, Kopelman P, Lean M, Williams G. One-year treatment of obesity: a randomized, double-blind, placebo-controlled, multicentre study of orlistat, a gastrointestinal lipase inhibitor. Int J Obes. 2000;24(3):306–13.
- Rossner S, Sjostrom L, Noack R, Meinders E, Noseda G. Weight loss, weight maintenance, and improved cardiovascular risk factors after 2 years treatment with orlistat for obesity. Obes Res. 2000;8(1):49–61.
- 94. Aldekhail NM, Logue J, McLoone P, Morrison D. Effect of orlistat on glycaemic control in overweight and obese patients with type 2 diabetes mellitus: a systematic review and meta-analysis of randomized controlled trials. Obes Rev. 2015;16(12):1071–80.
- Chanoine J-P, Hampl S, Jensen C, Boldrin M, Hauptman J. Effect of orlistat on weight and body composition in obese adolescents: a randomized controlled trial. JAMA. 2005;293(23):2873–83.

- McDuffie JR, Calis KA, Uwaifo GI, Sebring NG, Fallon EM, Hubbard VS, et al. Three-month tolerability of Orlistat in adolescents with obesity-related comorbid conditions. Obes Res. 2002;10(7):642–50.
- Norgren S, Danielsson P, Jurold R, Lötborn M, Marcus C. Orlistat treatment in obese prepubertal children: a pilot study. Acta Paediatr. 2003;92(6):666–70.
- Ozkan B, Bereket A, Turan S, Keskin S. Addition of orlistat to conventional treatment in adolescents with severe obesity. Eur J Pediatr. 2004;163(12):738–41.
- 99. Zhi J, Moore R, Kanitra L. The effect of short-term (21-day) orlistat treatment on the physiologic balance of six selected macrominerals and microminerals in obese adolescents. J Am Coll Nutr. 2003;22(5):357–62.
- Berkowitz RI, Wadden TA, Tershakovec AM, Cronquist JL. Behavior therapy and sibutramine for the treatment of adolescent obesity: a randomized controlled trial. JAMA. 2003;289(14):1805–12.
- 101. Godoy-Matos A, Carraro L, Vieira A, Oliveira J, Guedes EP, Mattos L, et al. Treatment of obese adolescents with sibutramine: a randomized, double-blind, controlled study. J Clin Endocrinol Metab. 2005;90(3):1460–5.
- 102. García-Morales LM, Berber A, Macias-Lara CC, Lucio-Ortiz C, Del-Rio-Navarro BE, Dorantes-Alvárez LM. Use of sibutramine in obese mexican adolescents: a 6-month, randomized, double-blind, placebo-controlled, parallelgroup trial. Clin Ther. 2006;28(5):770–82.
- 103. James WPT, Caterson ID, Coutinho W, Finer N, Van Gaal LF, Maggioni AP, et al. Effect of sibutramine on cardiovascular outcomes in overweight and obese subjects. N Engl J Med. 2010;363(10):905–17.
- 104. Fujioka K, Seaton T, Rowe E, Jelinek C, Raskin P, Lebovitz H, et al. Weight loss with sibutramine improves glycaemic control and other metabolic parameters in obese patients with type 2 diabetes mellitus. Diabetes Obes Metab. 2000;2(3):175–87.
- Inge TH, Krebs NF, Garcia VF, Skelton JA, Guice KS, Strauss RS, et al. Bariatric surgery for severely overweight adolescents: concerns and recommendations. Pediatrics. 2004;114(1):217–23.
- Pratt JS, Lenders CM, Dionne EA, Hoppin AG, Hsu GL, Inge TH, et al. Best practice updates for pediatric/adolescent weight loss surgery. Obesity. 2009;17(5):901–10.
- 107. Michalsky M, Reichard K, Inge T, Pratt J, Lenders C. ASMBS pediatric committee best practice guidelines. Surg Obes Relat Dis. 2012;8(1):1–7.
- 108. Alvarez-Leite JI. Nutrient deficiencies secondary to bariatric surgery. Curr Opin Clin Nutr Metab Care. 2004;7(5):569–75.
- Towbin A, Inge TH, Garcia VF, Roehrig HR, Clements RH, Harmon CM, et al. Beriberi after gastric bypass surgery in adolescence. J Pediatr. 2004;145(2):263–7.
- 110. Olbers T, Gronowitz E, Werling M, Mårlid S, Flodmark C-E, Peltonen M, et al. Two-year outcome of laparoscopic Roux-en-Y gastric bypass in adolescents with severe obesity: results from a Swedish Nationwide Study (AMOS). Int J Obes. 2012;36(11):1388–95.
- 111. Oberbach A, Neuhaus J, Inge T, Kirsch K, Schlichting N, Blüher S, et al. Bariatric surgery in severely obese adolescents improves major comorbidities including hyperuricemia. Metabolism. 2014;63(2):242–9.
- 112. Sinha M, Stanley TL, Webb J, Scirica C, Corey K, Pratt J, et al. Metabolic effects of Roux-en-Y gastric bypass in obese adolescents and young adults. J Pediatr Gastroenterol Nutr. 2013;56(5):528.
- 113. Crocker MK, Yanovski JA. Pediatric obesity: etiology and treatment. Endocrinol Metab Clin North Am. 2009;38(3):525-48.
- 114. O'Brien PE, Sawyer SM, Laurie C, Brown WA, Skinner S, Veit F, et al. Laparoscopic adjustable gastric banding in severely obese adolescents: a randomized trial. JAMA. 2010;303(6):519–26.
- Fielding GA, Duncombe JE. Laparoscopic adjustable gastric banding in severely obese adolescents. Surg Obes Relat Dis. 2005;1(4):399–405.
- Sysko R, Devlin MJ, Hildebrandt TB, Brewer SK, Zitsman JL, Walsh BT. Psychological outcomes and predictors of initial weight loss outcomes among severely obese adolescents receiving laparoscopic adjustable gastric banding. J Clin Psychiatry. 2012;73(10):1351–7.
- 117. Keidar A, Szold A, Carmon E, Blanc A, Abu-Abeid S. Band slippage after laparoscopic adjustable gastric banding: etiology and treatment. Surg Endosc. 2005;19(2):262–7.
- 118. Pallati P, Buettner S, Simorov A, Meyer A, Shaligram A, Oleynikov D. Trends in adolescent bariatric surgery evaluated by UHC database collection. Surg Endosc. 2012;26(11):3077–81.
- Frezza EE. Laparoscopic vertical sleeve gastrectomy for morbid obesity. The future procedure of choice? Surg Today. 2007;37(4):275–81.
- Langer FB, Reza Hoda MA, Bohdjalian A, Felberbauer FX, Zacherl J, Wenzl E, et al. Sleeve gastrectomy and gastric banding: effects on plasma ghrelin levels. Obes Surg. 2005;15(7):1024–9.
- 121. Till H, Blüher S, Hirsch W, Kiess W. Efficacy of laparoscopic sleeve gastrectomy (LSG) as a stand-alone technique for children with morbid obesity. Obes Surg. 2008;18(8):1047–9.
- 122. Boza C, Viscido G, Salinas J, Crovari F, Funke R, Perez G. Laparoscopic sleeve gastrectomy in obese adolescents: results in 51 patients. Surg Obes Relat Dis. 2012;8(2):133–7.
- Nadler EP, Barefoot LC, Qureshi FG. Early results after laparoscopic sleeve gastrectomy in adolescents with morbid obesity. Surgery. 2012;152(2):212–7.

- 124. Shi X, Karmali S, Sharma AM, Birch DW. A review of laparoscopic sleeve gastrectomy for morbid obesity. Obes Surg. 2010;20(8):1171–7.
- Branscum PW, Kaye G, Succop P, Sharma M. An evaluation of holiday weight gain among elementary-aged children. J Clin Med Res. 2010;2(4):167–71.
- 126. Moreno JP, Johnston CA, Woehler D. Changes in weight over the school year and summer vacation: results of a 5-year longitudinal study. J Sch Health. 2013;83(7):473–7.
- 127. Carrel AL, Clark RR, Peterson S, Eickhoff J, Allen DB. School-based fitness changes are lost during the summer vacation. Arch Pediatr Adolesc Med. 2007;161(6):561–4.
- 128. Pearce A, Li L, Abbas J, Ferguson B, Graham H, Law C. Is childcare associated with the risk of overweight and obesity in the early years? Findings from the UK Millennium Cohort Study. Int J Obes. 2010;34(7):1160–8.
- 129. Gordon R, McDermott L, Stead M, Angus K. The effectiveness of social marketing interventions for health improvement: what's the evidence? Public Health. 2006;120(12):1133–9.
- Lenhart A. Teens, social media & technology overview 2015. Washington, DC: Pew Research Center; 2015. [cited 2016 April 18]. Available from: http://www.pewinternet.org/2015/04/09/teens-social-media-technology-2015/.
- 131. Gold J, Pedrana AE, Stoove MA, Chang S, Howard S, Asselin J, et al. Developing health promotion interventions on social networking sites: recommendations from The FaceSpace Project. J Med Internet Res. 2012;14(1):e30.
- 132. Gracia-Marco L, Moreno LA, Vicente-Rodríguez G. Impact of social marketing in the prevention of childhood obesity. Adv Nutr. 2012;3(4):611S-5.

Chapter 12 Nutritional Interventions to Lower Cholesterol and Risk for Heart Disease in Children

Stephen R. Daniels, Jessica Hildebrandt, Laura K. Brennan, and Sarah C. Couch

Key Points

- The risk for atherosclerotic cardiovascular disease begins in childhood and progresses through adolescence and young adulthood to produce adverse outcomes later in life.
- The traditional risk factors that have been a focus in adults are also important for children and adolescents—they include obesity, diabetes, elevated blood pressure, dyslipidemia and cigarette smoking.
- Identification and management of cardiovascular risk factors in children and adolescents require a team of healthcare professionals, including physicians, nurses, dietitians and exercise physiologists.
- Children and adolescents do not usually live in an environment that makes the health choice for diet, physical activity or sleep the easy choice.
- Behavior change interventions must focus on changing the home environment to encourage healthier choices.
- Identifying risk factor development in children and adolescents is important so that appropriate treatment and intervention can be implemented.

Keywords Pediatric nutrition • Obesity or childhood obesity • Cholesterol • Cardiovascular disease • Dyslipidemia • Familial hypercholesterolemia

S.R. Daniels, MD, PhD ()

Department of Pediatrics, University of Colorado School of Medicine, Children's Hospital Colorado, 13123 East 16th Avenue, B065, Aurora, CO 80045, USA e-mail: Stephen.Daniels@childrenscolorado.org

J. Hildebrandt, MS, RD Lifestyle Medicine Program, Children's Hospital Colorado, 13123 East 16th Avenue, B065, Aurora, CO 80045, USA e-mail: Jessica.Hildebrandt@childrenscolorado.org L.K. Brennan, BS, MS Candidate • S.C. Couch, PhD, RD Department of Nutritional Sciences, University of Cincinnati Medical Center, 3202 Eden Avenue, Cincinnati, OH 45267-0394, USA e-mail: COUCHSC@UCMAIL.UC.EDU

Introduction

Cardiovascular disease is the number one cause of death in the USA despite improvement in mortality over several decades [1]. Studies such as the Bogalusa and Muscatine studies in the USA and the Cardiovascular Disease in Young Finns and Childhood Determinants of Adult Health (CDAH) internationally have demonstrated that the risk for atherosclerotic cardiovascular disease begins in childhood and progresses through adolescence and young adulthood to produce adverse outcomes later in life [2–5]. These studies also confirm that the traditional risk factors that have been a focus in adults are also important for children and adolescents. These risk factors include obesity, diabetes, elevated blood pressure, dyslipidemia and cigarette smoking.

Recent research has shown that the prevalence of obesity in young people has leveled off at approximately 17% [6]. However, some race/sex groups are disproportionately impacted, such as African American females [6]. While the stall in the increase in prevalence is good news, it should be noted that a prevalence of obesity at 17% is far above what is optimal, especially given that it was approximately 5% three decades ago [6]. With the increased prevalence and severity of obesity, there has also been an increase in the prevalence of type 2 diabetes mellitus in adolescents [7]. This is a major concern because it is well known that diabetes is a very potent cardiovascular disease risk factor in adults [8].

In 2011–2012, approximately one in five children and adolescents age 8–17 years had an abnormal lipid value for total cholesterol, high-density lipoprotein (HDL)-cholesterol or non-HDL-cholesterol based on data from the National Health and Nutrition Examination Survey (NHANES) [9]. In addition, slightly more than one in ten children and adolescents had borderline high or high blood pressure [9]. The prevalence of dyslipidemia had decreased somewhat between 1999–2000 and 2011–2012, but the prevalence of borderline high and high blood pressure remained stable during that time [9].

The concept of ideal cardiovascular health has been a focus of the American Heart Association (AHA) [10]. Ideal cardiovascular health means normal body mass index (BMI), no diabetes, normal cholesterol, normal blood pressure and no cigarette smoking. When ideal cardiovascular health is present in adults age 50, their subsequent risk of cardiovascular disease is quite low and longevity is increased on average by as much as 10 years compared to individuals with ASCVD risk factors present [11]. One question is the extent to which ideal cardiovascular health is genetically determined. Studies have shown that less than 20% is due to genetics, meaning that 80% or more is determined by lifestyle factors such as diet and physical activity [12].

Most children are born with ideal cardiovascular health. Unfortunately, this beneficial condition is lost by many in childhood and adolescence and young adulthood. At age 50, it is estimated that fewer than 5% of individuals in the USA have ideal cardiovascular health [10]. This emphasizes the importance of establishing healthy diet and physical activity patterns early in childhood and maintaining them throughout life. It is important to emphasize the family unit as one that can facilitate maintenance of optimum diet and physical activity for both adults and children.

There has been some controversy regarding the optimum approach to diet to reduce risk of cardiovascular disease. However, decades of research support the focus on dietary patterns, which emphasize higher consumption of fruits and vegetables, whole grains, low-fat dairy and fish, while limiting consumption of foods high in sugar or saturated fat. This means elimination of foods and beverages with added sugar and salt and focusing on protein from poultry while also choosing lower saturated fat cuts of red meat [13]. In this chapter, we will discuss approaches to achieve optimum nutrition in children and adolescents and present recommendations both for prevention of risk factor development and lifestyle treatment once risk factors emerge.

Pediatric Obesity

Obesity, or an excess percentage of body weight as fat, is a chronic disease resulting from the imbalance of energy intake from food and the energy expended through physical activity. The prevalence of obesity in US children and adolescents has grown into a major health problem [14]. Today, about one in three American children and adolescents are overweight or obese (~31.8%) [15]. This makes prevention and treatment of childhood overweight and obesity an important public health priority. The development of overweight and obesity in children is influenced by a complex interplay of various genetic, behavioral and environmental factors. Non-Hispanic black and Hispanic children have higher prevalence rates than non-Hispanic white and non-Hispanic Asian [16]. Low-income children and adolescents are more likely to be obese than their higher-income counterparts, but the relationship is not consistent across race and ethnicity groups [16]. Children who are obese are at increased risk of obesity as an adult [17].

Assessment of Pediatric Obesity

Weight status in children is defined using BMI. BMI is an anthropometric measurement of body weight in kilograms (kg) divided by the square of the height in meters. Percentile distributions relative to gender and age in the 2000 Centers for Disease Control (CDC) growth charts are used as reference points [18]. BMI has been accepted as a useful tool to assess body fat [19]. Increasing BMI levels correlate with excess body fat [20]. Children with a BMI above the 85th percentile are considered overweight and above the 95th percentile are considered obese (Table 12.1).

The risk of obesity can also be assessed by examining growth trends and change in weight velocity. Weight gains trending upward and crossing over two percentiles may be at risk of obesity. Children younger than 2 years who have a growth trend that crosses two percentile lines on the length for weight chart have the highest prevalence of obesity 5–10 years later [21]. Waist-to-hip ratios and skinfolds are not recommended for the assessment of adiposity in children and adolescents [22].

Health Impact of Obesity in Children

Pediatric obesity is associated with an increased risk of a broad range of health problems including hypertension [23], impaired glucose tolerance [24], sleep apnea [25], elevated serum lipid levels [23], elevated hepatic enzyme levels [26], type 2 diabetes [27], respiratory problems including sleep apnea

 Table 12.1
 National Heart, Lung and Blood Institute (NHLBI) expert panel on the identification, evaluation and treatment of overweight and obesity

Classification	BMI-for-age percentile
Underweight	<5th
Healthy weight	5–84th
Overweight	85–94th
Obesity	95th
Barlow [22]	

and asthma [28, 29] and joint and musculoskeletal problems [28, 30]. Psychological stress such as depression, behavioral problems and issues in school are associated with childhood obesity [31–33].

Behavioral Factors

Lifestyle behaviors have a great impact on health and weight. Some key behaviors have been identified as risk factors in the development of childhood overweight and obesity. Behavior changes may be considered as targets for intervention when working with overweight/obese children and their families.

Diet

Specific dietary factors that can increase the risk of excessive energy intake relative to energy expenditure include regular intakes of sugar-sweetened beverages (SSB), greater intakes of fast food, increased intakes of solid fats and added sugars and eating larger portions of food [34]. Increased intake of fruits and vegetables may be associated with a decreased risk of obesity in children [34]. Additionally, some observational research indicates that low dairy and calcium-rich food intake may increase risk [34]. Breakfast skipping has been indicated as a dietary behavior that is associated with obesity [34]. Consequently, eating breakfast daily is associated with weight loss and maintenance in addition to improving nutrient intake [35]. Family meals, especially those with positive family- and parent-level interpersonal dynamics (i.e., warmth, group enjoyment and parental positive reinforcement) and with positive parent-level food-related dynamics (i.e., food warmth, food communication and parental food positive reinforcement), are associated with a reduced risk of childhood overweight and obesity [36].

Physical Activity

Lack of physical activity and sedentary lifestyle play key roles in the development of pediatric obesity. Regular physical activity and participation in sports may decrease risk of development of obesity in children [34].

Screen Time

Television viewing, playing of video games and recreational use of computers and cell phones are sedentary behaviors. Screen time exceeding 2 h per day increases childhood obesity risk [34]. Additionally, foods high in total calories, sugars, salt and fat and low in nutrients are highly advertised and marketed through media targeted to children and adolescents [37]. This can influence food intake.

Sleep

Shorter sleep duration, poor sleep quality or poor sleep hygiene can increase risk of obesity [34, 38].

Environmental Factors

Environmental factors such as those in the child's home, school and community can influence their food intake and physical activity. Parental dietary and physical activity habits can impact a child's weight and health outcomes. Nearly half of US middle and high schools allow advertising of less healthy foods which can hinder their ability to make healthy food choices [39]. Some children and their families have less access to stores and supermarkets that sell healthy, affordable food such as fruits and vegetables. This can be especially true in rural, minority and lower-income neighborhoods [40]. Supermarket access is associated with a reduced risk for obesity [40]. For families who live in areas with an overabundance of less healthy food, such as convenience stores and fast food restaurants, making healthy food choices can be difficult. Many communities lack safe and appealing places to be physically active. Some are built in ways that make it difficult or unsafe to be physically active. For some families, getting to parks and recreation centers may be difficult, and public transportation may not be available. Policy changes may be required to help minimize environmental influences on the development of overweight and obesity in children.

Targets for Intervention of Childhood Obesity

The National Heart, Lung and Blood Institute (NHLBI) Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity Recommendations provides guidelines for practitioners working with pediatric patients and their families in all areas of obesity care [22]. Interventions are categorized into different stages according to severity of obesity. The appropriate stage of treatment is based on age, BMI-for-age percentile and the presence of other health risks (Table 12.2).

The Academy of Nutrition and Dietetics Evidence Analysis Library (EAL) Evidence-Based Pediatric Weight Management Nutrition Practice Guideline, 2007, provides complementary guidelines.

Stage 1: Prevention Plus

A family approach focusing on healthful eating and activity behaviors aimed at improving BMI status. The involvement of the child's parent or caregiver is recommended especially for children ages 6 to 12 years [34].

Counseling			Health	
stage	Age	BMI%	risk	Goal
Stage 1	<18	<94th	No	Weight maintenance until BMI-for-age is <85th percentile or a slower rate of weight gain
Stages 1–2	2–18	85–94th	Yes	Weight maintenance until BMI-for-age is <85th percentile or a slower rate of weight gain
Stages 1–2	6–18	95–98th	Yes	Weight maintenance until BMI-for-age is <85th percentile or a gradual weight loss of 1 lb/month
Stages 1–3	2–5	95–98th	Yes	Weight maintenance until BMI-for-age is <85th percentile or a gradual weight loss of 1 lb/month
Stages 1-3	2-5	≥99th	Yes	Gradual weight loss ≤ 1 lb/month
Stages 1-3	6-11	>99th	Yes	Weight loss of 2 lb/month
Stages 1-4	12-18	>95th	Yes	Weight loss ≤2 lb/week until BMI-for-age is <85th percentile
Barlow [22]				

 Table 12.2
 National Heart, Lung and Blood Institute (NHLBI) expert panel on the identification, evaluation, and treatment of overweight and obesity

Barlow [22]

- 1. limiting consumption of SSB and juice
- 2. encouraging diets with adequate fruit and vegetable consumption
- 3. limiting television and other screen time
- 4. eating breakfast daily
- 5. limiting eating out at restaurants especially fast food restaurants
- 6. encouraging family meals in which parents and children eat together
- 7. limiting portion sizes
- 8. consumption of diets rich in calcium
- 9. consumption of diets high in fiber
- eating a diet with balanced macronutrients (energy from fat, carbohydrates and protein in proportions for age as recommended by Dietary Reference Intakes-United States Department of Agriculture (USDA) dietary guidelines for Americans, 2005) [35], encouraging exclusive breastfeeding for 6 months of age
- 11. maintenance of breastfeeding after introduction of solid food to 12 months of age and beyond, consistent with American Academy of Pediatrics recommendations [41, 42]
- 12. promoting moderate to vigorous physical activity for at least 60 min each day
- 13. limiting consumption of energy-dense foods

Stage 2: Structured Weight Management

This stage includes the support of a Registered Dietitian Nutritionist (RDN). A nutrition prescription including a mild energy deficit is included in this stage [34].

Nutrition Prescription The nutrition prescription is based upon the patient's resting metabolic rate (RMR) and is intended to promote normalization of growth and eating patterns. If the RMR cannot be measured, then the Institute of Medicine's (IOM's) total energy expenditure equations for overweight boys, girls and adolescents can be used [34]. Formulas to estimate the energy required for maintaining energy balance for age- and gender-specific groups at three different physical activity levels are provided by the IOM's report on macronutrient Dietary Reference Intakes provided as defined in Table 12.3 [43].

Activity term	Definition		
Sedentary	Reflects basal metabolism, thermic effect of food and physical activities required for independent living		
Low active	Same as sedentary but includes daily physical activity equivalent to walking approximately 2 miles per day at 15–20 min per mile or an equal amount of other moderate-intensity activities, such as raking leaves, vigorous house work or low-impact aerobics class, each day		
Active	Same as sedentary but includes daily physical activity equivalent to walking approximately 7 miles per day at 15–20 min per mile—approximately 1 h and 45 min to 2 h and 40 min or 70 min of vigorous-intensity activities (bicycle riding, tennis or jogging)		
Very active	Same as sedentary but includes daily physical activity equivalent to walking approximately 7 miles per day at the rate of 3–4 miles per hour		

 Table 12.3
 Definition of Levels of Activity

Institute of Medicine (IOM) 2005

IOM equations have been validated in overweight children and adolescents aged 3-18 years

Estimated EER overweight females (3-18 years)
EER+389 – (41.2 × age in years)=(physical activity × [15 × weight in kg=701.6 × height in meters])
Female activity factors:
Sedentary: 1
Low active: 1.18
Active 1.35
Very active: 1.60

 Table 12.4
 Estimated Energy Requirement (EER)

Institute of Medicine (2005)

To promote a 1-lb weight loss per month, subtract 108 kcal/day from the estimated energy requirement (EER) (Table 12.4).

Balanced hypo-caloric diets are prescribed to achieve a healthier weight or promote weight stabilization. They should include 45–65% of energy from carbohydrate; 10–35% of energy from protein and 20–35% of energy from fat [34]. Nutrition prescriptions are used to help guide the clinician and not always disclosed to the parent or the patient. They can be translated into an eating plan. A meal and snack schedule as well as 1 h of supervised and planned daily physical activity are also included. Self-monitoring is implemented by the family, with staff support using motivational interviewing techniques to help set goals and identify barriers [34].

Stage 3: Comprehensive Multidisciplinary Intervention

The intensity of behavior changes and frequency of visits is increased in this intervention. An interdisciplinary team of specialists with experience working with overweight or obese children is also added. During this phase, medical nutrition therapy (MNT) should be a minimum of 3 months or until baseline weight management goals are reached. More successful weight loss and weight maintenance may be achieved by increasing the frequency of contacts between the patient and practitioner [34].

Stage 4: Tertiary Care Intervention

This stage may be appropriate for severely obese children (BMI-for-age greater than the 99 percentile) who have attempted the Stage 3 intervention. Interventions are more aggressive and include medications, very-low energy diets and weight-control surgery with standard clinical protocols.

Recommended Diet for Prevention

Nutrition plays a vital role in the healthy growth and development of children. A healthy diet and regular physical activity are important to prevent obesity in children. Eating patterns established in childhood often track into adulthood.

USDA Dietary Guidelines

The 2010 Dietary Guidelines for Americans developed by the USDA and the United States Department of Health and Human Services (USDHHS) provide recommendations for healthy eating and maintenance of a healthy weight based on current nutrition research [35]. The 2015 guidelines are expected to be released in late 2015 or early 2016. The current 2010 Dietary Guidelines are intended for all Americans ages 2 years and older including those at increased risk of chronic disease. Children are a particularly important focus of the guidelines because of the epidemic of childhood overweight and obesity in addition to the growing body of evidence that optimal nutrition plays a vital role throughout the lifespan.

According to the current dietary guidelines, a healthy eating pattern (1) includes only enough calories from foods and beverages to meet calorie and nutrient needs, (2) is balanced with physical activity, (3) limits intake of sodium, solid fats, added sugars and refined grains and (4) emphasizes nutrient-dense foods and beverages such as vegetables, fruits, whole grains, fat-free or low-fat milk products, seafood, lean meats and poultry, eggs, beans and peas, and nuts and seeds.

Key Recommendations

Balancing Calories to Manage Weight

- Prevent and/or reduce overweight and obesity through improved eating and physical activity behaviors.
- Control total calorie intake to manage body weight. For people who are overweight or obese, this will mean consuming fewer calories from foods and beverages.
- Increase physical activity and reduce time spent in sedentary behaviors.
- Maintain appropriate calorie balance during each stage of life: childhood, adolescence, adulthood, pregnancy and breastfeeding, and older age.

Foods and Food Components to Reduce

- Reduce daily sodium intake to less than 2,300 milligrams (mg) and further reduce intake to 1,500 mg among persons who are 51 and older and those who are African American at any age or have hypertension, diabetes or chronic kidney disease. The 1,500 mg recommendation applies to about half of the US population, including children and the majority of adults.
- Consume less than 10% of calories from saturated fatty acids by replacing them with monounsaturated and polyunsaturated fatty acids.
- Consume less than 300 mg per day of dietary cholesterol.
- Keep trans fatty acid consumption as low as possible by limiting foods that contain synthetic sources of trans fats, such as partially hydrogenated oils, and by limiting other solid fats.
- Reduce the intake of calories from solid fats and added sugars. A total of 5–15% of calories from solid fats and added sugars are reasonable in the USDA food patterns. To help accomplish this, limit added fats and sugars when cooking and consume smaller portions of foods and beverages that contain added fats such as grain based desserts, sodas and other sugar sweetened beverages.
- Limit the consumption of foods that contain refined grains, especially refined grain foods that contain solid fats, added sugars and sodium.

Foods and Nutrients to Increase

Individuals should meet the following recommendations as part of a healthy eating pattern while staying within their calorie needs:

- Increase vegetable and fruit intake. Fruits and vegetables prepared without added fats or sugars are relatively low in calories. Eating these instead of higher calorie foods can help children achieve and maintain a healthy weight. Consumption of fruits and vegetables is also associated with reduced risk of chronic disease. Two and a half cups of vegetables and fruits per day is associated with a reduced risk of cardiovascular disease, including heart attack and stroke [35].
- Eat a variety of vegetables, especially dark-green, red and orange vegetables and beans and peas.
- Consume at least half of all grains as whole grains. Increase whole-grain intake by replacing refined grains with whole grains.
- Increase intake of fat-free or low-fat milk and milk products such as milk, yogurt and cheese or fortified soy beverages.
- Choose a variety of protein foods, which include seafood, lean meat and poultry, eggs, beans and peas, soy products, and unsalted nuts and seeds.
- Increase the amount and variety of seafood consumed by choosing seafood in place of some meat and poultry.
- Replace protein foods that are higher in solid fats with choices that are lower in solid fats and calories and/or are sources of oils.
- Use oils to replace solid fats when possible.
- Choose foods that provide more potassium, dietary fiber, calcium and vitamin D, which are nutrients of concern in American diets. These foods include vegetables, fruits, whole grains and milk and milk products [35].

USDA Plate Model

Choose MyPlate illustrates the five food groups in the image of a place setting to guide American's food choices. The MyPlate or Plate Model represents a meal (plate) containing ½ vegetables or a combination of vegetables and fruits, ¼ whole grains or starch and ¼ lean protein foods. It is accompanied by a cup of milk. A daily food plan based on calorie levels can show targets for recommended daily servings of major food groups. See www.choosemyplate.gov

American Heart Association (AHA) Dietary Recommendations for Healthy Children

The AHA dietary recommendations start in infancy. Breastfeeding is considered ideal nutrition and sufficient to support optimal growth and development for the first 4–6 months of life. Maintaining breast feeding for 12 months is encouraged. Transition to other sources of nutrients should begin around 4–6 months of age to ensure sufficient micronutrients in the diet. It is recommended that 100% fruit juice introduction is delayed until at least 6 months of age and further limit to no more than 4–6 oz per day. Juice should only be fed from a cup. Overfeeding infants and young children is discouraged acknowledging that caloric intake can vary from meal to meal, and self-regulation abilities usually help growing children to consume the calories they need from day to day. Healthy foods should be introduced and offered even if they are initially refused. Low nutritional value foods should be avoided.

Eating patterns for families are also recommended by the AHA. Energy (calories) should be adequate to help support growth and development and reach or maintain desirable body weights. Foods low in saturated fat, trans fat, cholesterol, sodium and added sugars are encouraged. A total of 30–35 % of calories should come from fat for children 2–3 years of age. A total of 25–35 % of calories should come from fat for children and adolescents 4–18 years of age. The majority of the fats should come from monounsaturated and polyunsaturated sources such as fish, nuts and vegetable oils. A variety of carbohydrates including whole grain, high-fiber breads and cereals and fruits and vegetables are recommended. Each meal should contain at least one fruit or vegetable. One to two cups of fruits and three fourth to three cups of vegetables per day are recommended for children depending on age. Fruit juice should be limited. A variety of lean protein foods are recommended. Fish should be introduced and regularly served. Fat-free and low-fat dairy foods are also recommended. Two cups of milk or milk equivalents per day are needed for children ages 1–8 years. Three cups per day are needed for children ages 9–18 years. Overfeeding children is discouraged. Eating only enough calories to maintain a healthy weight is recommended. Sixty min or 1 h of physical activity every day is recommended for all children [44].

Sugar-Sweetened Beverages

The USDA Dietary Guidelines recommend reduced intakes of SSB for all children. Strong evidence shows that children and adolescents who consume more sugar-sweetened beverages have higher body weights compared to those who drink less [35]. SSB were the fourth highest source of calories among Americans ages 2 years and older according to the NHANES, 2005–2006 report. SSB provide excess calories and few essential nutrients to the diet and should only be consumed when nutrient needs have been met and without exceeding daily calorie limits. Intakes of 100% fruit juice for children and adolescents, especially those who are overweight or obese, should be monitored for appropriate portion size [35].

Physical Activity

The 2008 Physical Activity Guidelines for Americans (PAG) provides guidance to help Americans improve their health, including their weights, through appropriate physical activity. Children and adolescents ages 6-17 years should do 60 min (1 h) or more of physical activity daily. Types of activities include a combination of aerobic, muscle-strengthening and bone-strengthening type exercises. Aerobic activities include moderate (increases heart rate and breathing to a 5 or 6 on a scale of 0-10) or vigorous (increases heart rate and breathing to a 7 or 8 on a scale of 0-10) intensity exercises. Moderate types of activities include brisk walking, dancing, swimming or bicycling on a level terrain. Vigorous types include jogging, singles tennis, swimming continuous laps or bicycling uphill. Aerobic activity should be included at least 3 days of the week. Musclestrengthening activities increase skeletal muscle strength, power, endurance and mass. They include strength training, resistance training, and muscular strength and endurance type exercises. Muscle-strengthening exercises are recommended 3 days per week. Bone-strengthening activities produce an impact or tension on bones, which promotes bone growth and strength. Running, jumping rope and lifting weights are examples. Bone-strengthening type exercises are recommended on at least 3 days of the week. Physical activity for young people should be age appropriate, enjoyable and offer variety [45].

Screen Time

Children and adolescents are encouraged to spend no more than 2 h each day watching television, playing electronic games or using the computer. It is also recommended that eating is avoided while watching television which can result in overeating [35].

Dyslipidemias

Lipoproteins are complex particles that transport triglyceride, cholesterol, phospholipid and protein in the blood. Defects in the production, transport and/or clearance pathways for lipoproteins can result in abnormally high or low blood lipid concentrations (dyslipidemias). Depending on the type, severity and duration of the dyslipidemia, adverse cardiovascular consequences can occur, sometimes early in life. Lipoprotein metabolism occurs in many steps and involves a diverse array of specialized proteins. A brief overview of lipoprotein metabolism follows along with a description of the most common types of dyslipidemias.

Lipoprotein Metabolism

Following digestion and absorption into the enterocyte, lipids are re-aggregated with apolipoprotein B-48 into large, triglyceride-rich particles called chylomicrons. Chylomicrons transport dietary fat and cholesterol from the small intestine to the liver and periphery. The triglycerides contained in the particles are hydrolyzed by the enzyme lipoprotein lipase that resides on the capillary endothelium of adipose and muscle tissue. Apolipoprotein C-2 is necessary for the activation of this enzyme; the chylomicron acquires this protein and apolipoprotein E in circulation through interaction with high density lipoprotein (HDL) [46]. When approximately 80% of the triglyceride has been removed from the chylomicron, it is considered a remnant and is cleared by the hepatic low-density lipoprotein receptor (LDLR) that recognizes and binds with apolipoprotein E [47].

Very low-density lipoprotein (VLDL) particles are assembled in the liver from endogenously derived triglyceride and cholesterol bound to apolipoprotein B-100. These particles are released into the circulation and are metabolized in a manner similar to the chylomicron: Apolipoprotein C-2 and E adsorb to the particle surface, lipoprotein lipase hydrolyzes the VLDL triglyceride and the particle decreases in size and becomes a remnant (also known as intermediate-density lipoprotein or IDL). In contrast to chylomicrons, only about 50% of LDL is cleared by the LDLR, which recognizes and binds to apolipoprotein B-100 on the particle surface [48]. The remaining remnant undergoes further hydrolysis by hepatic triglyceride lipase, leading to the formation of a cholesterol ester-enriched particle called low-density lipoprotein (LDL). Approximately 50% of LDL is taken up by the LDLR in the liver, adrenals and other tissues. The remaining LDL is cleared by non-receptor pathways, including scavenger receptors that recognized chemically modified phospholipids on the LDL surface [49]. High blood concentrations of LDL can lead to increased uptake into the vascular sub-endothelium where chemically modified LDL particles are recognized and engulfed by mature white blood cells called macrophages, leading to the formation of foam cells. Further accumulation of cholesterol, smooth muscle cells and calcium in the vascular wall can lead to the formation of hardened plaque, which is the hallmark of atherosclerosis and may lead to adverse cardiovascular consequences. High LDL-cholesterol is specifically associated with the atherosclerotic process [50].

HDL is formed in the plasma as lipid poor particles with phospholipid bound to apolipoprotein A-1 [51]. These particles can penetrate through the capillary endothelium to the surface of extrahepatic tissue, including vascular endothelium, where HDL collect free cholesterol and phospholipid [6]. Interaction with cholesterol ester transfer protein enables the exchange of cholesterol in HDL with triglyceride from lipoproteins containing apolipoprotein B100. This exchange ultimately allows cholesterol to be transported back to the liver and cleared by the LDLR [51, 52]. High HDL cholesterol is a positive factor for protection against atherosclerosis [53].

Common Dyslipidemias Several forms of dyslipidemias have strong genetic components, and some of the more common are described below.

Familial Hypercholesterolemia (FH) The most common type of gene disorder of lipoprotein metabolism is familial hypercholesterolemia (FH) (type II a hyperlipidemia). FH is an autosomaldominant trait caused by any one of 1,100 identified mutations in the LDLR gene [54]. The heterozygous form is found in approximately 1 in 250 persons, and plasma cholesterol levels are two to three times higher than normal [55]. Individuals with heterozygous FH are at risk for developing premature coronary artery disease (CAD) between the ages of 30 and 60 years [56]. The homozygous type of FH is found in one in one million persons, and plasma cholesterol levels can be four to six times higher than normal [55]. Due to the excessively high cholesterol in the circulation, deposits of cholesterol form in the tendons (xanthomas) and eyelids (xanthelasmas). Children with homozygous FH have premature CAD and should be treated aggressively. Often, LDL apheresis is used to selectively remove apolipoprotein B-containing particles from the circulation, which can reduce LDL concentrations up to 72 % [57].

Familial Combined Hyperlipidemia (FCHL) This autosomal-dominant disorder occurs in 1-2% of the Western population [58]. The disorder is most commonly caused by an overproduction of VLDL. Depending on the efficiency of VLDL catabolic and clearance pathways, patients may present with different lipoprotein patterns such as elevated LDL levels with normal triglyceride (type II a), elevated LDL levels with elevated triglycerides (type II b) or normal LDL with elevated triglycerides (type IV) [58]. A diagnosis of FCHL is based on the patient having two or more first-degree relatives with serum LDL-cholesterol or triglycerides above the 90th percentile. Patients with FCHL usually have other risk factors for CAD, including insulin resistance, central obesity and hypertension, and are at increased risk of premature CAD [59]. The clustering of CVD risk factors known as metabolic syndrome is becoming more prevalent in children and presents with a similar phenotype to FCHL. Central obesity generally precedes both glucose and lipid abnormalities. Weight management strategies are recommended as an important component of care for overweight children with FCHL and metabolic syndrome [60].

Familial Hypertriglyceridemia (FHTG) The population prevalence of this autosomal-dominant disorder is ~5–10% [58]. Individuals with FHTG have the metabolic defect of hepatic secretion of unusually large TG-rich VLDL particles that are catabolized slowly. The genetic basis for this defect is currently unknown. Although typically expressed in adulthood, the prevalence of FHTG is increasing in children. Obesity can accelerate the expression of FHTG, which presents as moderate to very high serum triglycerides (200–500 mg/dl range) and low to normal LDL and HDL cholesterol levels [61].

Low HDL Cholesterol Dyslipidemia characterized by low HDL cholesterol most commonly occurs concurrent with high TG, with or without elevations in small dense LDL-cholesterol. Obesity generally precedes dyslipidemia of this type; therefore, therapeutic lifestyle change strategies should be the focus of treatment [59]. Less common familial disorders of HDL are

hypoalphalipoproteinemia, Tangier's disease and mutations of the apolipoprotein A-1. These disorders are characterized by a low HDL cholesterol but no other lipid abnormality [62]. The risk of premature CAD in these patients is mild to moderate.

Recommended Dietary Approached for Pediatric Dyslipidemias

Current pediatric recommendations for treating identified dyslipidemia include a two-step approach called the Cardiovascular Health Integrated Lifestyle Diet or CHILD-1 and CHILD-2 [63]. CHILD-1 is appropriate for all children and is consistent with the 2010 Dietary Guidelines for Americans (DGA) [64]. It is considered the first-line approach to managing elevated LDL-cholesterol level. CHILD-1 recommends a dietary fat composition of 25-35% of calories with less than 10% of calories from saturated fat, no or minimal trans fats and fewer than 300 mg of cholesterol per day. This latter recommendation may be revised soon based on the 2015 Dietary Guidelines Advisory Committee (DGAC) [65] recommendation that cholesterol not be designated as a nutrient of concern for overconsumption. This recommendation is based on available evidence showing no appreciable relationship between dietary cholesterol intake and serum cholesterol. For children with persistently high elevated LDLcholesterol, e.g., after 3-6 months of compliance with CHILD-1, the CHILD-2 diet is recommended. Restrictions of saturated fat to <7 % of total calories and cholesterol to <200 mg/day are advised on CHILD-2 (LDL). For both CHILD-1 and CHILD-2, weight management is recommended for children with a BMI ≥85th percentile. According to CHILD guidelines, weight-reduction approaches should focus on decreasing the child's weight-for-height (BMI) percentile while maintaining linear growth [63].

Approaches to achieve the total and saturated fat limits of CHILD-1 and -2 include early transition (between age 1 and 2 years) to a low-fat diet. This advice is supported by dietary safety data from the Special Turku Risk Intervention Program (STRIP) study that showed that dietary counseling to reduce saturated fat and cholesterol to DGA levels from 6 months to 20 years of age could be done safely under medical supervision [66]. In the STRIP study, indicators of diet safety included optimal growth and pubertal development, nutrient adequacy and favorable cardiovascular health metrics (lowering of LDL-cholesterol and blood pressure) in infants, children and adolescents [66]. To achieve the reduced fat goals of CHILD-1, which include a minimum of 30% of energy for children ages 2–3 years and a minimum of 25% of energy for older children, emphasis should be placed on dietary patterns high in fruits, vegetables and whole grains [63]. Unprocessed forms of these foods are low in fat and high in fiber and will help to add bulk to the diet. Consumption of high-fiber foods has been shown to be an effective means of displacing calorically dense, nutrient poor foods [67]. Transition to reduce fat, unflavored milk and lean protein sources is also recommended by age 2 years with special consideration given to gender and age-specific calorie and nutrient requirements for growth and development [64].

The Dietary Approaches to Stop Hypertension (DASH) diet [68, 69] is comparable to the CHILD-1 food plan. A Mediterranean-type diet, if appropriately planned, may also conform to CHILD-1 [70, 71]. These dietary patterns emphasize whole, unprocessed foods, unsaturated forms of fats and plants sources of protein. These food patterns have been recognized as effective in modifying cardiovascular risk factors including lowering blood pressure, improving blood lipids, and promoting weight loss [68–71]. Recently, greater improvement in vascular function was reported in obese adolescents with hypercholesterolemia on a 12-week Mediterranean-type diet intervention compared to a control group [68]. Meal plans have been published for these food patterns (68.70) and can be modified for progressive saturated fat lowering and calorie and nutrient adequacy for children. Caution should be applied in this regard as dietary fat reduction below the lower limit established for CHILD-2 (LDL) has been associated with suboptimal intakes of calcium, zinc, vitamin E and phosphorus [72] and possible

failure to thrive [73]. Therefore, CHILD-2 (LDL) diet should be administered under medical supervision and with consultation with a registered dietitian if possible. The expected LDL-cholesterol reduction for children with hypercholesterolemia on CHILD-2 (LDL) is 3–10% [74].

The efficacy of plant sterols for blood cholesterol lowering has been evaluated in children and adolescents with heterozygous FH. In these studies, plant sterols given in the form of prepared muffins, margarines or chews in combination with a low-fat diet contributed to average LDL-cholesterol reductions of 5–15% [75, 76]. Similar trials in adults with FH have demonstrated LDL-cholesterol improvements, but no positive impact on CVD events [77]. Concern has been raised about the potential for the malabsorption of fat and fat-soluble vitamins in children consuming plant sterols chronically [78]. For this reason, it is recommended that food preparations with plant sterols should be reserved for children with moderate to severe elevations in cholesterol with appropriate monitoring for fat-soluble vitamin status [63]. Soluble fiber given as psyllium in a dose of up to 6 g/day with a low-fat diet provided an additional 5–10% reduction in LDL-cholesterol [79, 80]. In general, the extent to which dietary adjuncts lower LDL-cholesterol depends on previous dietary intake and baseline LDL-cholesterol level.

A dietary pattern called CHILD-2 (TG) is recommended for children with dyslipidemia characterized by high TG and/or low HDL-cholesterol [63]. This diet emphasizes a dietary pattern low in saturated fats, trans fats and refined carbohydrates. CHILD-2 (TG) emphasizes substituting unsaturated fats for saturated fats, decreasing intake from sugar-sweetened beverages and desserts, and substituting refined carbohydrates from highly processed breads, rice and pasta with whole grain sources of these same foods. Processed foods high in sugar are generally high in calories, which may contribute to weight gain and elevate serum TG. Modest weight loss for children with a BMI \geq 85th percentile should favorably modify serum TG [81]. In addition, up to 60 min daily of moderate to vigorous activity is recommended for children ages 2–21 years with elevated TG as this degree of activity effectively reduces TG [82]. For children with very high TG, such as those with hyperchylomicronemia, medium chain triglyceride (MCT) oils may be considered. MCTs are comprised of fatty acids that are absorbed efficiently without the need for emulsification and do not require packaging into chylomicron for transport; therefore, MCTs are particularly beneficial for disorders that have defects in bile secretion or chylomicron aggregation or clearance processes [83].

Hypertension

High blood pressure results when there is an increase in cardiac output or vascular resistance without a compensatory decrease in the variable's counterpart [84]. According to the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents, pediatric hypertension is defined as having a systolic and/or diastolic blood pressure measure greater than or equal to the 95th percentile for age, gender and height on multiple occasions; while prehypertension is defined as having a systolic blood pressure above the 90th percentile but below the 95th percentile for age, gender and height or for adolescence above 120/80 mmHg [85]. Based on recent survey data, childhood hypertension occurs in approximately 1-5% of the pediatric population when repeated screenings are performed [86, 87] and prehypertension at a prevalence of 9-12% [86]. Diagnosis of pre-hypertension should be confirmed with no less than three blood pressure measurements taken on three separate occasions using standardized techniques and age-appropriate equipment [84, 88]. Primary hypertension, or essential hypertension, is hypertension for which there is no identifiable cause. By contrast, secondary hypertension manifests as a result of another medical condition such as chronic renal disease or obstructive sleep apnea. Early in life, secondary forms of hypertension are most common, while primary hypertension becomes more prevalent with increasing

age [88]. The prevalence of high blood pressure and obesity tends to be greater in minority children [89]. In treating pediatric hypertension, non-pharmacologic lifestyle intervention should be the first line of intervention [90].

Approaches to Intervention for Hypertension

Weight Loss for Overweight

One of the strongest predictors of pediatric hypertension is overweight and obesity [86], accounting for more than half of all cases of primary pediatric hypertension [91]. Tu et al. found that the effect of adiposity on blood pressure was minimal until a child reached a body mass index (BMI) >85th percentile, at which point blood pressure quadrupled [92]. This finding held true independent of race or sex. For overweight African American females, each 5 % increase in BMI percentile increased the risk of hypertension or pre-hypertension by 33 %; the risk was nearly doubled that for other race or gender groups [92]. Lifestyle interventions that achieve weight loss have consistently shown a favorable impact on blood pressure and other cardio-metabolic risk factors [93]. For example, in a 1-year study of diet and physical activity intervention in overweight children, a decrease in BMI of 0.5 or greater was associated with a 21-mmHg reduction in systolic blood pressure and a 9-mmHg reduction in diastolic blood pressure [94]. Weight loss, therefore, is recommended as a primary intervention in the effort to reduce blood pressure in hypertensive, overweight and obese children [85, 87]. Indeed, studies in young adults showed that a 10-kg weight reduction was associated with a 5- to 20-mmHg decrease in systolic blood pressure [95]. Importantly, in these studies, findings suggest that it is not necessary for patients to achieve their ideal body weight in order to gain significant blood pressure benefits. The exact mechanism by which overweight contributes to hypertension remains uncertain; however, it is traditionally accepted that increased sympathetic activity plays a role [96].

Dietary Patterns Emphasizing Fruits, Vegetables, and Low-Fat Dairy

In single-nutrient studies, several minerals including potassium, calcium, magnesium and dietary fiber have been identified as exerting favorable effects on blood pressure in youth, although the evidence is conflicting [90, 91, 97]. Capitalizing on the potential for a synergistic effect of these nutrients when combined, the Dietary Approaches to Stop Hypertension (DASH) diet was developed. The DASH diet emphasizes foods rich in the aforementioned minerals and fiber for lowering blood pressure, while minimizing nutrients associated with elevated blood pressure such as saturated fat. Specifically, for a 2,000-cal diet, the DASH diet recommends 7-10 servings/day of fruits and vegetables, 2-3 servings/day of low-fat or non-fat dairy, beans and nuts 4-5 times per week, 2 or less servings/day of lean meats, fish and poultry, 7-8 servings/day of grains (at least 3 of these should be whole grains) and a limited intake of foods high in fat, sugar and sodium. In a small sample of hypertensive and pre-hypertensive children, a behaviorally focused dietary intervention emphasizing the DASH dietary pattern showed promise as an effective means of lowering blood pressure [98, 99]. In this study, 50% of hypertensive and pre-hypertensive youth achieved blood pressure normalization following a DASH-type dietary intervention compared to just 36% of those following routine nutrition care offered to this population. The relative change in systolic blood pressure in this study was -7.9%in the DASH diet group compared to -1.5% in the usual care group, which was significantly different.

Individual components of the DASH dietary pattern, specifically fruits, vegetables and low-fat dairy, have also proven efficacious in lowering blood pressure in children [100–102]. In a recent study by Moore et al., girls 9–10 years of age who consumed two or more servings of dairy per day experienced a 33% reduced risk of elevated blood pressure late in adolescence compared to those who did not. The combined intake of both dairy products and fruits and vegetables (DASH-like diet) resulted in a 36% reduction in the risk of high blood pressure in late adolescence. This effect was not observed with increased fruit and vegetable intake alone [103]. Similarly, using 8 years of follow-up data from the Framingham Children's Study, children with the highest fruit, vegetable and dairy intakes experienced the smallest age-related blood pressure increases among their peers. The combination of high fruit, vegetable and dairy intake provided the greatest blood pressure-lowering benefits compared with high intakes of any of the food groups consumed alone [103]. Taken as a whole, these studies support the use of a DASH-type dietary pattern as a means of lowering high blood pressure in a hypertensive pediatric population. Enlisting the aid of a registered dietitian may be helpful in implementing a DASH meal plan in this population [85, 104].

Dietary Sodium

Reducing dietary sodium has also been shown to significantly decrease blood pressure in children [105, 106]. In a pooled analysis of 13 controlled trials in children less than 18 years of age, of which all but one was randomized, a significant reduction in both systolic and diastolic blood pressures was associated with a modest reduction in salt consumption [107]. Of the ten studies conducted on children and adolescents, a median net reduction of salt intake of 42% over 4 weeks correlated with a decrease in systolic and diastolic blood pressure of 1.17 mmHg and 1.29 mmHg, respectively. In the same pooled analysis, three intervention trials in infants found that a 54% median net reduction in salt intake over 20 weeks was associated with an average decrease in systolic blood pressure of 2.47 mmHg [107]. Much of the salt in the infant diet was found to come with the introduction of solid foods at \sim 6–9 months of age.

Reducing dietary sodium is recommended, especially in hypertensive patients and those who are overweight or obese, as these at-risk subgroups frequently exhibit increased sodium sensitivity [108, 109]. The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents recommends that sodium intake be kept to 1,200 mg/day for 4–8-year-olds and 1,500 mg/day for older children [85]. Given that the average sodium intake of children and adolescents is well above this value, reducing dietary sodium to the recommended levels may be challenging [110]. Therefore, focusing on key sources of sodium in the diets of children and working collaboratively to limit those foods is an important first step. In a recent study of first-year middle school children in Italy, the impact of snack intake on blood pressure was investigated. Snack intake was found to account for nearly half of participants' total sodium intake and was highly correlated with children's blood pressure. In this study, children with systolic and diastolic blood pressure in the highest quartile had a significantly higher sodium intake from snacks, as well as greater weight, BMI and BMI percentiles [111]. In the USA, an average of 83 % of total sodium consumption comes from pre-prepared foods purchased from grocery stores (65%) and restaurants (18%) [112]; pizza and breads/rolls are the greatest sources of sodium in the diets of children, ages 2–18 [112]. Therefore, replacing regular versions of these foods in favor of low sodium varieties would be a useful strategy in lowering sodium in children's diets. In addition, foods cooked from scratch are naturally lower in sodium than most instant and boxed meals and take-out foods. To aid in sodium reduction, families should be assessed for their food literacy and counseled accordingly on food shopping skills and their ability to read food labels, cook and flavor foods using low sodium methods [106].

Conclusion

It is clear that lifestyle factors, including diet, physical activity and sleep, play a very important role in first preventing the emergence of cardiovascular risk factors, and, second, in treating risk factors, such as obesity, dyslipidemia and hypertension, when they develop in young individuals.

Identifying risk factor development in children and adolescents is important so that appropriate treatment and intervention can be implemented. This is the rationale behind the recommendations that BMI and blood pressure are measured at health maintenance visits after age 2 and 3 years, respectively. This is also the rationale for measuring cholesterol once in all children between age 9 and 11 years.

Unfortunately, children and adolescents do not usually live in an environment that makes the health choice for diet, physical activity or sleep the easy choice. This means that behavior change interventions must focus on changing the home environment to encourage healthier choices. Often, the best approach is for the entire family to work together to make improved lifestyle choices. This may be broadly beneficial as often cardiovascular risk factors will cluster in families because of shared environment and shared genetics.

Identification and management of cardiovascular risk factors in children and adolescents requires a team of healthcare professionals, including physicians, nurses, dietitians and exercise physiologists. It is clear that appropriate management can be successful, which should provide a sense of optimism. On the other hand, the prevention of the development of risk factors in the first place is by far the best strategy.

References

- Mozaffarian D, Benjamin EJ, Go AS, Arnett DK, Blaha MJ, Cushman M, de Ferranti S, Després JP, Fullerton HJ, Howard VJ, Huffman MD, Judd SE, Kissela BM, Lackland DT, Lichtman JH, Lisabeth LD, Liu S, Mackey RH, Matchar DB, McGuire DK, Mohler 3rd ER, Moy CS, Muntner P, Mussolino ME, Nasir K, Neumar RW, Nichol G, Palaniappan L, Pandey DK, Reeves MJ, Rodriguez CJ, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Willey JZ, Woo D, Yeh RW, Turner MB, American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2015 update: a report from the American Heart Association. Circulation. 2015;131:e29–322.
- Berenson GS, Srinivasan SR, Bao W, Newman 3rd WP, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. N Engl J Med. 1998;338:1650–6.
- Lauer RM, Connor WE, Leaverton PE, Reiter MA, Clarke WR. Coronary heart disease risk factors in school children: the Muscatine study. J Pediatr. 1975;86:697–706.
- Raitakari OT, Juonala M, Rönnemaa T, Keltikangas-Järvinen L, Räsänen L, Pietikäinen M, Hutri-Kähönen N, Taittonen L, Jokinen E, Marniemi J, Jula A, Telama R, Kähönen M, Lehtimäki T, Akerblom HK, Viikari JS. Cohort profile: the cardiovascular risk in Young Finns Study. Int J Epidemiol. 2008;37:1220–6.
- 5. Gall SL, Jose K, Smith K, Dwyer T, Venn A. The childhood determinants of adult health study: a profile of a cohort study to examine the childhood influences on adult cardiovascular health. Australas Epidemiol. 2009;16:35–9.
- Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999–2010. JAMA. 2012;307:483–90.
- Dabelea D, Mayer-Davis EJ, Saydah S, Imperatore G, Linder B, Divers J, Bell R, Badaru A, Talton JW, Crume T, Liese AD, Merchant AT, Lawrence JM, Reynolds K, Dolan L, Liu LL, Hamman RF, SEARCH for Diabetes in Youth Study. Prevalence of type 1 and type 2 diabetes among children and adolescents from 2001 to 2009. JAMA. 2014;311:1779–86.
- Goff Jr DC, Gerstein HC, Ginsberg HN, Cushman WC, Margolis KL, Byington RP, Buse JB, Genuth S, Probstfield JL, Simons-Morton DG, ACCORD Study Group. Prevention of cardiovascular disease in persons with type 2 diabetes mellitus: current knowledge and rationale for the Action to Control Cardiovascular Risk in Diabetes (ACCORD) trial. Am J Cardiol. 2007;99:4i–20.

- Kit BK, Kuklina E, Carroll MD, Ostchega Y, Freedman DS, Ogden CL. Prevalence of and trends in dyslipidemia and blood pressure among US children and adolescents, 1999–2012. JAMA Pediatr. 2015;169:272–9.
- 10. Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, Greenlund K, Daniels S, Nichol G, Tomaselli GF, Arnett DK, Fonarow GC, Ho PM, Lauer MS, Masoudi FA, Robertson RM, Roger V, Schwamm LH, Sorlie P, Yancy CW, Rosamond WD, American Heart Association Strategic Planning Task Force and Statistics Committee. Defining and setting national goals for cardiovascular health promotion and disease reduction: the American Heart Association's Strategic Impact Goal through 2020 and beyond. Circulation. 2010;121: 586–613.
- 11. Liu K, Daviglus ML, Loria CM, Colangelo LA, Spring B, Moller AC, Lloyd-Jones DM. Healthy lifestyle through young adulthood and the presence of low cardiovascular disease risk profile in middle age: the Coronary Artery Risk Development in (Young) Adults (CARDIA) study. Circulation. 2012;125:996–1004.
- 12. Stampfer MJ, Hu FB, Manson JE, Rimm EB, Willett WC. Primary prevention of coronary heart disease in women through diet and lifestyle. N Engl J Med. 2000;343:16–22.
- McGuire S. U.S. Department of Agriculture and U.S. Department of Health and Human Services, Dietary Guidelines for Americans, 2010. 7th Edition, Washington, DC: U.S. Government Printing Office, January 2011. Adv Nutr. 2011;2:293–4.
- Centers for Disease Control (CDC) Childhood Obesity Facts. Prevalence of childhood obesity in the United States, 2011–2012. http://www.cdc.gov/obesity/data/childhood.html.
- Fryar CD, Carroll MD, Ogden CL. Prevalence of overweight and obesity among children and adolescents: United States, 1963–1965 through 2011–2012. Division of Health and Nutrition Examination Surveys. September, 2014.
- Ogden CL, Lamb MM, Carroll MD, Flegal KM. Obesity and socioeconomic status in children and adolescents: United States, 2005–2008. NCHS Data Brief. 2010;51:1–8.
- 17. Whitlock EP, Williams SB, Gold R, Smith PR, Shipman SA. Screening and interventions for childhood overweight: a summary of evidence for the US Preventative Services Task Force. Pediatrics. 2005;116(1). Available at www.pediatrics.org/cgi/conent/full/116/1e125_
- 2000 CDC Growth Charts. BMI for age 2–20 years Boys and Girls. http://www.cdc.gov/growthcharts/data/setlclinical/cj41c019.pdf.
- Pietrobelli A, Faith MS, Allison DB, Gallagher D, Chiumello G, Heymsfield SB. Body mass index as a measure of adiposity among children and adolescents: a validation study. J Pediatr. 1998;132:204–10.
- Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, Dietz WH. Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. Am J Clin Nutr. 2002;75:978–85.
- Taveras EM, Rifas-Shiman SL, Oken E, Haines J, Kleinman K, Rich-Edwards JW, Gillman MW. Crossing growth percentiles in infancy and risk of obesity in childhood. Arch Pediatr Adolesc Med. 2011;165(11):993–8.
- Barlow SE. Expert Committee recommendations regarding the prevention, assessment and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007;120 Suppl 4:S164–92.
- Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. Pediatrics. 2001;108(3):712–8.
- 24. Weiss R, Dziura J, Burget T, Tamborlane W, Taksali S, Yeckel C, Allen K, Lopes M, Savoye M, Morrison J, Sherwin R, Caprio S. Obesity and metabolic syndrome in children and adolescents. N Engl J Med. 2004;350:2362–74.
- Wing YK, Hui SH, Pak WM, Ho CK, Cheung A, Li AM, Fok TF. A controlled study of sleep-related disordered breathing in obese children. Arch Dis Child. 2003;88(12):1043–104.
- Patton H, Sirlin C, Behling C, Middleton M, Schwimmer J, Lavine J. Pediatric nonalcoholic fatty liver disease: a critical appraisal of current data and implications for future research. J Pediatr Gastroenterol Nutr. 2006;43(4):413–27.
- Fagot-Campagna A, Petit DJ, Engelgau MM, Burrows NR, Geiss LS, Valez R. Type 2 diabetes among North American children and adolescents: an epidemiologic review and a public health perspective. J Pediatr. 2000;136(5):664–72.
- 28. Han JC, Lawlor DA, Kimm SY. Childhood obesity. Lancet. 2010;375(9727):1737–48.
- 29. Sutherland ER. Obesity and asthma. Immunol Allergy Clin North Am. 2008;28(3):589-602, ix.
- Taylor ED, Theim KR, Mirch MC, et al. Orthopedic complications of overweight in children and adolescents. Pediatrics. 2006;117(6):2167–74.
- Morrison KM, et al. Association of depression & health related quality of life with body composition in children and youth with obesity. J Affect Disord. 2015;172:18–23.
- 32. Mustillo S, et al. Obesity and psychiatric disorder: developmental trajectories. Pediatrics. 2003;111(4):851-9.
- 33. Halfon N, Larson K, Slusser W. Associations between obesity and comorbid mental health, developmental, and physical health conditions in a nationally representative sample of US children aged 10 to 17. Acad Pediatr. 2013;13(1):6–13.

- 34. Academy of Nutrition and Dietetics Evidence Analysis Library (EAL). Evidence-based pediatric weight management nutrition practice guideline. Published May 2007. Available at: http://www.andeal.org (login required).
- 35. U.S. Department of Agriculture U.S. Department of Health and Human Services. Dietary Guidelines for Americans, 2010. www.dietaryguidelines.gov.
- Berge JM, Rowley S, Trofholz A, Hanson C, Rueter M, MacLehose RF, Neumark-Sztainer D. Childhood obesity and interpersonal dynamics during family meals. Pediatrics. 2014;134:923–32. doi:10.1542/peds.2014-1936.
- 37. Institute of Medicine. Food marketing to children and youth: threat or opportunity?. Washington, DC: National Academies Press; 2005.
- Cappuccio FP, Taggart FM, Kandala NB, Currie A, Peile E, Stranges S, Michelle MA. Meta-analysis of short sleep duration and obesity in children and adults. Sleep. 2008;31(5):619–26.
- Centers for Disease Control and Prevention. Children's food environment state indicator report, 2016. http://www. cdc.gov/obesity/downloads/childrensfoodenvironment.pdf. Accessed 16 Aug 2016.
- Larson N, Story M, Nelson M. Neighborhood environments: disparities in access to healthy foods in the U.S. Am J Prev Med. 2009;36(1):74–81.e10.
- Krebs NF, Himes JH, Jacobson D, Nicklas TA, Guilday P, Styne D. Assessment of child and adolescent overweight and obesity. Pediatrics. 2007;120 Suppl 4:S193–228.
- 42. Owen CG, Martin RM, Whincup PH, Smith GD, Cook DG. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. Pediatrics. 2005;115:1367–77.
- 43. Institute of Medicine. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients). Washington, DC: National Academies Press; 2005. p. 218–20.
- 44. American Heart Association (AHA). Dietary recommendations for healthy children. 2014. http://www.heart.org/ HEARTORG/GettingHealthy/Dietary-Recommendations-for-Healthy-Children_UCM_303886_Article.jsp#.
- 45. Physical Activity Guidelines for Americans (PAG) Chapter 3: Active Children and Adolescents. http://health.gov/ paguidelines/guidelines/chapter3.aspx. Office of disease prevention and health promotion (USDHHS).
- 46. Sook Sul H, Storch J. Cholesterol and lipoproteins: synthesis, transport, and metabolism. In Stipanuk, editor. Biochemical, physiological and molecular aspects of human metabolism. 3rd ed., St. Louis: Saunders/Elsevier, 2012. p. 393–415.
- 47. Wang H, Eckel RH. Lipoprotein lipase: from gene to obesity. Am J Physiol. 2009;297:E271-88.
- 48. Beglova N, Blacklow SC. The LDL receptor: how acid pulls the trigger. Trends Biochem Sci. 2005;30:309–17.
- 49. Riggotti A. Scavenger receptors and atherosclerosis. Biol Res. 2000;33:97-103.
- 50. Hegele RA. Plasma lipoproteins: genetic influences and clinical implications. Nat Rev Genet. 2009;10:109-21.
- Krimou L, Marcil M, Genest J. New insights into the biogenesis of human high-density lipoproteins. Curr Opin Lipidol. 2006;17:258–67.
- 52. Ohashi R, Mu H, Wang X, et al. Reverse cholesterol transport and cholesterol efflux in atherosclerosis. QJM. 2005;98:845–56.
- Schaefer EJ, Azstalos BF. Cholesteryl ester transfer protein inhibition, high density lipoprotein metabolism and heart disease risk reduction. Curr Opin Lipidol. 2006;17:394–8.
- Benn M. Apolipoprotein B, levels, APOB alleles, and risk of ischemic cardiovascular disease in the general population, a review. Atherosclerosis. 2009;206:17–30.
- McCrindle BW. Familial hypercholesterolemia in children and adolescents. Curr Opin Lipidol. 2012;23(6):525–31.
- Goldstein JL, Brown MS. History of discovery: the LDL receptor. Aterioscler Throm Vasc Biol. 2009;29: 431–8.
- Lee WP, Datta B, Ong BB, et al. Defining the role of lipoprotein apheresis in the management of familial hypercholesterolemia. Am J Cardiovasc Drugs. 2011;11:363–70.
- Miller M, Stone NJ, Ballantyne C, et al. Triglycerides and cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2011;123:2292–333.
- 59. Eckel RH, Grundy SM, Zimmer PZ. The metabolic syndrome. Lancet. 2005;365:1415-28.
- Gidding SS, Dennison BA, Birch LL, American Heart Association, et al. Dietary recommendations for children and adolescents: a practitioner's guide. Pediatrics. 2006;117:544–59.
- Berglund L, Brunzell JD, Goldberg AC, Goldberg IJ, Sacks F, Murad MH, Stalenhoef AF. Evaluation and treatment of hypertriglyceridemia: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2012;97(9):2969–89.
- Singh V, Sharma R, Kumar A, Deedwania P. Low high-density lipoprotein cholesterol: current status and future strategies for management. Vasc Health Risk Manag. 2010;6:979–96.
- Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents. Summary report. Pediatrics. 2011;128: S1–44.
- 64. US Department of Agriculture and US Department of Health and Human Services. Dietary guidelines for Americans 2010. 7th ed. Washington, DC: US Government Printing Office. 2011.

- 65. Dietary Guidelines Advisory Committee. Scientific report of the 2015 Dietary Guidelines Advisory Committee. Last updated 12/18/2015. Available at http://health.gov/dietaryguidelines/2015-scientific-report/. Accessed 18 Dec 2015.
- 66. Gidding S. The STRIP Study: long-term impact of a low saturated fat/low cholesterol diet. Current Cardiovasc Risk Rep. 2014;8:410.
- 67. Ritchie LD, Spector P, Stevens MJ, et al. Dietary patterns in adolescents are related to adiposity in young adulthood in black and white females. J Nutr. 137:399–406.
- 68. Van Horn L, Vincent E. The CHILD-1 and DASH diets: rationale and translational applications. Pediatr Ann. 2013;42:372–4.
- 69. Obarzanek E, Sacks FM, Vollmer WM, et al. Effects on blood lipids of a blood pressure lowering diet: the Dietary Approaches to Stop Hypertension (DASH) trial. Am J Clin Nutr. 2001;74:80–9.
- Willett WC, Sacks F, Trichopoulou A, et al. Mediterranean diet pyramid: a cultural model for healthy eating. Am J Clin Nutr. 1995;61:1402S–6.
- Giannini C, Diesse L, D'Adamo E, et al. Influence of the Mediterranean diet on carotid-intima media thickness in hypercholesterolemic children: a 12-month intervention study. Nutr Metab Cardiovasc Dis. 2014;24:75–82.
- Jacobson MS, Tomopoulus S, Williams CL, et al. Normal growth in high risk hyperlipidemic children and adolescents with dietary intervention. Prev Med. 1998;27:775–80.
- 73. Lifshitz F, Moses N. A complication of dietary treatment of hypercholesterolemia. Am J Dis Child. 1989;143:537–42.
- 74. Williams CL, Hayman LL, Daniels SR, et al. Cardiovascular health in childhood: a statement of health professionals from the Committee on Atherosclerosis, Hypertension, and Obesity in the Young (AHOY) of the Council on Cardiovascular Disease in the Young. American Heart Association. Circulation. 2002;106:143–60.
- 75. Tammi A, Ronnemaa T, Gylling H, et al. Plant stanol ester margarine lowers serum total and low-density lipoprotein cholesterol concentrations of health children: the STRIP project. Special Turku Coronary Risk Factors Intervention Project. J Pediatr. 2000;136:503–10.
- Gylling H, Siimes MS, Miettinen TA. Sitostanol ester margarine in dietary treatment of children with familial hypercholesterolemia. J Lipid Res. 1995;36:1807–12.
- 77. Malhotra A, Shafiq N, Arora A, Singh M, Kumar R, Malhotra S. Dietary interventions (plant sterols, stanols, omega-3 fatty acids, soy protein and dietary fibers) for familial hypercholesterolemia (Review). Cochrane Database Syst Rev. 2014;6:CD001918.
- 78. Expert Panel on Detection. Evaluation and Treatment of High Cholesterol in Adults (2001), Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on the Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 2001;285:2486–97.
- 79. Williams CL, Bollella M, Spark A, et al. Soluble fiber enhances the hypercholesterolemic effect of the step 1 diet in childhood. J Am Coll Nutr. 1995;14:251–7.
- Davidson MH, Dugan LD, Burns JH, et al. A psyllium-enriched cereal for treatment of hypercholesterolemia in children: a controlled, double-blind, cross-over study. Am J Clin Nutr. 1996;63:1147–54.
- 81. Johnson RK, Appel LJ, Brands M, et al. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. Circulation. 2009;120:1011–20.
- Graham TE. Exercise, postprandial triacylglyceridemia, and cardiovascular disease risk. Can J Appl Physiol. 2004;29:781–99.
- 83. Shah AS, Wilson DP. Primary hypertriglyceridemia in children and adolescents. J Clin Lipidol. 2015;9:520-8.
- Opie LH. The Heart: physiology, from cell to circulation. 4th ed. Philadelphia: Lippincott-Raven; 2004. p. 431–59.
- National High Blood Pressure Education Program Working Group on High Blood Pressure in Children. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics. 2004;114:555–76.
- Flynn J. The changing face of pediatric hypertension in the era of the childhood obesity epidemic. Pediatr Nephrol. 2012;28(7):1059–66.
- 87. Bassareo P, Mercuro G. Pediatric hypertension: an update on a burning problem. World J Cardiol. 2014;6(5):253-9.
- 88. Spagnolo A, Giussani M, Ambruzzi A, et al. Focus on prevention, diagnosis and treatment of hypertension in children and adolescents. Ital J Pediatr. 2013;39(1):20.
- Muntner P, He J, Cutler A, Wildman P, Whelton K. Trends in blood pressure among children and adolescents. JAMA. 2004;291(17):2107.
- Lande B, Kupferman J. Treatment of pediatric hypertension: lessons learned from recent studies. Curr Cardiovasc Risk Rep. 2013;7(5):307–14.
- 91. Torrance B, McGuire K, Lewanczuk R, McGavock J. Overweight, physical activity and high blood pressure in children: a review of the literature. Vasc Health Risk Manag. 2007;3(1):139.

- Tu W, Eckert G, DiMeglio L, Yu Z, Jung J, Pratt J. Intensified effect of adiposity on blood pressure in overweight and obese children. Hypertension. 2011;58(5):818–24.
- Ho M, Garnett SP, Baur L, Burrows T, Steward L, Neve M, Collins C. Effectiveness of lifestyle interventions in child obesity: systematic review with meta-analysis. Pediatrics. 2012;130:e1647–71.
- Reinehr T, Andler W. Changes in the atherogenic risk factor profile according to degree of weight loss. Arch Dis Child. 2004;89(5):419–22.
- 95. Chobanian A, Bakris G, Black H, National High Blood Pressure Education Program Coordinating Committee, et al. The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. JAMA. 2003;289(19):2560–71.
- Landsberg L. Insulin-mediated sympathetic stimulation: role in the pathogenesis of obesity-related hypertension (or, how insulin affects blood pressure, and why). J Hypertens. 2001;19(3):523–8.
- Simons-Morton D, Hunsberger S, Van Horn L, et al. Nutrient intake and blood pressure in the Dietary Intervention Study in Children. Hypertension. 1997;29(4):930–6.
- 98. Simons-Morton G, Obarzanek E. Diet and blood pressure in children and adolescents. Pediatr Nephrol. 1997;11(2):244–9.
- Couch S, Saelens B, Levin L, Dart K, Falciglia G, Daniels S. The efficacy of a clinic-based behavioral nutrition intervention emphasizing a DASH-type diet for adolescents with elevated blood pressure. J Pediatr. 2008;152(4):494–501.
- 100. Moore L, Bradlee M, Singer M, Qureshi M, Buendia J, Daniels S. Dietary Approaches to Stop Hypertension (DASH) eating pattern and risk of elevated blood pressure in adolescent girls. Br J Nutr. 2012;108(09):1678–85.
- 101. Damasceno M, de Araújo M, Freire de Freitas R, de Almeida P, Zanetti M. The association between blood pressure in adolescents and the consumption of fruits, vegetables and fruit juice – an exploratory study. J Clin Nurs. 2011;20(11–12):1553–60.
- Rangan A, Flood V, Denyer G, et al. The effect of dairy consumption on blood pressure in mid-childhood: CAPS cohort study. Eur J Clin Nutr. 2012;66(6):652–7.
- 103. Moore L, Singer M, Bradlee M, et al. Intake of fruits, vegetables, and dairy products in early childhood and subsequent blood pressure change. Epidemiology. 2005;16(1):4–11.
- 104. Flynn JT, Couch S, Daniels S. Non-pharmacologic treatment of pediatric hypertension. In: Flynn JT, Inglefinger JR, Portman RJ, editors. Pediatric hypertension. 3rd ed. Totowa: Humana Press; 2013. p. 529–37.
- 105. Hanevold C. Sodium intake and blood pressure in children. Curr Hypertens Rep. 2013;15(5):417-25.
- Appel L, Lichtenstein A, Callahan E, Sinaiko A, Van Horn L, Whitsel L. Reducing sodium intake in children: a public health investment. J Clin Hypertens. 2015;17(9):657–62.
- 107. He F, MacGregor G. Importance of salt in determining blood pressure in children: meta-analysis of controlled trials. Hypertension. 2006;48(5):861–9.
- 108. Kaplan NM, Flynn JT. Kaplan's clinical hypertension. 9th ed. Philadelphia: Lippincott Williams & Wilkins; 2006.
- 109. Cogswell M, Yuan K, Gunn J, et al. Vital signs: sodium intake among US school-aged children—2009–2010. Morb Mortal Wkly Rep (MMWR). 2014;63(36):789–97.
- Ponzo V, Ganzit G, Soldati L, et al. Blood pressure and sodium intake from snacks in adolescents. Eur J Clin Nutr. 2015;69(6):681–6.
- 111. Fox T, Hirschman J, Fox MK, Crepinsek MK. School Nutrition Dietary Assessment Survey IV: Current state of school meals and snacks. National Alliance for Nutrition and Activity 2013. Available at: http://www.mathematicampr.com/~/media/publications/pdfs/nutrition/snda-iv_webinar_13mar2013.pdf. Accessed 2015.
- 112. Sources of Sodium among the US Population, 2005–06. Applied Research Program Web site. National Cancer Institute. http://appliedresearch.cancer.gov/diet/foodsources/sodium/. Updated April 11, 2014 November 24, 2015.

Chapter 13 School Meal Programs: Are They Nutritionally Sound?

Dayle Hayes

Key Points

- Millions of US students enjoy school meals every day; the majority of these are served to children who are eligible for free and reduced-priced meals.
- Since the implementation of the 2010 Healthy, Hungry-Free Kids Act, improvements in school meal intake have been documented by both plate-waste studies and self-reported intakes.
- Improvements in school meals may also be connected to childhood BMIs improvements in some specific populations.
- Popular initiatives like farm to school, school gardens, and culinary education are becoming more common and may be enhancing school meal programs.
- Multiple resources are available to school nutrition programs seeking ways to meet school meal patterns and provide the best possible food to students in schools.

Keywords School nutrition • School breakfast • School lunch • Competitive foods • Local wellness policy • Farm to school • US Department of Agriculture • Healthy • Hunger-Free Kids Act

Abbreviations

CEP	Community Eligibility Provision
CDC	Centers for Disease Control and Prevention
CSH	Coordinated School Health
DGA	Dietary Guidelines for Americans
FFVP	Fresh Fruit and Vegetable Program
FRAC	Food Research and Action Center
HHFKA	Healthy Hungry-Free Kids Act
IOM	Institute of Medicine
NSLP	National School Lunch Program

D. Hayes, MS, RD

Nutrition for the Future, Inc., 3112 Farnam Street, Billings, MT 59102, USA e-mail: EatWellatSchool@gmail.com

SBP	School Breakfast Program
SFSP	Summer Food Service Program
SHPSS	School Health Policies and Practices Study
USDA	US Department of Agricultural
USDA=FNS	Department of Agriculture Food and Nutrition Services

Introduction

Schools in the USA serve over 14 million breakfasts and 30 million lunches every school day, as well as multiple snacks and supper meals in some locations. These meals are funded by federal legislation and regulated by the US Department of Agriculture (USDA) nutrition standards, which are designed to follow the Dietary Guidelines for Americans (DGAs). School nutrition professionals face many challenges when working to improve student consumption patterns and campus food environments.

For many decades, US school nutrition programs served millions of meals in relative obscurity. However, since 2004, school meals are frequently in the political, legislative, and news spotlight, featured in media headlines, Congressional debates, and YouTube viral videos. School nutrition professionals, once derided as "lunch ladies in hairnets," have become professional leaders in discussions about the rise of childhood obesity, the decline of youthful eating habits, and the appropriate role of schools in a supporting a healthier generation of American children.

Brief History of School Nutrition Programs in the USA

The USA has a long history of school feeding programs focused on the health, nutrition, and well-being of young people. While current programs are federally funded and regulated by the US Department of Agriculture (USDA), school food programs have been around since the mid-1800s. Philadelphia was one of the first cities to establish a school meal program with local funding from a variety of sources. Emma Smedley, a dietitian and first director in Philadelphia, wrote *The School Lunch: Its Organizations and Management in Philadelphia*, the first standards for school food [1].

The need for a national nutrition program for youth became more evident during World War II when 40–60% of draft eligible males failed military physicals because of malnutrition-related conditions [2]. In 1946, Congress passed and President Truman signed the National School Lunch Act outlining the purpose and policies for a comprehensive federal program offering a lunch for school-aged children. The goal was to serve lunch at a reasonable price for paying students and at no charge for those who were economically disadvantaged. The National School Lunch Program (NSLP) was designed to provide one-third to one-half of a child's daily calorie needs [3]. It was also acknowledged to be an agricultural support program for American farmers and ranchers.

Congress approved a second national school food program with the 1966 Child Nutrition Act, which introduced school breakfast meals as a pilot project. The national School Breakfast Program (SBP) was fully funded as an entitlement program in 1975 [4]. Over the next 40 years, additional school nutrition programs have been added as childhood health and nutrition needs have been identified. Today, the roster of programs that may be offered, depending on district demographics and interest, includes SBP, NSLP, After School Snack Program, Supper Program, Fresh Fruit and Vegetable Program (FFVP), Summer Food Service Program (SFSP), and Special Milk Program. Table 13.1 provides US Department of Agriculture, Food and Nutrition Services (USDA-FNS) participation data from these programs, where available, as of December 2015 (the USDA Farm to School initiative and its 2015 census information will be covered in a separate section).

Program	Participation (2015 data)
School Breakfast Program	14,900,000 meals average per day
National School Lunch Program	30,500,000 meals average per day
Special Milk Program	46,900,000 half-pints per month
Summer Food Service Program	163,700,000 meals per summer
http://www.fns.usda.gov/pd/child	-nutrition-tables

 Table 13.1
 Participation in USDA School Food Programs

Nutrition Standards in School Meals

As health concerns and nutritional problems have changed over the years, the rules and regulations guiding federal school food programs have necessarily evolved as well. Every 5 years, all the USDA child nutrition programs, including all school meals, must be reviewed and Congress must reauthorize their funding [5]. Current school food program regulations and funding were established by the Healthy, Hunger-Free Kids Act of 2010 (HHFKA) [6]. As of August 2016, the next round of Child Nutrition Reauthorization, due in 2015, is still under discussion with many unresolved issues between the US Senate and the House of Representatives versions of legislation.

Healthy, Hunger-Free Kids Act of 2010

The 2010 HHFKA and the resulting USDA regulations have led to a major overhaul of school meal patterns and other guidelines for the school nutrition environment. The legislation passed by Congress and the regulations created by USDA had a dual focus: (1) meeting children's nutritional needs associated with the rise in obesity, based on the Dietary Guidelines for Americans; and (2) on providing needed nutrition for children living in food-insecure homes. The major changes included new nutrition standards for breakfast and lunch meals, new regulations for foods sold outside of meals (Smart Snacks in School), new requirements for Local Wellness Policies, new professional standards for school nutrition staff, and new provisions for high-poverty areas (Community Eligibility Provision, CEP), as well as substantial increases in funding for Farm to School initiatives. The changes are described below with impact and evaluation data, where available.

School Nutrition Program Standards

The scientific foundation for the current National School Lunch Program and School Breakfast Program meal patterns was outlined in a 2009 report from the Institute of Medicine (IOM): School Meals, Building Blocks for Healthy Eating [7]. The IOM recommended multiple changes to align school meals with the Dietary Guidelines for Americans and to address childhood health concerns, including obesity and other risks for chronic diseases like hypertension. The 2010 Healthy, Hunger-Free Kids Act (HHFKA), signed into law by President Obama in December 2010, has also become a centerpiece of the First Lady Michelle Obama's Let's Move campaign [8].

With HHFKA funding, recommendations from the IOM report, and over 300,000 public comments, USDA's Nutrition Standards in the National School Lunch and School Breakfast Programs were released in January 2012 (Table 13.2) [9]. The new school meal patterns followed the IOM recommendations closely and included the following changes:

	Breakfast	meal patter	n	Lunch mea	l pattern	
Grades	K-5	6–8	9–12	K-5	6–8	9–12
Meal pattern	Amount o	of food per v	veek (minin	num per day)		
Fruits (cups)	5(1)	5(1)	5(1)	21/2(1/2)	21/2(1/2)	5(1)
Vegetables (cups)	0	0	0	33/4(3/4)	33/4(3/4)	5(1)
Dark green (cups)	0	0	0	1/2	1/2	1/2
Red/orange (cups)	0	0	0	3⁄4	3⁄4	1 1/4
Beans/peas (cups)	0	0	0	1/2	1/2	1/2
Starchy (cups)	0	0	0	1/2	1/2	1/2
Other (cups)	0	0	0	1/2	1/2	3⁄4
Additional vegetable to reach total (cups)	0	0	0	1	1	1 1/2
Grains (oz/eq)	7(1)	8(1)	9(1)	8(1)	8(1)	10(2)
Meats/meat alternate (oz eq)	0	0	0	8(1)	9(1)	10(2)
Fluid milk (cups)	5(1)	5(1)	5(1)	5(1)	5(1)	5(1)
Other specifications: Daily amount based on the average for a 5-day week						
Min-max calories (kcal)	350-500	400-550	450-600	550-650	600-700	750-850
Saturated fat (% of total calories)	<10	<10	<10	<10	<10	<10
Sodium (mg) Target 1 (2014–2015) ^a	≤540	≤600	≤640	≤1,230	≤1,360	≤1,420
Trans fat		label or mai at per servii		pecifications	must indicate	zero grams of

Table 13.2Nutrition Standards in School Breakfast and National School Lunch Programs – January 2012 andsubsequent revisions

http://www.fns.usda.gov/school-meals/nutrition-standards-school-meals

^aSodium targets 2 and 3 have been established for school years 2017–2018 and 2020–2022. These are under discussion in the 2016 Child Nutrition Reauthorization negotiations. http://www.fns.usda.gov/sites/default/files/ops/HHFKA-Sodium.pdf

- Specific requirements for three grade groupings (K-5, 6–8, and 9–12)
- · Calorie minimums and maximums for breakfast and lunch
- All grain foods must be whole grain-rich by July 1, 2014
- Limitations on grain-based desserts (maximum 2 per week)
- Five new vegetable sub-groups with specific serving sizes by grade group
- New fruit requirements for breakfast and lunch
- Mandatory ¹/₂ cup serving of fruit or vegetable for meal reimbursement
- Milk served must be 1 % or fat-free plain or fat-free flavored
- Limits on sodium, with a 10-year implementation timeline for compliance
- Limitation of saturated fat to <10% of total calories
- Elimination of added trans fats in foods
- Encouragement of scratch cooking techniques and more fresh produce when available

School Meal Improvements and Consumption Patterns

Since the implementation of the HHFKA meal patterns (NSLP in school year 2012–2013, SBP in 2013–2014), national and local studies have documented improvements in both the meals served and in the consumption patterns of students. Turner et al. evaluated school lunches served in 4,630 public US elementary schools, first in 2006–2007 and then in 2013–2014 following implementation of the

HHFKA meal pattern [10]. There were significant overall increases in healthier lunch options, including more vegetables, fresh fruit, whole grains, healthier pizzas, and salad bars as part of lunches served in school year 2013–2014. However, schools with more minority students and low socioeconomic demographics served less fresh fruits and had fewer salad bars. The data showed that many districts had been making gradual changes before the new meal patterns were mandates. Using data from CDC's School Health Policies and Practices Study (SHPSS), Merlo et al. also documented significant improvements in school meals through 2000, 2006, and 2104 [11]. Significantly, more schools offered at least two different non-fried vegetables daily at lunch (79% in 2014 compared to 62% in 2000), and more schools also reported culinary practices to reduce the sodium content of meals (e.g., using other seasonings instead of salt).

An analysis of school lunch meals 16 months before and 15 months after HHFKA meal pattern implementation in Washington state middle and high schools found significant improvements in the nutritional quality of meals chosen by students. This longitudinal study also found a decrease in energy density and a negligible difference in participation (47% before, 46% after) [12]. A cohort study of low-income middle-school students in Connecticut found less waste and greater consumption of fruits, vegetables, and entrées after HHFKA [13]. Comparing 2012–2014, there was a significant increase in the students choosing fruit (from 54 to 66%) while fruit consumption remained high at 74%. Even though the percentage of students choosing a vegetable dropped (from 68 to 52%), students who selected vegetables at nearly 20% more of them. Entrée consumption also increased (from 71 to 84%), while milk consumption stayed the same. The authors concluded that the HHFKA standards and policies appear to have substantially lowered plate waste in these middle-school cafeterias.

In addition to mandated meal patterns, school nutrition programs have begun to use behavioral economics, choice architecture, and chef-enhanced meals to influencing the selection of healthier school meal options. The Smarter Lunchroom Initiative, part of the Food and Brand Lab at Cornell University, has shown that these techniques can have a positive impact on students' food selections at school [14]. In one pilot study, making healthy foods more convenient in school lunchrooms line resulted in 18% more healthy foods selected and a 28% reduction in less healthy food selection [15].

Creative names for healthy foods, like X-Ray Vision Carrots, have also been shown to increase their consumption in elementary schools [16]. Combining choice architecture (Smarter Lunchroom techniques called "smart café") with chef-led changes in the palatability of recipes can also increase healthy food selection and consumption by students. The Massachusetts Modifying Eating and Lifestyles at School Study (MEALS) showed that the smart café intervention alone had no effect, but combining them with chef-enhanced meals increased fruit and vegetable consumption after repeated exposure over 7 months [17]. These authors caution that the positive effects were noted after extended exposure to new foods, suggesting that schools should not abandon healthier options just because they are met with initial resistance.

Approximately 40% of children do not purchase an NSLP meal, and many bring a packed lunch to school. It is important to consider and compare the NSLP meals to alternative meals that students may be consuming at school. While relatively few studies have examined the differences between NSLP meals and those brought from home, a few have shown that lunches brought from home compare unfavorably to the guidelines for school meals. An observational study in Teas schools under the previous NSLP meal pattern showed that meals from homes contained more sodium, as well as desserts, snack chips, and sweetened beverages not allowed in school lunch. They also contained fewer fruits, vegetables, whole grains, and fluid milk [18]. In the Tufts University Project Lunch Box Study, only 27% of packed lunches met at least three of five school lunch standards [19]. An examination of home-packed lunches for kindergarten and pre-K students in rural Virginia found that they were significantly lower in protein, sodium, fiber, vitamin A, and calcium than school lunches. They were also higher in calories, fat, saturated fat, sugar, vitamin C, and iron [20].

Food Insecurity

Hunger and food insecurity remain serious problems for America's school children. In Map the Meal Gap 2015, Feeding America reported that 21.4% of children in the USA – one in four – live in foodinsecure homes [21]. Childhood food insecurity exists in every US county with rates ranging from 6 to 43%. Most of these children are eligible for and regularly participate in free or reduced price meals at school. According to United States Department of Agriculture (USDA) participation data, the number of children receiving free meals has continued to climb, due in part to the Community Eligibility Provision (CEP) of the HHFKA. This option allows high-poverty schools to offer break-fast and lunch at no charge to all students. In 2015, approximately 12 million eligible children ate free or reduced-price lunch [22].

Unfortunately, gaps still exist in access to and participation in federal school feeding programs. The 2016 School Breakfast Scorecard, an annual report by the Food Research and Action Center (FRAC), provided a detailed analysis of participation during the 2014–2015 school year and noted that there is still significant room for improvement. School breakfast participation grew by 4.2% during the year; however, only 54.3% of eligible low-income children participated in school breakfast for every 100 participating in school lunch, leaving a breakfast gap of 45.7% [23]. While some of these children may eat at home, many others may start the school day too hungry to learn. There is also a significant gap in participation for the Summer Food Service Program (SFSP). Realizing that "hunger does not take a summer vacation," the FRAC also tracks participation in SFSP. In summer 2015, there were increases in both the number of SFPS sites and the number of children 2–19 years eating free meals. However, at maximum participation, only 16.2 children participated in SFSP for every 100 low-income children participating in school lunch during the 2013–2014 school year [24].

Childhood Obesity

Discussions in the popular press have often blamed school meals for the rise in childhood obesity rates. The reality, of course, is that school meals represent only a portion of children's intake. Many provisions of HHFKA regulations were designed to control caloric intake at school and to insure that the calories provided are as nutrient-rich as possible [25]. The relationship between the new nutrition standards and childhood body mass index (BMI) has not been studied on a nationwide basis. However, we do know that among youth 2–18 years of age, the prevalence of obesity did not change significantly from 2003–2004 through 2013–2014 [26].

Several studies have indicated positive effects of school nutrition programs on childhood BMI in specific populations. Qian and colleagues used measured BMI from a panel of Arkansas school children participating in the USDA Fresh Fruit and Vegetable Program (FFVP) [27]. The results suggest that FFVP participation can help lower obesity rates, overweight rates, and average BMI z-score. In the first longitudinal study of the effect of school breakfast consumption on BMI, Connecticut researchers found that concerns about a second breakfast at school increasing risk of excessive weight gain were unsupported. In this 2-year study conducted before the implementation of the new HHFKA breakfast pattern, students who regularly consumed breakfasts at school, including double breakfast eaters, were more likely to exhibit a healthy weight trajectory than breakfast "skippers" [28]. An analysis of Breakfast in the Classroom (BIC) in New York City schools over a similar time period agreed: Consuming BIC did not increase BMI and may have had a very small positive effect on rates of obesity [29].

Competitive Foods and Beverages: Smart Snacks in School

The 2010 HHFKA also gave USDA the authority, for the first time, to develop standards for snack foods and beverages served to students during the school day outside school meals. Traditionally called competitive foods, these include à la carte items in cafeterias vending machines, student stores, concession stands, and fundraising events. The rule developed by USDA, Smart Snacks in School, limits low nutrient items and requires that healthier competitive foods and beverages be sold during the school day, with flexibility for local control of fundraising [30]. Foods have specific calorie, sodium, fat, and sugar limits, and beverages include grade limitations for size, calories, and other ingredients.

Implementation of the Smart Snack in School regulations began in 2014 so there has been limited evaluation of their impact. Previous research has shown that removing competitive foods from the cafeteria line can increase NSLP participation, replacing nutrient poor à la carte foods with a nutritionally balanced lunch [31].

A research review of the effect of competitive food and beverage policies, prior to Smart Snacks in School, found that most resulted in positive outcomes in terms of product availability [32]. The authors caution, however, that more research is necessary because the influence on overall student consumption, BMI, and weight outcomes was mixed. A small-scale analysis of Smart Snacks in School after initial implementation in Appalachian Virginia found that only 36.6% of carte and vending machine foods met the standards while most beverages (78.2%) did. A 2015 USDA survey of school food service directors found that transitioning to Smart Snacks in School was not difficult, and they were able to find products. They did cite two main challenges: (1) maintaining food service revenue, and (2) obtaining support from school administrators, staff, and parents. Successful strategies to address these challenges included: research new product options and availability with vendors; involve students in decision making about choices; and involve administrators, staff, teachers, and parents in promoting healthier options [33].

Farm to School Program Initiatives

Since 2000, several popular initiatives have been introduced that may affect all school nutrition programs. These include farm to school, school gardens, sustainability, and culinary education for staff, students, and family. There is no doubt that the programs are popular, growing in number, and often championed by those inside and outside of school programs. There are many case studies, poster presentations, and how-to manuals from initiatives like the National Farm to School Network [34], USDA Food Waste Challenge [35], and Chefs Move to School [36]. The theoretical framework for the impact of these programs on child health and nutrition, first outlined in 2012 [37, 38], is a relatively new area of study. The theory was refined in the 2014 Evaluation for Transformation: A Cross-Sectoral Evaluation Framework for Farm to School [39], which USDA will use as the basis for on-going evaluation of Farm to School grantees and other programs.

The 2015 USDA Farm to School Census demonstrates the rapid growth in this area and its wide acceptance. A few of the notable findings [40]:

- 42% of districts surveyed by USDA say they participate in farm to school activities, which translates to 5,254 districts and 42,587 schools across the USA.
- \$789 million invested in local communities through farm to school purchases. This represents a 105% increase over the first USDA Farm to School Census in school year 2011–2012. Nearly half (47%) of these districts plan to purchase even more local foods in future school years.

- 17,089 salad bars, representing 62% of school districts with farm to school programs that operate salad bars, often stocked with local options. Seventy-eight percent of schools with salad bars increased their purchase of fresh fruits and vegetables.
- 7,101 school gardens, representing an increase of 42 % from the previous census.

Schools with a farm to school program also reported wider benefits, including greater community support for school meals (38%), greater acceptance of HHFKA changes (28%), lower meals costs (21%), increased participation (17%), and reduced food waste (18%).

While these metrics are impressive and hopeful, it is important to note that few of these initiatives have yet to report strong quantitative evidence for positive benefits on food choice, meal consumption, nutrient intake, and health status of students. The existing evidence is largely based on self-reported outcome measures and qualitative benefits. Taylor and Johnson noted the lack of peer-reviewed research and recommended use of validated dietary assessment materials [41]. Similar results were noted in a review of the impact of cooking classes on the food-related preferences, attitudes, and behaviors of school-aged children [42]. A 2015 commentary found many gaps and little evidence for the effective-ness of salad bars on choice, consumption, and waste [43]. A systematic review of more than 40 school gardens studies from the USA, UK, and Australia [44] also found poor quantitative research while noting that qualitative reports were positive. All these reviews point to the need for more rigorous study designs and more consistency in how impacts are measured. Cross sector use of the evaluation framework adopted by USDA will hopefully provide more substantial proof in the future.

Factors Affecting School Nutrition Program Quality and Student Consumption

There are many other factors that can influence both the nutritional quality of school meals and their consumption by students. Here is a brief description of three issues that are important in virtually every school district in the USA.

Reimbursement Rates and School Food Budgets

Since passage of the HHFKA in 2014, the new regulations, along with higher food costs and rising labor expenses, have increased the cost of preparing and serving school meals. The law allowed for an additional \$.06 per meal when districts complied with the mandated changes. However, as directors pointed out, this would not buy a whole grain roll. USDA reimbursement rates vary depending on the level of need and are higher in Alaska and Hawaii. For the lower 48 states, the 2015–2016 lunch reimbursement rates ranged from \$3.13 to \$3.30 for free meals and from \$.35 to \$.43 for paid meals. Breakfast rates range from \$1.66 to \$1.99, depending on the poverty level in a school [45].

In general, school nutrition programs must cover all costs with these reimbursements, including food, labor, benefits, utilities, and transportation of food. Some districts can supplement budgets with catering and sales of à la carte foods (now regulated by the Smart Snacks in School rule). Districts may also receive USDA foods, previously known as commodities; some states subsidize meals in the reduced category and local foods purchases; and a few districts provide extra funding for farm to school budgets. Grant funding is available for special programs and equipment purchases, but directors must be savvy financial managers to meet the mandates under current budgetary constraints. In its 2016 Position Paper on Child Nutrition Reauthorization, the School Nutrition Association has asked Congress to increase the per-meal reimbursement for school breakfast and lunch by 35 cents to

ensure School Food Authorities (SFAs) can afford to meet federal requirements and to provide additional commodity support for breakfast meals [46].

Time to Eat

School nutrition researchers, directors, and managers recognize the connection between giving students enough time to eat lunch and reductions in food waste. They also know that scheduling recess before lunch can reduce waste and improve consumption of school lunch vegetables, entrées, milk, and water [47]. According to an on-going Robert Wood Johnson survey, 40% of schools provide 30 min to eat lunch, while only 21% schedule recess before lunch [48]. A 2015 analysis of lunch times in Seattle (WA) elementary schools found that schools had an average 20 min of official lunch periods, but, on average, students had less than 13 min to eat lunch [49]. Schools with more students participating in free and reduced lunch had less time to each lunch. Consistent with previous research, students with longer lunch times consumed more calories and nutrients than students with shorter lunch times. The survey also found that the issue was not a priority for school administrators, who usually have control over school scheduling.

Local Wellness Policies

Over the past two decades, there has been a concerted effort to make school nutrition part of a more comprehensive approach to school health, starting with the Coordinated School Health (CSH) model, promoted and funded in some states, by the Centers for Disease Control and Prevention (CDC). CSH has now evolved into a Whole School, Whole Community, Whole Child Model [50]. While not all districts use the full CDC model, since 2004, those participating in USDA child nutrition programs have been required to have a written wellness policy with goals for nutrition education, physical activity, and other school-based activities designed to promote student wellness. In other words, schools are required to have policies to promote healthy lifestyles and create healthy campus environments. With the 2010 HHFKA passage, additional requirements were added addressing wellness policy implementation, evaluation, and public wellness policy progress reports [51].

Researchers agree that wellness policies can positively impact school districts if they are written with strong policy language and implemented effectively at the local school level [52]. An evaluation of wellness policy strength in 180 Minnesota districts reported responses from principals and teachers. Stronger wellness policies increased the nutrient density of food and beverage options available through vending in the districts studied. However, secondary schools showed the greatest inconsistency in overall policy implementation [53]. The authors note that the development and implementation of effective wellness policies continue to be a challenge and recommended periodic assessments to insure school level compliance with wellness policies.

Resources for School Food Improvements

For school wishing to improve school meals, there are virtually unlimited resources available through federal and state agencies, national and local non-profits, and agricultural producer groups. A sample of these resources is outlined in Table 13.3.

Organization/agency	Information/services	URL
US Department of Agriculture Food and Nutrition Services	Federal agency administering all school food programs	http://www.fns.usda.gov/school-meals/ child-nutrition-programs
Team Nutrition Healthier US School Challenge: Smarter Lunchrooms	Free materials and resources for all school nutrition programs Voluntary certification program recognizing schools for excellence in nutrition and physical activity	http://www.fns.usda.gov/tn/team-nutrition http://www.fns.usda.gov/hussc/ healthierus-school-challenge-smarter-lunchrooms
US Food Waste Challenge: K-12 schools	Resources for schools to reduce, recover, and recycle food waste on their premises Strategies for recovering wholesome excess food for donation	http://www.usda.gov/oce/foodwaste/resources/K12_ schools.html
Institute of Child Nutrition, University of Mississippi	USDA-funded institute offering applied child nutrition research, training, education, and technical support	http://www.nfsmi.org
School Nutrition Association	School nutrition resources, publications, and conferences for members and non-members Publishes <i>The Journal of Child Nutrition & Management</i>	https://schoolnutrition.org/ https://schoolnutrition.org/jcnm
Academy of Nutrition and Dietetics Kids Eat Right	Large organization of food and nutrition professionals includes School Nutrition Services Dietetic Practice Group (SNS DPG) Initiative of Academy and its Foundation with resources for families and schools	http://www.eatright.org/ http://www.snsdpg.org http://www.eatright.org/resources/for-kids
National Farm to School Network	Network providing information, training, and technical assistance for local food sourcing, gardens, and agricultural education for school systems	http://www.farmtoschool.org/
Pew Charitable Trusts Kids' Safe and Healthful Foods Project	Collaboration between Pew Charitable Trusts and Robert Wood Johnson Foundation offers non-partisan analysis, policy recommendations, and resources for schools	http://www.pewtrusts.org/en/projects/ kids-safe-and-healthful-foods-project
Alliance for a Healthier Generation	Founded by American Heart Association and Clinton Foundation, AHG offers tools, resources, and technical assistance for schools	https://www.healthiergeneration.org
Action for Healthy Kids	AFHK offers grants, tools, and events across the USA, including team activities in some states	http://www.actionforhealthykids.org
Vermont FEED	Farm to school resources, training, and technical assistance Resources for download include <i>New School Cuisine: Nutritious and</i> <i>Seasonal Recipes for School Cooks by School Cooks</i> , plus numerous Farm to School guides	http://vtfeed.org
The Lunch Box: Tools for School Food Change Fuel Up to Play 60	Recipes, resources, and tools from the Chef Ann Foundation In-school nutrition and physical activity program launched by National Dairy Council and NFL, in collaboration with USDA, to help	http://www.thelunchbox.org https://www.fueluptoplay60.com
	encourage today's youth to lead healthier lives Grants, resources, and technical assistance for school programs to enhance nutrition and activity programming	

260

Trends in School Nutrition Programs

Participation in school food programs is affected by numerous factors, among them the economy, nutrition standards, and public perceptions. According to the Food Research and Action Center (FRAC) 2016 report on trends in participation, shifts in the categories of school meal payments predated the 2010 Healthy, Hunger-Free Kids Act (HHFKA) [54]. Participation rates had been increasing among the lowincome students eligible for free school meals and declining among children in the reduced-price and paid categories. These trends have continued with the implementation of HHFKA nutrition standards. While it is important to maximize participation by low-income, food-insecure children, the overall financial viability of nutrition programs in schools depends on strong participation by paid customers. The FRAC report describes multiple ways to enhance participation by all students, recommending ongoing support for science-based nutrition standards regulating both meals and competitive foods.

Conclusion

Today's school food programs are based on the latest nutrition science, the Dietary Guidelines for Americans. As planned, prepared, and served to millions of children across the USA every day, these meals are nutritionally sound. However, the most critical part of the process is not getting the food onto the tray. It is making certain that the nutritious meals are consumed by students rather than being thrown into trash cans. School nutrition professionals are facing the myriad challenges of changing campus food environments and introducing unfamiliar, healthier foods to their customers. They are using multiple, innovative strategies to serve healthier meals to students – often without the support of administrators and families. In thousands of school kitchens and dining areas, they are doing an extraordinary job and helping to build a healthier generation – one well-balanced meal at a time.

References

- 1. Smedley E. The school lunch; its organization and management in Philadelphia. Media, Pennsylvania: Innes & Sons; 1920.
- 2. Hinrichs P. The effects of the national school lunch program on education and health. J Policy Anal Manage. 2010(29):479–505.
- 3. Martin M. Understanding the past; Shaping the future. Am J Lifestyle Med. 2013:1-4.
- US Department of Agriculture Food and Nutrition Service. The school breakfast program. September 2013. http:// www.fns.usda.gov/sites/default/files/SBPfactsheet.pdf. Accessed 15 Mar 2016.
- Food Research and Action Center. Child nutrition reauthorization. http://frac.org/pdf/cnr_primer.pdf. Accessed 15 Mar 2016.
- US Department of Agriculture Food and Nutrition Service. Healthy hunger-free kids act, 2010. http://www.fns. usda.gov/school-meals/healthy-hunger-free-kids-act. Accessed 15 Mar 2016.
- Stallings VA, Suitor CW, Taylor CL, editors. School meals: building blocks for healthy children. National Academies Press; Washington, DC 2010.
- Let's Move! Program developed by First Lady Michelle Obama to solve the epidemic of childhood obesity within a generation. http://www.letsmove.gov/. Accessed 15 Mar 2016.
- Nutrition Standards in the National School Lunch and School Breakfast Programs. Department of Agriculture Food and Nutrition Service. Federal Register. Thursday, January 26, 2012;77(17). http://www.gpo.gov/fdsys/pkg/ FR-2012-01-26/pdf/2012-1010.pdf. Accessed 15 Mar 15, 2016.
- Turner L, Ohri-Vachaspati P, Powell L, Chaloupka FJ. Improvements and Disparities in Types of Foods and Milk Beverages Offered in Elementary School Lunches, 2006–2007 to 2013–2014. Prev Chronic Dis. 2016;13:1–9.
- Merlo C, Brener N, Kann L, McManus T, Harris D, Mugavero K. School-level practices to increase availability of fruits, vegetables, and whole grains, and reduce sodium in school meals: United States, 2000, 2006, and 2014. MMWR Morb Mortal Wkly Rep. 2015;64(33):905–8.

- Johnson DB, Podrabsky M, Rocha A, Otten JJ. Effect of the healthy hunger-free kids act on the nutritional quality of meals selected by atudents and school lunch participation rates. JAMA Pediatr. 2016;170(1):e153918.
- Schwartz MB, Henderson KE, Read M, Danna N, Ickovics JR. New school meal regulations increase fruit consumption and do not increase total plate waste. Child Obes. 2015;11(3):242–7.
- Hanks AS, Just DR, Wansink B. Smarter lunchrooms can address new school lunchroom guidelines and childhood obesity. J Pediatr. 2013;162(4):867–9.
- Hanks AS, Just DR, Smith LE, Wansink B. Healthy convenience: nudging students toward healthier choices in the lunchroom. J Public Health. 2012;34(3):370–6.
- Wansink B, Just DR, Payne CR, Klinger MZ. Attractive names sustain increased vegetable intake in schools. Prev Med. 2012;55(4):330–2.
- Cohen JF, Richardson SA, Cluggish SA, Parker E, Catalano PJ, Rimm EB. Effects of choice architecture and chefenhanced meals on the selection and consumption of healthier school foods: a randomized clinical trial. JAMA Pediatr. 2015;169(5):431–7.
- Caruso ML, Cullen KW. Quality and cost of student lunches brought from home. JAMA Pediatr. 2015;169(1):86–90.
- Hubbard KL, Must A, Eliasziw M, Folta SC, Goldberg J. What's in children's backpacks: foods brought from home. J Acad Nutr Diet. 2014;114(9):1424–31.
- 20. Farris AR, Misyak S, Duffey KJ, Davis GC, Hosig K, Atzaba-Poria N, McFerren MM, Serrano EL. Nutritional comparison of packed and school lunches in pre-kindergarten and kindergarten children following the implementation of the 2012–2013 national school lunch program standards. J Nutr Educ Behav. 2014;46(6):621–6.
- Gundersen, C., Engelhard, E., America, F., Satoh, A., & Waxman, E. (2011). Map the Meal Gap 2014: Technical Brief.http://www.feedingamerica.org/hunger-in-america/our-research/map-the-meal-gap/2012/2012-map-the-mealgap-tech-brief.pdf. Accessed 15 Mar 2016.
- Child Nutrition Tables, Department of Agriculture Food and Nutrition Service. http://www.fns.usda.gov/pd/childnutrition-tables. Accessed 15 Mar 2016.
- Breakfast Scorecard, Food Research and Action Center. 2016. http://frac.org/pdf/School_Breakfast_Scorecard_ SY_2014_2015.pdf. Accessed 15 Mar 2016.
- Hunger Doesn't Take a Vacation: Summer Nutrition Status Report, Food Research and Action Center. 2015. http:// frac.org/pdf/2015_summer_nutrition_report.pdf. Accessed 15 Mar 2016.
- HHFKA Implementation Research Brief: Childhood Obesity, Department of Agriculture Food and Nutrition Service. 2016. http://www.fns.usda.gov/sites/default/files/ops/HHFKA-ChildhoodObesity.pdf. Accessed 15 Mar 2016.
- Ogden CL, Carroll MD, Fryar CD, Flegal KM. Prevalence of obesity among adults and youth: United States, 2011–2014. NCHS Data Brief. 2015;219:1–8.
- Qian Y, Nayga RM, Thomsen MR, Rouse HL. The Effect of the Fresh Fruit and Vegetable Program on Childhood Obesity. Applied Economic Perspectives and Policy. 2015 Jun 20:ppv017.
- Wang S, Schwartz MB, Shebl FM, Read M, Henderson KE, Ickovics JR. School breakfast and body mass index: a longitudinal observational study of middle school students. Pediatr Obes. 2016 Mar 17. doi: 10.1111/ijpo.12127.
- 29. Corcoran SP, Elbel B, Schwartz AE. The effect of breakfast in the classroom on obesity and academic performance: evidence from New York City. In: Health & Healthcare in America: from economics to policy. Ashecon; Washington, DC 2014
- Tools for Schools: Focusing on Smart Snacks, Department of Agriculture Food and Nutrition Service. http://www. fns.usda.gov/healthierschoolday/tools-schools-focusing-smart-snacks. Accessed 15 Mar 2016.
- Bhatia R, Jones P, Reicker Z. Competitive foods, discrimination, and participation in the National School Lunch Program. Am J Public Health. 2011;101(8):1380–6.
- Chriqui JF, Pickel M, Story M. Influence of school competitive food and beverage policies on obesity, consumption, and availability: a systematic review. JAMA Pediatr. 2014;168(3):279–86.
- HHFKA Implementation Research Brief: Smart Snacks, Department of Agriculture Food and Nutrition Service. 2016. http://www.fns.usda.gov/sites/default/files/ops/HHFKA-SmartSnacks.pdf. Accessed 15 Mar 2016.
- 34. National Farm to School Network. http://www.farmtoschool.org. Accessed 15 Mar 2016.
- Food Waste Challenge K-12, Department of Agriculture Food and Nutrition Service. 2016. http://www.usda.gov/ oce/foodwaste/resources/K12_schools.html. Accessed 15 Mar 2016.
- 36. Chefs Move to School: A Let's Move Initiative. http://www.chefsmovetoschools.org/. Accessed 15 Mar 2016.
- 37. Joshi A, Ratcliffe MM. Causal pathways linking farm to school to childhood obesity prevention. Child Obes (Formerly Obesity and Weight Management). 2012;8(4):305–14.
- Ratcliffe MM. A sample theory-based logic model to improve program development, implementation, and sustainability of Farm to School programs. Child Obes (Formerly Obesity and Weight Management). 2012;8(4):315–22.

- Joshi A, Henderson T, Ratcliffe MM, Feenstra G. Evaluation for transformation: a cross-sectoral evaluation framework for farm to school, national farm to school network. 2014. http://www.farmtoschool.org/Resources/ Framework-08-25-14_web.pdf. Accessed 15 Mar 2016.
- Farm to School Census, Department of Agriculture Food and Nutrition Service. 2016. https://farmtoschoolcensus. fns.usda.gov. Accessed 1 Apr 2016.
- Taylor JC, Johnson RK. Farm to School as a strategy to increase children's fruit and vegetable consumption in the United States: research and recommendations. Nutr Bull. 2013;38(1):70–9.
- 42. Hersch D, Perdue L, Ambroz T, Boucher JL. Peer reviewed: the impact of cooking classes on food-related preferences, attitudes, and behaviors of school-aged children: a systematic review of the evidence, 2003–2014. Prev Chronic Dis. 2014;11.
- Adams MA, Bruening M, Ohri-Vachaspati P. Use of salad bars in schools to increase fruit and vegetable consumption: where's the evidence? J Acad Nutr Diet. 2015;115(8):1233–6.
- 44. Ohly H, Gentry S, Wigglesworth R, Bethel A, Lovell R, Garside R. A systematic review of the health and well-being impacts of school gardening: synthesis of quantitative and qualitative evidence. BMC Public Health. 2016;16(1):1.
- 45. School Programs Meal Snack and Milk Payments to States and School Food Authorities, Department of Agriculture Food and Nutrition Service. 2015. http://www.fns.usda.gov/sites/default/files/cn/NAPS15-16nslpchart.pdf. Accessed 15 Mar 2016.
- School Nutrition Position Paper, School Nutrition Association. 2016. https://schoolnutrition.org/positionpaper/. Accessed 15 Mar 2016.
- 47. Price J, Just DR. Lunch, recess and nutrition: responding to time incentives in the cafeteria. Prev Med. 2015;71:27–30.
- Turner L, Eliason M, Sandoval A, Chaloupka FJ. Most US public elementary schools provide students only minimal time to eat lunch. BTG Research Brief. 2014.
- 49. Lunch Time at School: How Much Time is Enough? University of Washington, School of Public Health. 2015. http://depts.washington.edu/nutr/wordpress/wp-content/uploads/2015/07/Time-For-Lunch-FINAL_NUTR531winter2015-1.pdf. Accessed 15 Mar 2016.
- Lewallen TC, Hunt H, Potts-Datema W, Zaza S, Giles W. The Whole School, Whole Community, Whole Child model: a new approach for improving educational attainment and healthy development for students. J Sch Health. 2015;85(11):729–39.
- Local School Wellness Policy Requirements, Department of Agriculture Food and Nutrition Service. 1 Sept 2015. http://www.fns.usda.gov/tn/local-school-wellness-policy-requirements. Accessed 15 Mar 2016.
- Schwartz MB, Henderson KE, Falbe J, Novak SA, Wharton CM, Long MW, O'Connell ML, Fiore SS. Strength and comprehensiveness of district school wellness policies predict policy implementation at the school level. J Sch Health. 2012;82(6):262–7.
- Larson N, Davey C, Hoffman P, Kubik MY, Nanney MS. District wellness policies and school-level practices in Minnesota, USA. Public Health Nutr. 2016;19(01):26–35.
- National School Lunch Program: Trends and Factors Affecting Student Participation, Food Research and Action Center. 2016. http://frac.org/pdf/national_school_lunch_report_2015.pdf. Accessed 15 Mar 2016.

Part IV Nutrition in Athletes and Physically Active Adults

Chapter 14 Nutritional Considerations for Young Athletes

Rebecca Boulos and Anne-Marie Davee

Key Points

- Physical and social changes and variations in the timing of adolescent maturation have implications for the health of the growing athlete.
- Fluid and food consumption patterns have physiological and biological implications, and can serve to either impair or improve adolescents' athletic performance.
- Dietary intakes and habits that minimize unhealthful weight gain, maximize athletic performance, and promote lifelong health behaviors.

Keywords Adolescent athlete • Youth nutrition • Youth sports • Youth healthy diet

Introduction

Adolescence is a time marked by significant biological, physical, psychological, and behavioral growth; therefore, understanding the types of dietary intakes and habits that minimize unhealthful weight gain, maximize performance, and promote the development of positive lifelong health behaviors is of critical importance. Recent estimates suggest 60 million children ages 6–18 years participate in some form of organized sport in the USA, and 44 million participate in more than one sport [1]. However, research shows that even in active sports, such as soccer, the average participant accumulates only 17 min of moderate-to-vigorous physical activity per 50 min of game time [2]. Research also suggests the foods and beverages marketed in sports venues, sold at concession stands, and used for sports fundraising are commonly unhealthy, with few healthful options available [3]. The marketing and sales of unhealthy foods and beverages in the youth sport environment has the potential to undermine the possible, and typically minimal, physical activity benefits of participating in youth sports. This chapter will provide the latest research related to active adolescents' (1) dietary needs; (2)

A.-M. Davee, MS, RD, LD University of New England, Portland, ME, USA

R. Boulos, MPH, PhD (⊠) University of New England School of Community and Population Health, Portland, ME, USA e-mail: rboulos@une.edu

beverage consumption patterns; (3) use of supplements and ergogenic aids; and (4) behavioral techniques to promote healthful dietary patterns. Lastly, this section will provide practical information for how health practitioners, coaches, and parents can support a healthful environment for young athletes.

Dietary Needs for Young Athletes

During adolescence, youth undergo rapid changes in height and weight, hormonal composition, secondary sexual characteristics, and brain development. This period of development can be conceptualized as occurring in three distinct stages: early adolescence (ages 11–14 years), middle adolescence (ages 15–17 years), and late adolescence (18–21 years) [4]. Different phases of development occur within each of these time points. Early adolescence is marked by the onset of physiological and physical changes related to puberty (e.g., menarche, gains in height and weight). For example, during their peak 1-year growth spurt, males can gain an average of 4.1 inches and females of 3.5 inches [5]. Females tend to experience this change before males, during the ages of 10–13 years, versus 12–15 years for males [6]. This growth spurt is especially important because it has been shown to predict 15% of desirable adult height and 50% of desirable adult weight [7, 8]. In terms of body composition, females tend to increase both their fat and muscle mass as they gain weight, whereas males tend to gain mostly lean mass. Both sexes experience gains in bone mass; in fact, 85–90% of adult bone mass is accrued by the end of the adolescent years [9, 10].

During middle adolescence, teens begin to increase their social and psychological autonomy, puberty ends, and growth slows for females and while continuing for males. Throughout this stage, adolescents are learning how to balance their developing autonomy and self-identity with peer group affiliation [11]. The quality of these relationships has been linked with physical and mental health in adulthood [12]. For this reason, it is particularly important to encourage adolescents to foster positive peer relationships. Lastly, in late adolescence, females' growth and development is usually complete while males are still experiencing growth in body mass. By late adolescence, both genders are typically more independent, have developed a self-identity, and are more future-oriented, with increased concern about life decisions, such as college or career choices.

Collectively, these physical and social changes, and variations in the timing of maturation, have implications for the growing young athlete. For example, during different stages of adolescence, youth may experience increased insulin resistance and variations in physiological responses to exercise, such as changes to glycolytic and metabolic rates, and higher fat oxidation levels [13]. These changes impact how adolescents' bodies use the nutrients they consume, both during and after physical activity. For example, carbohydrates are stored in the body as glycogen; however, adolescents' glycolytic capacity is still developing (only 0.4–0.5% of body weight) [14], which means their glycogen stores are 50–60% less than adults, and they have lower levels of circulating lactate [15]. Taken together, this means the benefits of "carb-loading" and other similar training tactics may not have the same effect in adolescents, as they rely more on aerobic metabolism [15].

Accordingly, adequate consumption of nutrient-dense foods including high-quality protein, complex carbohydrates, and unsaturated fats, as well as vitamins and minerals such as vitamin D, calcium, and iron, is especially important. Conversely, high intakes of some micronutrients, such as sodium, can adversely influence healthy development. Teaching youth how to adopt health-promoting behaviors during this stage will continue to benefit them as they progress into adulthood. To provide a more comprehensive context for how to support positive growth and development of youth athletes through the practice of good nutrition, this section focuses on adolescents' dietary needs, including recommended total calorie intake, macronutrient intake, and important micronutrient considerations.

Calorie Needs

Since adolescents experience physical growth at different rates and ages, setting energy requirements for this population is challenging. Calorie intake ranges are based on gender and adolescent stage (i.e., age), within the context of three different levels of activity: sedentary, moderately active, and active (Table 14.1) [16]. While these reference values are a useful guide, this limitation presents a challenge when trying to determine how many calories an adolescent of a given maturation stage needs. Active adolescents' energy needs will vary depending on the duration, intensity, and frequency of their sport; maturation stage; and dietary patterns and/or restrictions. Therefore, an additional consideration is calculating how many more calories are needed to compensate for training to meet needs above and beyond adolescents' natural growth and development, given their maturation stage instead of age, and for their level of physical activity that is more vigorous than the "Active" category. Using athletes' training intensity and duration, as well as fitness level, height, and weight as guides for calculating energy needs is a useful strategy for assuring they are consuming a sufficient amount of calories.

Macronutrients

There are three macronutrients: carbohydrates, fats, and protein. They are called macronutrients because of the larger quantities required by our bodies, and because they are our only sources of energy. This section will discuss these three nutrients in more detail, including recommended intakes, roles in the body, food sources, and composition in the diet of youth.

Carbohydrates

Carbohydrates, or more specifically glucose, are our brain's and muscles' primary energy source. The recommended intake of carbohydrates for adolescents is 45–65% of total calorie intake; this is equivalent to a Recommended Dietary Allowance (RDA) of 130 g/day and represents the amount of glucose needed for brain metabolism. While most youth consume sufficient carbohydrates, dietary sources tend to come from refined or processed food products (such as simple sugars like candy or sugar-sweetened beverages, SSBs) and fewer from complex carbohydrates, such as whole grains, legumes, fruits, and vegetables. Using National Health and Nutrition Examination Survey (NHANES) data (2011–2012), Mozaffarian and colleagues (2016) report that SSB consumption was high among

Table 14.1	Calorie estimates of adolescents based on gender and activity level	
-------------------	---	--

Gender	Adolescent stage	Sedentary	Moderately active	Active
Female	Early (9-13 years)	1,400-1,600	1,600-2,000	1,800-2,200
	Mid (14-18 years)	1,800	2,000	2,400
	Late (19-30 years)	1,800-2,000	2,000-2,200	2,400
Male	Early (9-13 years)	1,600-2,000	1,800-2,200	2,000-2,600
	Mid (14-18 years)	2,000-2,400	2,400-2,800	2,800-3,200
	Late (19-30 years)	2,400-2,600	2,600-2,800	3,000

Sedentary: Typical day-to-day activities

Moderately active: Walks 1.5–3.0 miles/day at 3–4 miles/h, in addition to typical day-to-day needs Active: Walks 3+ miles/day at a 3–4 miles/h pace, in addition to typical day-to-day needs

youth and increased with age: ranging from 6 to 8 servings/week among youth 5–9 years of age to 12–14 servings/week for youth 15–19 years. Conversely, this same analysis found that average fruit consumption was low (between 3 and 14% of youth of different genders and age groups meeting recommended ≥ 2 cups/day) and decreased as children aged. Youth ages 5–9 years ate 1.7–1.9 servings/day (males and females, respectively), youth 10–14 years ate 1.4 servings/day, and youth 15–19 years ate 0.8–1.3 servings/day (males and females, respectively). Vegetable consumption was also low, ranging from 1.1 to 1.5 servings/day, and fewer than 1.5% of children met the recommended \geq 2.5 cups/day [17]. Complex carbohydrates, such as whole grains, fruits, vegetables, and low-fat dairy products, should be strongly encouraged in adolescence.

Fiber

Fibers are indigestible parts of plants. Consuming a sufficient amount of fiber is essential for several reasons: Fiber supports healthy GI function (both by promoting excretion and slowing digestion to encourage maximal nutrient absorption); it provides a feeling of fullness, which can limit overeating; fiber can be metabolized by gut bacteria to synthesize B-vitamins; and it binds to bile, which can lower cholesterol. In fact, too little fiber can lead to digestive problems, excessive calorie intake, and peaks in blood sugar levels. Too much fiber, on the other hand, can lead to GI distress and micronutrient deficiencies (as fibers bind to minerals such as calcium and iron).

The adequate intake (AI) for fiber is 31 g/day for males ages 9–13, and 38 g/day for males ages 14–18 years. For females ages 9–18 years, the AI is 26 g/day. Using NHANES 2009–2010 data, Reicks and colleagues (2014) found youth ages 2–18 years consumed only 14 g of fiber/day [18]. Good sources of fiber include beans, whole grains (such as whole oats and whole wheat bread), as well as fruits and vegetables. These sources of fiber provide 2–8 g per serving.

Added Sugar

While sugars can be a source of quick energy that are naturally occurring in fruits, vegetables, whole grains, and dairy products, added sugars in the diet are a substantial contributor to the nearly 600 "empty" calories (in the form of solid fats, added sugars, alcohol) consumed by youth [19]. Using 2005–2010 NHANES data (n=4,047 youth), Zhang and colleagues found, on average, 16% of calories youth ages 12–19 years consumed were from added sugar; 88% of youth consumed usual intakes of added sugar $\geq 10\%$ of total calories, and 6% consumed usual intakes $\geq 25\%$ of total calories. Overall, youth consumed an average of 87.4 g/day or 350 cal, of added sugars [20]. As a point of reference, for an active adult male, which is the highest reference value for intake, the upper limit for added sugar consumption is 36 g/day, or 144 cal [21]. Excess sugar consumption has been linked with metabolic syndrome, weight gain, dental carries, type II diabetes, micronutrient deficiencies, and a host of other adverse health effects [20, 22–24].

To address the need for guidance on how much added sugar adolescents should be consuming, the American Heart Association (AHA) published dietary recommendations (2005), which offered the following: "sweetened beverages and naturally sweet beverages, such as fruit juice, should be limited to ...8 to 12 oz per day for children 7–18 years old [25]. In a large, nationally representative sample of 21,995 youth, the percentages of daily soft drink consumption (12 oz serving) were 51% of 8th graders, 46% of 10th graders, and 43% of 12th graders [26]". Parents, coaches, and practitioners should seek to limit intakes of added sugars in adolescents' diets, including sources such as SSBs (including juice and sports drinks), sugar-sweetened cereals, candy, and baked goods.

Fats

Fats play an important role in human health; they protect internal organs and bones, are needed for vitamin absorption and storage, help to regulate body temperature, and are used in the formation of cells' membrane and structure. There are different kinds of fats: unsaturated fats (including poly- and mono-unsaturated), saturated, and *trans* fats (each of these prefixes refers to its chemical structure) as well as cholesterol. The recommended intake of fat for adolescents is between 25 and 35% of total calories (with <10% from saturated and 0 g *trans* fat). Too much dietary fat can lead to an increase in body weight and elevated cholesterol levels. Too little fat can impair vitamin absorption, metabolism, and storage; increase risk of fracture after a fall; and impair body temperature regulation (e.g., increase perceived extremes of hot and cold). It is important for adolescents to pay attention to the kind of fat they are concuming: Uncertained fats are preferable given their role in payrological and visual daval

they are consuming: Unsaturated fats are preferable given their role in neurological and visual development, and in the fluidity and permeability of cell walls. Saturated fat, on the other hand, has been shown to increase low-density lipoprotein (LDL; "bad cholesterol"), and *trans* fats have been shown to concurrently increase LDL and decrease high-density lipoprotein (HDL; "good cholesterol"). Good sources of dietary fat include fatty fish, such as salmon and sardines, nuts, avocado, and oils (e.g., canola, olive, and coconut).

Most adolescents are meeting the recommended total fat intake. Using NHANES data, Ervin and Ogden (2013) found that, on average, youth consumed one-third of their calories from fat [27]. However, intakes of saturated fat are higher than the recommendation of <10% of total calories: Using NHANES data (2011–2012), Mozaffarian and colleagues (2016) report that youth consumed approximately 11% of calories from saturated fat [17].

Protein

Protein is needed in the body for multiple physiological and biological processes, including antibody synthesis, tissue repair, and cell structures. The recommended protein intake ranges from 1.2 to 2.0 g/ kg of body weight for the average adolescent, based on gender, age, height, weight, and physical activity [28]. This amount reflects the quantity needed to maintain adequate nitrogen balance to maintain lean body mass. However, the evidence is inconclusive (and research ongoing) about how much protein the active adolescent actually needs. Research does suggest that with increased physical activity, protein utilization efficiency improves [29, 30], which means greater intakes are likely unnecessary (and not evidence-based), and needs may be met from dietary intake alone. Therefore, any substantive and intentional increase in protein intake – especially from supplements – should be preceded by a meeting with a registered dietitian. More specifically, while insufficient intakes of protein can lead to an irregular heartbeat, impairments in blood clotting and tissue repair, and poor immune health, high intakes of protein can lead to renal damage and increased fat mass. There is an additional concern that too much protein can lead to increased urinary calcium excretion, and thus adversely influence bone health; however, the research findings on this are mixed, and the effects seem to be influenced by the interaction of multiple variables including source (food or supplement) and intake amounts for both protein and calcium [31].

Typically, adolescents consume a sufficient amount of protein. Using NHANES data (2009–2010), Ervin and Ogden (2013) found that males and females, ages 2 to 19 years, were consuming 14.7% and 14.3%, respectively, of their total calories from protein [27]. Also using NHANES data, Keast and colleagues (2013) found that the primary protein sources for youth ages 2–18 years were milk (13.2%), poultry (12.8%), and beef (11.5%). Eggs and shellfish ranked lowest at 2.3% and 2.1%, respectively [32].

Micronutrients

Micronutrients are nutrients our body requires in much smaller amounts (measured in µg and mg instead of grams) and are non-caloric. Vitamins are organic substances that our body uses for fundamental and essential physiological processes, such as cholesterol synthesis, vision, immune function, and blood clotting. Minerals are inorganic compounds that are also important for biological processes, including maintaining fluid balance, blood pressure and pH balance, hemoglobin synthesis, muscle contractions, and bone structure. This next section will discuss key micronutrients for the active adolescent: vitamin D, iron, calcium, and sodium.

Vitamin D

Vitamin D is a fat-soluble vitamin that is essential for calcium absorption, cholesterol synthesis, cell differentiation, and insulin secretion. We are able to absorb vitamin D from sunlight, and can also consume it in our diet. Some foods, such as egg yolks, mushrooms, and fatty fish (e.g., salmon, herring, mackerel), contain naturally occurring vitamin D; fortified milk, cereals, and orange juice also contain vitamin D. The vitamin D RDA for adolescents is 600 IU (15 μ g) [33]. Currently, the definition for deficiency is ≤ 10 ng/mL; insufficiency is between 11 and 20 ng/mL; and optimal is >20 ng/mL [34].

There is a high prevalence of both vitamin D insufficiency and deficiency among youth in the USA. Using NHANES data, Karalius and colleagues (2014) found that 10% of US children ages 6–18 years fell in the insufficiency range [35]. Low levels of vitamin D can increase risk of fracture, impair athletic performance, lead to a softening of the bones, and osteoporosis later in life; it can also cause increased muscle pain and weakness. Other populations at risk for deficiency include overweight and obese individuals (as excess body fat sequesters vitamin D, making it less available to the body); vegans and vegetarians; and individuals with dark skin. In order to absorb enough vitamin D from the sun, it is recommended to expose face, arms, and legs, without sunscreen, twice/week between the hours of 10:00 am and 3:00 pm for 5–30 min, depending on the season, latitude, and skin color, as SPFs of 8 and greater reduce skin synthesis by 95% [36, 37].

Iron

Given their rapid growth and development, adolescents need a sufficient amount of iron. The RDA is 8 mg for both males and females ages 9–13 years; this recommendation increases to 11 mg for males and 15 mg for females ages 14–18 years. Iron is used in the body for hemoglobin synthesis, cell growth, and oxygen transport. There are two different kinds of iron: heme and non-heme. Heme iron is bound in hemoglobin, so it is found in animal products, such as fish and poultry; this form is the most bioavailable. Sources of non-heme iron include fortified cereals, dark green leafy vegetables (e.g., spinach, kale), and nuts. Multiple inhibitors such as polyphenols, phytates, and fiber limit the bioavailability of non-heme iron. Therefore, vegetarians and vegans are at risk for iron deficiency given the limited bioavailability of non-heme iron. To improve bioavailability, non-heme food sources should be consumed with vitamin C-rich food sources, such as red bell peppers, strawberries, and citrus fruits to maximize its absorption. Consistently low intakes of iron can lead to chronic fatigue and decreased immune function, as well as impaired aerobic capacity and athletic performance. It has been recommended that populations at risk for deficiency take a low-dose iron supplement during intense training and with guidance from a physician and/or dietitian [38].

Calcium

Calcium performs multiple roles in the body. It is essential for muscle contractions, nerve transmissions, blood vessel volume regulation, and bone health; bone mineral content (BMC) is about 80% calcium [39]. Since blood levels of calcium are homeostatic, insufficient intakes from the diet can lead to the leeching of calcium from the bone, resulting in soft bones and elevated risk for stress fractures as well as muscle cramps and arrhythmias. For growing youth, calcium consumption is particularly important. Total body BMC more than doubles in children between the ages of 8 and 15 years, with peak gains attained between the ages of 12.5 (girls) and 14.0 (boys) years [40], resulting in 85–90% of total bone accrual by the age of 18 years for females and age 20 years for males. Martin and colleagues found in a group of 228 youth that maximal calcium accretion occurs at rates of 282 mg/day for males at age 13.3 years and 212 mg/day for females at age 11.4 years [41].

Given the critical role calcium plays in laying down adequate bone, the RDA is 1,300 mg/day (males and females ages 9–18 years). However, most youth are not consuming a sufficient amount of calcium. Approximately 50% of males and 75% of females ages 9–18 years of age are not meeting recommended calcium intakes, based on the estimated average requirement (1,100 mg) [42]. While activities that increase skeletal demand, such as running and gymnastics, also positively contribute to BMC [43], if adolescent athletes do not consume a sufficient amount of calcium, they increase their risk for injury and impaired performance.

Aside from dairy products such as milk, yogurt, and cheese, other good sources of calcium include fortified sources such as orange juice and soy milk as well as dark green leafy vegetables (e.g., spinach and kale). Calcium absorption can be maximized by consuming it concurrently with a source of vitamin D (such as eggs or salmon), and waiting an hour between ingesting a calcium-rich source and an iron-rich source, as they compete for absorption. In fact, Malczewska and colleagues found that iron-deficient female endurance athletes had greater calcium levels than their non-athletic peers with adequate iron status [44]. This underscores the importance of adequate intakes of all micronutrients among adolescent athletes in order to avoid deficiencies, and for those with dietary restrictions, such as vegans, who are at risk for both iron and calcium deficiencies.

Other Micronutrients

While the nutrients outlined above are critical for active youth, other micronutrients are also important for growth and development. Tables 14.2 and 14.3 below provide an overview of intake recommendations for all the vitamins and minerals.

Beverages

Our body is about 60% water, and our brain is 75–85% water. Water is needed for the digestion, absorption, metabolism, and transportation of nutrients; regulation of body temperature, physiological/biological reactions, and electrolyte balance; and adequate lubrication of joints. Being well-hydrated boosts cognitive and physical performance, helps protect against illness, and encourages the maximal use of dietary nutrients. Ensuring adequate hydration is essential, as water losses of as little as 1-2% can cause fatigue and negatively impact performance [45]. Water is lost through respiration, gastrointestinal excretions, and sweat; electrolytes are also lost in perspiration. Heat stress from being physically active in warm climates can further impair performance and increase the risk of heat-related illness if an individual is insufficiently hydrated [28, 46]. The body's ability to regulate temperature, maximize cardio-respiratory function, and otherwise adapt to increasing physical

	Aª	Dª	E ^b	Ka	C^{\flat}	Thia- min ^ь	Ribo- flavin ^b	Niacin ^b	B6 ^b	Folate ^a	B12 ^a	Panto. acid ^b	Biotin ^a	Choline ^b
Female														
9–13	600	15	11	60	45	0.9	0.9	12	1.0	300	1.8	4	20	375
14–18	700	15	15	75	65	1.0	1.0	14	1.3	400	2.4	5	25	550
Male														
9–13	600	15	11	60	45	0.9	0.9	12	1.0	300	1.8	4	20	375
14–18	900	15	15	75	75	1.2	1.3	16	1.2	400	2.4	5	25	400

Table 14.2 Vitamin needs of adolescents

^aµg/d ^bmg/d

Table 14.3 Mineral needs of adolescents

	Ca ^b	Cr ^a	Cu ^a	F ^{-b}	I ^a	Fe ^b	Mg^{b}	Mn ^b	Mo ^a	\mathbf{P}^{b}	Se ^a	Zn ^b	Kc	NaCl ^c	Clc
Female															
9–13	1,300	21	700	2	120	8	240	1.6	34	1,250	40	8	4.5	1.5	2.3
14-18	1,300	24	890	3	150	15	360	1.6	43	1.250	55	9	4.7	1.5	2.3
Male															
9–13	1,300	25	700	2	120	8	240	1.9	34	1,250	40	8	4.5	1.5	2.3
14-18	1,300	35	890	3	150	11	410	2.2	43	1,250	55	11	4.7	1.5	2.3

^bmg/d

demands are largely predicted by hydration status, and are impaired when athletes are not sufficiently hydrated [47].

The Institute of Medicine (IOM's) AI for water is 2,400 mL/day for males ages 9–13 years and 2,100 mL/day for females ages 9–13 years; 3,300 mL/day for males ages 14–18 years and 2,300 mL/ day for females ages 14–18 years [48]. Using NHANES (2009–2010) data (n=4,766), Drewnowski and colleagues [49] found that youth ages 4–13 years consumed 431.0±13.1 mL of water; younger children (4–8 years) consumed less than older children (9–13 years: 364.9±13.5 mL vs. 496.1±19.4 mL, respectively). There were no differences in intake by gender. Overall, 75% of children ages 4–8 years, 83% of females ages 9–13 years, and 85% of males ages 9–13 years did not meet recommended water intakes [49]. Kenney and colleagues [50] looked more specifically at the beverages youth were consuming, and found that among children 6–19 years, on average, they were consuming 2.9 servings/day of plain water, 2.0 servings/day of SSBs (including sports drinks), and 1.1 serving/day of milk [50].

Sports and energy drinks, in particular, have become a popular source of hydration among adolescents. With the recent surge of energy drinks available on the market, these beverages have become synonymous with sports drinks; this is especially problematic since these drinks contain significant amounts of caffeine and added sugar. While consuming sports drinks (containing protein, carbohydrates, or electrolytes) can be helpful for adolescent athletes, particularly if exercising for longer than 60 min [28, 51], a recent (2011) statement by the American Academy of Pediatrics found that energy drinks have "no place in the diets of children or adolescents." [28] However, in a sample of 21,995 youth, 35% of 8th graders, 30% of 10th graders, and 31% of 12th graders consumed at least one energy drink or shot per day [26]. In another study of 779 youth ages 12–17 years, 20% agreed energy drinks are safe for teens and 13% agreed that energy drinks are a type of sports drink. There was also a positive association between physical activity and the consumption of energy drinks, such that those who were physically active 3 to 6x/week consumed 10.9 ± 2.1 energy drinks in the past week

[°]g/d

compared to 5.5 ± 1.2 drinks for those active <3 days per week, and 5.2 ± 2.3 drinks for those physically active 7 times/week (p=0.02) [52].

Sports drinks are appropriate, however, when adolescents are engaging in prolonged activity (longer than 1 h), especially if it requires short bursts of energy (e.g., soccer, basketball). As discussed earlier, adolescents' glycolytic capacity is still developing, which means they are relying on circulating carbohydrates for energy needs; sports drinks can satisfy this need. There are concerns with improper and unguided consumption of sports drinks; these include the displacement of calorie-free – and essential – water, which can lead to an increase in daily caloric intake, as well as the displacement of other health-promoting beverages such as milk. Some strategies that can ensure adolescents are replenishing their electrolytes while hydrating properly include using sports drinks only during prolonged activity, diluting the sports drink with water (50/50), and drinking seltzer water instead of soda and tea instead of coffee.

Paying attention to the timing of water consumption will also benefit young athletes. Between 1 and 2 h prior to exercise, athletes should consume $2\frac{1}{2}$ cups (-20 oz) of fluid; 15–30 min before, they should consume $1\frac{1}{2}$ cups (12 oz). The purpose of drinking before exercise is to ensure sufficient hydration and maximal electrolyte balance. During exercise, water consumption is needed to prevent drastic changes in electrolyte balance, as well as excess dehydration (>2% of body weight). For workouts lasting longer than 1 h, adolescent athletes should consume 6-8% carbohydrate–electrolyte beverages frequently (about $\frac{1}{2}$ cup every 10-15 min), as this will maintain appropriate fluid balance by replenishing sodium and potassium levels (thus preventing dehydration) and spare glycogen stores (particularly important if participating in multiple training sessions in 1 day) [53]. Due to differences in variables such as duration, frequency, type, and intensity of activity; training quality; hydration status; sweat rate; and exercise environment, individuals should monitor their hydration status in order to determine their appropriate re-hydration strategy. Adolescent athletes can calculate their own replacement needs by calculating weight loss during workouts. For each pound lost, individuals should consume 16 oz of water and avoid caffeine. Urine output is a good litmus test for hydration levels; it should be pale yellow to clear at the end of each day.

Dietary Supplements and Ergogenic Aids

Sales of dietary supplements sold in the USA totaled approximately \$36.7 billion in 2014 [54]. Research, however, has yet to consistently support the reliability, efficacy, and safety of dietary supplements [55]. This is especially true for adolescents. For example, research in adults suggests creatine has positive effects on both changes in total body and fat-free mass, and performance in sports that require short, intense bursts of energy (e.g., power lifting); [56] safety data for youth is limited [57]. In fact, one 2016 study found that the use of creatine supplements with young elite football players may increase inflammation of the airways, possibly leading to increased prevalence of asthma [58].

In fact, the American College of Sports Medicine (ACSM) does not recommend supplements for persons under 18 years of age due to insufficient, supportive data and the risk for developmental problems. Despite this, in a sample of 73.7 million children, 1.64% reported using a dietary supplement. Among those citing sport performance as their reason (1.65%), 95% took a multi-vitamin and/or mineral supplement (e.g., calcium), followed by fish oil/omega 3/DHA (43.5%), creatine (34.1%), and fiber (25.9%). Males were more likely than females to use supplements (OR=2.1; 95% CI, 1.3–3.3) [59]. The use of supplements in adolescent athletes should be done with the consultation of a dietitian or physician, and only in cases where there is an established nutrient deficiency (or significant risk for one).

Behavioral Techniques to Promote Healthful Dietary Patterns

Fluid and food consumption patterns are particularly important for the active adolescent, as intake habits have physiological and biological implications, and can serve to either impair or improve athletic and academic performance. While in-season, dietary behaviors should be seen as part of training, athletes should be encouraged to "practice" healthful eating in order to see the effects different foods have on them. Athletes' meal timing also matters. Prior to competitions, athletes should consume 1–4 g of carbohydrate/kg of body weight: 1 h prior, consume 1 g/kg; 4 h prior, 4 g/kg. While the meal should be high in carbohydrate, it should also be low in protein, fat, and fiber, and moderate in sodium.

For athletes playing endurance sports (longer 1 h) that require short bursts of energy (e.g., soccer, volleyball), consuming a combination of simple sugars has been shown to be beneficial; [53] discretionary calories should be kept under 200. For every hour of exercise, adolescents should ingest 30–60 g of carbohydrate and drink 4–8 oz of fluid every 15 min. After an event, athletes should consume a mixed carbohydrate/protein meal or snack within 15–20 min; this will boost glycogen storage by as much as 300%. Waiting 2 h post-exercise before refueling can reduce glycogen synthesis by 50%, thus impairing recovery.

Special Considerations: Weight Status and Eating Disorders

As adolescents age, the prevalence of eating disorders increases, with dieting being a strong predictor of onset [60]. One prospective cohort community study (duration 8 years; n=496) found that between the ages of 12 and 20 years, lifetime prevalence of eating disorders were as follows: 0.8% for anorexia nervosa, 2.6% for bulimia nervosa, and 3.0% for binge-eating disorder. Thirteen percent of the participants were diagnosed with an eating disorder by age 20 [61].

Individuals who do not meet Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) criteria for an eating disorder diagnosis may still exhibit disordered eating patterns; in fact, disordered weight control behaviors, such as self-induced vomiting and the use of laxatives, have been found to be 20 times more likely than a clinical diagnosis [62]. Furthermore, the prevalence of disordered weight control behaviors may be higher among youth athletes compared to non-athletes. One study assessed weight control strategies among 15,260 6th to 8th grade students and found that 3.6% of females and 3.1% of boys displayed disordered weight control behaviors. The odds, though, were highest for youth who engaged in strengthening/toning exercises 7 days/week compared to those who engaged for 0–3 days/week (females' OR = 1.9; 95% CI, 1.3–3.0; males' OR = 1.5; 95% CI, 1.0–2.2), with no variation by race/ethnicity or weight status.

Some sports, such as wrestling and martial arts, place competitors in certain weight classes. Others, such as gymnastics and dance, place a strong emphasis on lean physique. These types of sports place an additional burden on the athlete and can lead to short periods of disordered eating and weight adjustment patterns, such as those intended to lose water weight, including wearing plastic suits, using diuretics, and sitting for long periods of time in saunas. Over time, however, these behaviors can evolve into chronic unhealthy weight control behaviors that are clinically diagnosable, such as anorexia, bulimia, and muscle dysmorphia. For example, some athletes engage in purging or dietary restrictive behaviors prior to competition in order to "make weight." After competition, these athletes then compensate by consuming an excess amount of calories (binge), which leads to rapid weight gain. This dietary pattern can place such a strongly negative demand on the body that adverse physiological and developmental changes can occur [63].

Therefore, these athletes, in particular, may need additional guidance to ensure they are consuming a sufficient amount of nutrients (within potentially restrictive calorie allowances) in order to meet the

demands being placed on their body. Scheduling an appointment with a dietitian may be helpful. Coaches, too, can play an important role in helping to recognize and intervene if disordered eating patterns are observed. They can also serve as role models for youth. Kroshus and colleagues [64] conducted an electronic survey with 227 high school coaches from New York State about their knowledge, attitudes, communication, and management of behaviors related to the Female Athlete Triad. The researchers found gender differences among the coaching staff, such that while both coaches recognized low energy and menstrual disturbances as unhealthy and warranting attention, the rating of concern was higher among female versus male coaches. Male coaches reported stronger agreement to the statement, "Having an irregular menstrual cycle is often a sign that the athlete is in peak competitive shape" than female coaches (p=0.049). However, female coaches felt more comfortable discussing menstrual irregularities with their athletes [64].

Meal Timing

Fluid and food consumption patterns are particularly important for the active adolescent, as dietary habits have physiological and biological implications, and can serve to either impair or improve athletic and academic performance. Especially while in-season, dietary behaviors should be seen as part of training; athletes should be encouraged to "practice" healthful eating in order to see the effects different foods have on them. Athletes' meal timing also matters. Prior to competitions, athletes should consume 1–4 g of carbohydrate/kg of body weight: 1 h prior, consume 1 g/kg; 4 h prior, 4 g/kg. While the meal should be high in complex carbohydrates, it should also be low in protein, fat, and fiber, and moderate in sodium.

For athletes playing endurance sports that require short bursts of energy (e.g., soccer, volleyball), consuming a combination of simple sugars has been shown to be beneficial [53]; however, discretionary calories should be kept under 200. For every hour of exercise, adolescents should ingest 30–60 g of carbohydrate and drink 4–8 oz of fluid every 15 min. After an event, athletes should consume a mixed carbohydrate/protein meal or snack within 15–20 min; this will boost glycogen storage by as much as 300%. Waiting 2 h post-exercise before refueling can reduce glycogen synthesis by 50%, thus impairing recovery.

Promoting Healthy Environments for Youth

While youth sports are commonly seen as an encouraging strategy for promoting physical activity, the food environment in which many youth (and their parents) find themselves may be unhealthy and even obesogenic. Thomas and colleagues [3] conducted focus groups exploring parent perceptions about the youth (ages 6–13) sport environment. Parents reported that few healthful foods and beverages are available, and it is common for youth to consume unhealthful foods and beverages. Another study conducted by Irby and colleagues [65] assessed the youth baseball food environment. The authors found that 72% of the team snacks, provided by parents during or after games, were unhealthy – including French fries, chips, candy, and cookies. Beverages were most likely to be diet soda (33%), water (32%), or sugar-sweetened sports drinks (27%). Of those drinks consumed in the dugout during games, 53% were sugar-sweetened. Of foods consumed by spectators, 85% were purchased from the concession stand, and 73% of the items were unhealthy (French fries, chips, candy, cookies, ice cream) [65].

Conclusion

Research suggests that adolescent dietary habits can predict lifelong health behaviors [66], and youth's dietary behaviors tend to worsen as they age; [67, 68] this means establishing health-promoting dietary behaviors at this stage can have a lifelong impact for youth. Like all nutrition-related research, the literature base in this area continues to evolve, especially as more young adults participate in competitive sports.

While adolescent athletes may have performance needs to consider when establishing their dietary intake patterns, their needs do not appear to warrant the default consumption of additional nutrients, such as supplements or ergogenic aids. To the contrary, research appears to support the need for adolescent athletes to pay increasing attention to the quality of their diets – ensuring they are consuming a sufficient quantity of calories and micronutrients to meet their developmental and athletic performance needs and limiting the intake of "empty calories" often found in SSBs and salty snack foods. Practitioners can support this by encouraging adolescent athletes to consider their food and fluid intake as part of their training and by educating youth on the role nutrition plays in their competitive performance. Awareness of the challenges and social constraints placed on adolescents can aid practitioners in successfully communicating with this population.

References

- 1. National Council of Youth Sports. Report on trends and participation in youth sports 2008. http://www.ncys.org/ pdfs/2008/2008-ncys-market-research-report.pdf. Accessed 26 Feb 2014.
- Sacheck J, Nelson T, Ficker L, Kafka T, Kuder J, Economos CD. Physical activity during soccer and its contribution to physical activity recommendations in normal weight and overweight children. Pediatr Exerc Sci. 2011;23(2): 291–2.
- Thomas M, Nelson TF, Harwood E, Neumark-Sztainer D. Exploring parent perceptions of the food environment in youth sport. J Nutr Educ Behav. 2012;44(4):365–371. doi:http://dx.doi.org.ezproxy.library.tufts.edu/10.1016/j. jneb.2011.11.005.
- U.S. Department of Health & Human Services. Stages of adolescent development. http://eclkc.ohs.acf.hhs.gov/hslc/ tta-system/teaching/eecd/Curriculum/Planning/_34_Stages_of_adolescence1.pdf. Accessed 17 May 2011.
- 5. Steinberg L. Adolescence. 8th ed. New York: McGraw-Hill Humanities/Social Sciences; 2008.
- 6. Wardlaw GM, Kessel MW. Perspectives in nutrition. 5th ed. New York: McGraw-Hill Higher Education; 2002.
- 7. Guo S, Roche A, Chumlea W, Gardner J, Siervogel R. The predictive value of childhood body mass index values for overweight at age 35 y. Am J Clin Nutr. 1994;59(4):810–9.
- Stang J. Adolescent nutrition. In: Brown JE, editor. Nutrition through the life cycle. Belmont: Wadsworth/Thomson Learning; 2001. p. 325–54.
- 9. Heaney RP, Abrams S, Dawson-Hughes B, et al. Peak bone mass. Osteoporos Int. 2000;11(12):985–1009. doi:10.1007/s001980070020.
- Talwar SA, Swedler J, Yeh J, Pollack S, Aloia JF. Vitamin-D nutrition and bone mass in adolescent black girls. JAMA. 2007;99(6):650–7.
- 11. Allen JP, Loeb EL. The autonomy-connection challenge in adolescent? Peer relationships. Child Dev Perspect. 2015;9(2):101–5. doi:10.1111/cdep.12111.
- Allen JP, Uchino BN, Hafen CA. Running with the pack: teen peer-relationship qualities as predictors of adult physical health. Psychol Sci. 2015;26:1574–83.
- 13. Montfort-Steiger V, Williams C. Carbohydrate intake considerations for young athletes. J Sports Sci Med. 2007;6:343–52.
- 14. Nemet D, Eliakim A. Pediatric sports nutrition: an update. Curr Opin Clin Nutr Metab Care. 2009;12(3):304-9.
- 15. Riddell MC. The endocrine response and substrate utilization during exercise in children and adolescents. J Appl Physiol. 2008;105(2):725–33. doi:10.1152/japplphysiol.00031.2008.
- U.S. Department of Agriculture, U.S. Department of Health and Human Services. Dietary guidelines for Americans, 7th ed. Washington DC: U.S. Government Printing Office; 2010.
- Mozaffarian D, Benjamin EJ, Go AS, et al. Executive summary: heart disease and stroke statistics 2016 update: a report from the American Heart Association. Circulation. 2016;133:447–54. doi:10.1161/CIR.00000000000366.

- Reicks M, Jonnalagadda S, Albertson AM, Joshi N. Total dietary fiber intakes in the U.S. population are related to whole grain consumption: results from the National Health and Nutrition Examination Survey 2009 to 2010. Nutr Rev. 2014;34:226–34.
- Lipsky LM, Nansel TR, Haynie D, et al. Behavioral and sociodemographic correlates of overall diet quality over 4 years in a national cohort of U.S. emerging adults. FASEB J. 2016;30(1):152–3.
- Zhang Z, Gillespie C, Welsh JA, Hu FB, Hu FB, Yang Q. Usual intake of added sugars and lipid profiles among the U.S. adolescents: National Health and Nutrition Examination Survey, 2005–2010. Adolescent Health. 2015;56(3):352–9.
- Johnson RK, Appel LJ, Brands M, et al. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. Circulation. 2009;120(11):1011–20. doi:10.1161/CIRCULATIONAHA.109.192627.
- Rodríguez LA, Madsen KA, Cotterman C, Lustig RH. Added sugar intake and metabolic syndrome in US adolescents: cross-sectional analysis of the National Health and Nutrition Examination Survey 2005–2012. Public Health Nutr. 2016;FirstView:1–11. doi:10.1017/S1368980016000057.
- Basu S, Yoffe P, Hills N, Lustig RH. The relationship of sugar to population-level diabetes prevalence: an econometric analysis of repeated cross-sectional data. PLoS One. 2013;8:e57873.
- Yang Q, Zhang Z, Gregg EW, Flanders WD, Merritt R, Hu FB. Added sugar intake and cardiovascular diseases mortality among U.S. adults. JAMA. 2014;174(4):516–24.
- 25. Endorsed by the American Academy of Pediatrics, Gidding SS, Dennison BA, et al. Dietary recommendations for children and adolescents: a guide for practitioners: consensus statement from the American Heart Association. Circulation. 2005;112(13):2061–75. doi:10.1161/CIRCULATIONAHA.105.169251.
- Terry-McElrath YM, O'Malley PM, Johnston LD. Energy drinks, soft drinks, and substance use among United States secondary school students. J Addict Med. 2014;8:6–13.
- Ervin RB, Ogden CL. Trends in intake of energy and macronutrients in children and adolescents from 1999–2000 through 2009–2010. NCHS Data Brief. 2013;113:1–8.
- Committee on Nutrition and the Council on Sports Medicine and Fitness. Sports drinks and energy drinks for children and adolescents: are they appropriate?. Pediatrics. 2011;127(6):1182–9. doi:10.1542/peds.2011-0965.
- 29. Phillips SM. Protein requirements and supplementation in strength sports. Nutrition. 2004;20(7–8):689–95. doi:10.1016/j.nut.2004.04.009.
- Bolster DR, Pikosky MA, McCarthy LM, Rodriguez NR. Exercise affects protein utilization in healthy children. J Nutr. 2001;131(10):2659–63.
- 31. Heaney RP, Layman DK. Amount and type of protein influences bone health. Am J Clin Nutr. 2008;87(5): 1567S-70.
- Keast DR, Fulgoni VL, Nicklas TA, O'Neil C. Food sources of energy and nutrients among children in the United States: National Health and Nutrition Examination Survey 2003–2006. Nutrients. 2013;5(1):283–301.
- 33. Thacher TD, Clarke BL. Vitamin D insufficiency. Mayo Clin Proc. 2011;86(1):50-60.
- Karalius VP, Zinn D, Wu J, et al. Prevalence of risk of deficiency and inadequacy of 25-hydroxyvitamin D in U.S. children: NHANES 2003–2006. J Pediatr Endocrinol Metab. 2014;27(5–6):461–6.
- 35. Institute of Medicine. Dietary reference intakes for calcium and vitamin D. Washington, DC: National Academy of Sciences; 2010. Available from: http://www.iom.edu/~/media/Files/Report%20Files/2010/Dietary-Reference-Intakes-for-Calcium-and-Vitamin-D/Vitamin%20D%20and%20Calcium%202010%20Report%20Brief.pdf.
- 36. Holick MF. Vitamin D deficiency. N Engl J Med. 2007;357(3):266–281.
- Matsuoka LY, Ide L, Wortsman J, MacLaughlin JA, Holick MF. Sunscreens suppress cutaneous vitamin D3 synthesis. J Clin Endocrinol Metab. 1987;64(6):1165–8.
- 38. Beard J, Tobin B. Iron status and exercise. Am J Clin Nutr. 2000;72(2):594S-7.
- 39. Ilich JZ, Kerstetter JE. Nutrition in bone health revisited: a story beyond calcium. J Am Coll Nutr. 2000;19(6):715–37.
- Bailey DA, Martin AD, McKay HA, Whiting S, Mirwald R. Calcium accretion in girls and boys during puberty: a longitudinal analysis. J Bone Miner Res. 2000;15:2245–50.
- Martin A, Bailey D, McKay H, Whiting S. Bone mineral and calcium accretion during puberty. Am J Clin Nutr. 1997;66(3):611–5.
- 42. Wallace TC, Reider C, Fulgoni VL. Calcium and vitamin D disparities are related to gender, age, race, household income level, and weight classification but not vegetarian status in the United States: Analysis of the NHANES 2001–2008 data set. J Am Coll Nutr. 2013;32(5):321–30.
- Burrows M. Exercise and bone mineral accrual in children and adolescents. J Sports Sci Med. 2007;6:305–12.
- Malczewska J, Raczynski G, Stupnicki R. Iron status in female endurance athletes and in non-athletes. Int J Sport Nutr. 2000;10(3):260–76.
- Baker LB, Dougherty KA, Chow M, Kenney WL. Progressive dehydration causes a progressive decline in basketball skill performance. Med Sci Sports Exerc. 2007;39(7):1114–23. doi:10.1249/mss.0b013e3180574b02.
- 46. Stover EA, Zachwieja J, Stofan J, Murray R, Horswill CA. Consistently high urine specific gravity in adolescent American football players and the impact of an acute drinking strategy. Int J Sports Med. 2006;27(4):330–5. doi:1 0.1055/s-2005-865667.

- Bergeron MF, Waller JL, Marinik EL. Voluntary fluid intake and core temperature responses in adolescent tennis players: sports beverage versus water. Br J Sports Med. 2006;40(5):406–10. doi:10.1136/bjsm.2005.023333.
- 48. Campbell SM. Hydration Needs throughout the Lifespan. J Am Coll Nutr. 2007;26 Suppl 5:585S-587.
- Drewnowski A, Rehm CD, Constant F. Water and beverage consumption among children age 4-13y in the United States: analyses of 2005–2010 NHANES data. Nutr J. 2013;12:85.
- Kenney EL, Long MW, Cradock A, Gortmaker SL. Prevalence of inadequate hydration among US children and disparities by gender and race/ethnicity: National Health and Nutrition Examination Survey, 2009–2012. Am J Public Health. 2015;105(8):e113–8.
- 51. Smith EL, Mechanick JI. Hydration and nutrition for the athlete. In: Herrera JE, Cooper G, editors. Essential sports medicine. Totowa: Humana Press; 2008. p. 169–80.
- 52. Kumar G, Park S, Onufrak S. Perceptions about energy drinks are associated with energy drink intake among U.S. Youth. Am J Health Promot. 2015;29(4):238–44. doi:10.4278/ajhp.130820-QUAN-435.
- Sawka MN, Burke LM, Eichner R, Maughan RJ, Montain SJ, Stachenfeld NS. Exercise and fluid replacement. Med Sci Sports Exerc. 2007;39(2):377–90. doi:10.1249/mss.0b013e31802ca597.
- 54. Penton Media I. NBJ's supplement business report 2015. Nutrition Business. 2015.
- 55. Greydanus DE, Patel DR. Nutritional supplements: sports doping in the adolescent. Int J Disabil Hum Dev. 2009;8(2):119-24.
- Volek JS, Duncan ND, Mazzetti SA, et al. Performance and muscle fiber adaptations to creatine supplementation and heavy resistance training. Med Sci Sports Exerc. 1999;31(8):1147–56.
- Castillo EM, Comstock RD. Prevalence of use of performance-enhancing substances among United States Adolescents. Pediatr Clin North Am. 2007;54(4):663–75. doi:10.1016/j.pcl.2007.04.002.
- 58. Kippelen P, Simpson A, Horne S, Sharp P, Sharps R. Effects of creatine supplementation on the airways of youth, elite football players. Proc Physiol Soc. 2016; 35, PC03.
- 59. Evans MW, Ndetan H, Perko M, Williams R, Walker C. Dietary supplement use by children and adolescents in the United States to enhance sport performance: results of the National Health Interview Survey. J Prim Prev. 2012;33(1):3–12.
- 60. Patton GC, Selzer R, Coffey C, Carlin JB, Wolfe R. Onset of adolescent eating disorders: population based cohort study over 3 years. BMJ. 1999;318(7186):765–8.
- Stice E, Marti N, Rohde P. Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. J Abnorm Psychol. 2012;122:445–57.
- 62. Favaro A, Ferrara S, Santonastaso P. The spectrum of eating disorders in young women: a prevalence study in a general population sample. Psychosom Med. 2003;65(4):701–8.
- Boisseau N, Vera-Perez S, Poortmans J. Food and fluid intake in adolescent female judo athletes before competition. Pediatr Exerc Sci. 2005;17:62–71.
- 64. Kroshus E, Sherman RT, Thompson RA, Sossin K, Austin SB. Gender differences in high school coaches' knowledge, attitudes, and communication about the female athlete triad. Eat Disord. 2014;22(3):193–208. doi:10.1080/1 0640266.2013.874827.
- 65. Irby MB, Drury-Brown M, Skelton JA. The food environment of youth baseball. Child Obes. 2014;10(3):260-5.
- 66. Nelson MC, Story M, Larson NI, Neumark-Sztainer D, Lytle LA. Emerging adulthood and college-aged youth: an overlooked age for weight-related behavior change. Obesity. 2008;16(10):2205–11. doi:10.1038/oby.2008.365.
- 67. Reedy J, Krebs-Smith SM. Dietary sources of energy, solid fats, and added sugars among children and adolescents in the United States. J Am Diet Assoc. 2010;110(10):1477–84. doi:10.1016/j.jada.2010.07.010.
- Striegel-Moore RH, Thompson D, Affenito SG, et al. Correlates of beverage intake in adolescent girls: the National Heart, Lung, and Blood Institute Growth and Health Study. J Pediatr. 2006;148(2):183–7. doi:10.1016/j. jpeds.2005.11.025.

Chapter 15 Effects of an Active Lifestyle on Water Balance

Gethin H. Evans, Ronald J. Maughan, and Susan M. Shirreffs

Key Points

- Body water content is normally maintained within narrow limits by regulatory mechanisms affecting both intake and loss. Water balance is intimately linked with salt balance to regulate osmolality of body water compartments.
- The physiological mechanisms that govern intake may be over-ridden by conscious control, resulting in disturbances of water and salt balance.
- Transient modest disturbances in water balance are common and of little or no consequence. Both a reduction (hypohydration) and increase (hyperhydration) in body water content may, if sufficiently severe, lead to adverse effects on health and performance.
- Water losses are increased by exercise and warm environments, but responses are highly individual.
- Undertaking physical activity in a hypohydrated state appears to increase an individual's perceived exertion which may negatively influence exercise performance and self-selected exercise intensity, and may decrease the likelihood of further participation in physical activity.
- Certain populations, such as the elderly, are more likely to become hypohydrated, which may lead to other illnesses and contribute to morbidity and mortality.

Keywords Hydration • Hypohydration • Water turnover • Physical activity • Occupational activity • Perceived exertion • Exercise performance • Exercise participation • Over-hydration

G.H. Evans

R.J. Maughan (🖂)

S.M. Shirreffs Department of Medicine, St Andrews University, St Andrews, UK

School of Healthcare Science, Manchester Metropolitan University, Manchester M1 5GD, UK

School of Sport, Exercise and Health Sciences, Loughborough University, Loughborough, UK e-mail: r.maughan@lboro.ac.uk

Abbreviations

ANP	Atrial natriuretic peptide
AVP	Arginine vasopressin
EFSA	European Food Safety Authority
mL	millilitre
mosm	milliosmole
RPE	Rating of Perceived Exertion
VO_{2max}	Maximum oxygen uptake

Background

Water is the most abundant component in the human body, except in the very obese, making up about 50–70% of body mass. Approximately two thirds of this water is found in intracellular compartments, with the remaining third in extracellular compartments. Within the extracellular compartment, water is found in the interstitial fluid (approximately 15% of body mass) and the blood plasma (5% body mass). Despite the abundance of water in the human body, it is kept within relatively narrow limits. Euhydration is defined as a "normal" body water content, usually denoted by a plasma osmolality between 280 and 290 mosmol/kg, although this increases with age [31]. The body water content fluctuates over the day as losses are continuous and intake is episodic, but it is normally maintained within about $\pm 0.2\%$ of body mass in temperate conditions and within $\pm 0.5\%$ when in the heat or during exercise [29]. Individuals are considered to be hypohydrated or hyperhydrated if values fall out with these limits. Both of these conditions, if sufficiently severe, can impair all aspects of physiological function and may prove fatal in extreme cases.

Water Balance

Water balance is achieved when water intake matches water losses, but water intake is episodic whereas water losses are continuous, so water balance fluctuates throughout the course of a day. Over a 24-h period, water balance is generally maintained through normal physiological function. Avenues of water intake include fluid consumed through drinking and that found in ingested food with a small proportion generated via oxidative metabolism (Table 15.1). At rest and in temperate environments, the main avenue of fluid loss is via urine production with some water lost via respiration, skin loss,

Water intake		Water output		
Variable	Volume (L)	Variable	Volume (L)	
Water consumption	1.6	Urine	1.4	
Water in food	1.0	Respiration	0.3	
Metabolic water production	0.4	Skin (transcutaneous)	0.5	
		Sweat	0.7	
		Faeces	0.1	
Total	3.0	Total	3.0	

Table 15.1 Typical values (mL) of different avenues of daily water input and output for a 70–80 kg sedentary male living and working in a temperate environment

sweating and faecal loss. During exercise and in hot environments, a greater proportion of water is lost via sweating in order to limit the rise of core temperature that occurs in these situations.

Urine production is regulated by hormones including arginine vasopressin (AVP), aldosterone and atrial natriuretic peptide (ANP). AVP is secreted from the posterior pituitary and acts on the collecting ducts of the kidney to increase water reabsorption, leading to production of small volumes of concentrated urine. The main physiological factors that initiate the secretion of AVP are an increase in plasma osmolality and a decrease in blood volume. A 1 mosm/kg change in plasma osmolality results in a 0.41 pmol/L change in AVP [8], and a 3 mosm/kg change in plasma osmolality results in a 250 mosm/kg change in urine osmolality [69]. A change in blood volume of 7–10% is required for similar magnitudes of change in urine osmolality, making plasma osmolality the main determinant of AVP release [8]. The renin–angiotensin aldosterone system is also activated in response to decreases in blood volume, with aldosterone acting on the collecting ducts of the kidney to increase sodium reabsorption, which, in turn, leads to water reabsorption and the production of small volumes of concentrated urine. ANP is secreted from the right atrium in response to increase blood volume and leads to decreases in sodium reabsorption in the nephron and also inhibits renin secretion, leading in turn to an increase in urine sodium content and urine volume.

One of the main drivers for fluid intake is thirst, though much drinking behaviour is dictated by habit rather than stimulus-driven. Thirst is the result of a complex interaction between physiological, psychological and behavioural factors [1]. Changes in plasma osmolality and blood volume are thought to play important roles in the thirst response, thus the same factors that regulate water loss are also involved in water intake. Animal models have suggested that substances such as angiotensin 2, bradykinin and serotonin may also be involved in the thirst response. Recently, there have been some suggestions that genetic factors may also account for some variation in thirst response between individuals, although the picture is not entirely clear at present [74, 88]. Generally, thirst is considered to be a poor indicator of short-term deviations in body water, given observations that unlimited access to drinks after periods of fluid restriction tends not to fully restore water losses [78], and rapid fluid ingestion in these circumstances appears to alleviate the perception of thirst before euhydration is restored and before major changes in circulating osmolality are observed [71]. The thirst response is considered, however, to be sufficient to restore body water levels over longer time periods.

The European Food Safety Authority (EFSA) has concluded that adequate total (i.e., water from food and fluid) daily water intake is 2.0 L for an average adult female and 2.5 L for an average adult male 21. Different adequate total water intakes are reported by the Institute of Medicine in the USA with values reported as 2.7 L and 3.7 L for adult females and males, respectively. The values reported by these bodies are an average (or median) volume consumed by healthy adults and are, therefore, classed as adequate intakes rather than recommended intakes. Considerable variation in the volumes of water and other beverages habitually consumed by healthy adults is observed. For physically active individuals that will lose greater volumes of water via sweating, which is exacerbated in hot environments, achieving the adequate intakes reported is not a guarantee that water intake is sufficient for that individual. It is likely, however, that water intake throughout the day will match water losses due to a combination of habitual fluid intake and the individual's thirst response driving further fluid intake.

Sweating, Water Balance and Water Turnover

At rest, metabolic heat production is approximately 60 W, but metabolic demand increases in a linear fashion and can reach 1 kW during activities such as marathon running. This metabolic heat must be dissipated in order to avoid large increases in core temperature, and this is met largely by an increase in sweat rate. The main factors that determine sweat rate are exercise intensity, duration and frequency as well as the environment in which physical activity is undertaken.

In recent years, a large number of studies have been undertaken to measure pre-exercise hydration status and to assess sweat rate loss and fluid replacement in physically active individuals. Most of these studies have focussed on elite athletes undertaking training sessions and/or competitive events in different environmental conditions. Maughan et al. [49] studied elite football players during a training session in warm conditions and observed a median pre-exercise urine osmolality of 666 mosm/kg with a range of 103-1,254 mosm/kg. During exercise, median fluid intake was 971 mL with a range of 265–1,661 mL, and percentage body mass loss was 1.4% with a range of 0.5–2.6%. In a similar study performed in a cold environment, Maughan et al. [51] observed that pre-exercise urine osmolality was 872 mosm/kg (range: 481-1,228 mosm/kg), fluid intake was 420 mL (range: 44-950 mL) and percentage body mass loss was 1.6% (range: 0.9–2.5%). Similar results have been observed during competitive football matches [52] and throughout a competitive handball tournament [18]. These studies demonstrate that some individuals are likely to begin exercise in a hypohydrated state, that there are large inter-individual differences in sweat rate and fluid intake during exercise and that some individuals are likely to become significantly dehydrated by undertaking relatively short but intense periods of physical activity. If such results are seen in elite performers with a large professional support team, it is likely that recreational athletes also experience similar or greater levels of hypohydration.

Sweat consists of water, minerals and organic components. Consequently, when sweat rate increases during exercise, it is not just water that is lost: The main electrolytes lost via sweat are sodium and chloride at concentrations of approximately 15–80 mmol/L. There is large inter-individual variation in the composition of sweat leading to large variations in electrolyte and salt loss between individuals [49, 51]. Sweat is invariably hypotonic, however, and one effect of high sweat rates is to cause an increase in the osmolality of body fluids. The variability in sweat rate and sweat composition, in addition to the large individual variability in sweat loss, means that a single hydration strategy is not appropriate for all physically active individuals, and hydration advice should be tailored to the individual in question.

Body water turnover, or the replacement of water lost over a given period, is approximately 5–10% of total body water content per day for a sedentary individual living in a temperate environment, but can increase to 20–40% if prolonged exercise is undertaken in a hot environment [36]. Fractional water turnover is higher in children up to 15 years of age than in adults and higher in physically active individuals than in age-matched sedentary individuals [76]. Leiper et al. [43] reported that average median water turnover in a cool environment for a group of regular cyclists was 47 mL/kg/d compared to 36 mL/kg/d for a group of age-matched sedentary controls, with the difference in water turnover due primarily to non-renal water losses. When similar studies [41] were undertaken in temperate environments, water turnover rates were higher (4,673 mL/d) in recreational joggers than sedentary age-matched controls (3,256 mL/d), but the difference was due to an increased renal loss rather than differences in sweat loss. This suggests that these individuals may have over-estimated their fluid requirements.

Physical activity comprises both voluntary exercise and occupational activity. In many cases, occupational activity may be performed in hot and humid environments or when wearing uniforms and/or personal protective equipment that prevent physical heat loss and restrict evaporation of sweat. Consequently, undertaking occupational activity in these situations will have an influence on hydration status and body water balance. While this may lead to health consequences such as heat stroke, it may also affect safety, productivity, worker morale and operating costs.

Brake [13] monitored the hydration status, sweat rate and fluid intake of metal and coal miners. In a group of 64 miners tested at the start of their shift, only 41% were classed as having "good" or "fair" hydration status based on urine-specific gravity measurements and were classed as fit to work in a thermally stressful environment. The remaining 59% were classed as unfit to work in this environment, with 9% being classed as "clinically dehydrated". Further analysis of a cohort of 413 miners who were tested after the completion of their shift suggested that 86% of these workers were adequately hydrated. This would suggest that access to fluids during their working hours was sufficient

in most cases. In a group of 39 miners, fluid intake over the course of a variety of shift lengths averaged 6.5 L with a range of 2.4–12.5 L. Analysis of urine-specific gravity measures at the start, middle and end of the shifts showed no change in hydration status, which would suggest that fluid intake was adequate to meet sweat losses in these individuals. In a study of outdoor workers in North-West Australia, Miller and Bates [55] reported that only 21% of 382 urine samples analysed before, during and after summer shifts were classed as "adequately hydrated" or "marginally adequately hydrated". Fifty-one per cent of workers were classed as "hypohydrated" with 16% classed as "clinically dehydrated". Similar findings were reported by Bates et al. [7] when investigating expatriate workers in the Middle East during the summer months. Urine samples were collected from workers at four different working sites, and the percentage of samples classed as "hypohydrated" varied from 14 to 31%. Fluid intake in these individuals appeared to be sufficient to match sweat losses induced during occupational activity. The previous studies were undertaken in relatively high ambient temperatures, but Polkinghorne et al. [66] recently reported that 58% of miners working in temperate conditions were classed as "hypohydrated" both before and after a shift. They also noted that miners that began their shift dehydrated were nearly three times more likely to end their shift dehydrated than if they started in an adequate hydration state. Interestingly, workers that were classed as hypohydrated for the entire shift were more likely to be obese and at risk of metabolic complications (as assessed by waist circumference measurements) than those that were adequately hydrated for the duration of their shift.

The evidence described suggests that a degree of hypohydration is common in workers beginning demanding occupational physical activity in both temperate and hot environmental conditions. While this increases the risk of heat illness as well as having several potential effects on work productivity, workers in these situations appear to have adequate access to fluids during the working period to match sweat loss and, therefore, avoid exacerbated levels of dehydration. It would appear, however, that attempts need to be made to avoid the number of workers reporting to the workplace with significant levels of dehydration.

In a study of forestry workers, a situation in which there may be limited access to fluids, Biggs et al. [10] observed that 43% of 103 workers in autumn were dehydrated at the start of their shift and this increased to 64% at the end of the shift. Similarly, in winter, 47% of 79 workers were dehydrated at the onset of work and 63% were dehydrated afterwards. This would suggest that, in this working environment, fluid intake was not sufficient to replace fluid losses, although further studies have suggested that adequate hydration status is likely to be restored by the following morning [68] which would be expected as a result of longer-term regulatory processes of water balance.

Miller and Bates [56] set out a number of practical guidelines to assist in the avoidance of dehydration in manual workers. These include supplying workers with appropriate drink containers both prior to and during manual labour, drinking 0.6–1.0 L of fluid in a 2–3-h period, providing electrolyte replacement solutions when in hot environments, encouragement of rehydration after shifts and education of workers regarding monitoring of their hydration status. The observations and practical applications described above apply mostly to occupations that involve a high degree of manual labour, but this is not the case for most workers. Despite this, it appears that a significant number of more sedentary (or at least less physically active) workers also begin work with inadequate hydration levels and remain in this condition throughout the working day [53]. While the risk of heat illness and other health issues is likely to be relatively low in these individuals, this is still an issue to be considered in the workplace.

Hydration Status and Performance

A considerable volume of research has been undertaken to investigate the effects of deviations in body water on physical capacity. Case studies demonstrate that prolonged physical activity in challenging environmental conditions without fluid ingestion can lead to collapse, significantly impaired

physiological function and hypernatraemia [62]. Similarly, other case studies demonstrate that extreme over-ingestion of fluid during exercise can also lead to impaired physiological function and hyponatraemia which can, in rare cases, prove to be fatal [63]. These studies demonstrate that large acute disturbances in body water can undoubtedly affect performance and physiological function, but such extreme disturbances are relatively rare. The relatively small deviations from normal body water levels that are normally encountered appear to have little effect on performance and/or physiological function. Given that the main issue related to physical activity and hydration status is the increased rate of sweating and consequent water loss, most research has focussed on the impact of dehydration on exercise performance rather than hyperhydration. Despite the volume of research, much debate still exists regarding the point at which a reduction in body water is likely to affect physiological function. This is primarily due to methodological issues in experimental design in this area.

Armstrong et al. [3] observed that dehydration equivalent to about 1.8% of body mass on average induced via administration of a diuretic led to increases in running time and reductions in running velocity at distances of 1,500, 5,000 and 10, 000 m. Cheuvront et al. [15] reported that total work performed during a 30-min endurance exercise time trial in temperate conditions at 50% VO2_{peak} was less when participants were hypohydrated by 3% of their body mass than when they were euhydrated. Similarly, McConell et al. [54] observed that endurance exercise performance in a temperate environment was reduced when participants were hypohydrated that undertaking exercise in a dehydrated condition reduced VO_{2max} and time to fatigue in a warm environment when compared to a euhydrated condition. Similar observations have been reported by others [6, 9]. A recent review by Cheuvront and Kenefick [16] concluded that the majority of studies in this area have suggested that a reduction in body mass of 2% or more is likely to reduce endurance exercise performance particularly when that exercise is performed in a hot environment.

The effects of hypohydration on cardiovascular function have also been extensively studied. Gonzalez-Alonso et al. [28] reported that stroke volume was reduced and heart rate increased when participants were dehydrated and undertook exercise in a hyperthermic environment. This reflects at least in part the phenomenon of cardiovascular drift that has been reported to occur in prolonged exercise for at least 30 years [59, 72]. In addition to the impact of hypohydration on stroke volume and heart rate, Gonzalez-Alonso et al. [26] have also reported a reduction in skin blood flow compared to the euhydrated state which could reduce the extent of heat loss and lead to increases in core temperature. An individual's rating of perceived exertion (RPE) at a given exercise intensity has also been shown to increase when exercising in a hypohydrated state compared to a euhydrated state [5, 9, 58] which is likely to be another mechanism by which a reduction in body water negatively influences exercise performance. Interestingly, a recent study by Fleming and James [24] demonstrated that habituation to undertaking exercise in a hypohydrated state reduced the detrimental effects of a reduction in body water on endurance exercise performance. This effect was not mediated by changes in cardiovascular system function but apparently by habituation reducing RPE.

It has been suggested that a reduction in body water of 3-4% is required to induce detrimental effects on muscle function [35]. A recent meta-analysis of 28 articles [75] concluded that hypohydration results in meaningful reductions in anaerobic power, muscle endurance and strength and that the method of inducing the reduction in body water and training status were important considerations. In particular, hypohydration induced by passive means resulted in less effect on muscular performance than those that involved an active component. This meta-analysis also concluded that body-weight-dependent muscular performance, such as vertical jumping ability, may be improved by a reduction in body water of 3% or more.

Relatively mild levels of hypohydration have been shown to negatively affect cognition. Many of the older studies in this area suffer from poor methodology, but recent studies use more robust methods. Watson et al. [85] required participants to follow a drinking regimen that involved ingestion of 25% of suggested daily guidelines on the day prior to the trial, resulting in a reduction in body mass

of 1.1%. During a 2-h simulated driving task, a greater number of minor driving errors were recorded when participants were hypohydrated compared to when they performed the task having followed current drinking guidelines. Similarly, Lindseth et al. [45] reported that flight performance and spatial cognition tests were reduced in pilots who were hypohydrated due to low levels of fluid ingestion compared to those who were euhydrated with adequate levels of fluid ingestion. Ganio et al. [25] observed that mild hypohydration of approximately 1.5% body mass resulted in reduced cognitive aspects such as visual vigilance and visual working memory when compared to when euhydrated. These studies suggest that even mild levels of hypohydration appear to have a negative impact on cognition.

Body water deficits can be avoided or reversed only by ingestion of drinks or water-containing foods, so much attention has been given to the development of appropriate drinking strategies during exercise. As previously discussed, relatively small acute reductions in body water (as assessed by changes in body mass) are unlikely to cause any negative effects on performance and, therefore, a drinking strategy that restores all body water lost during exercise is unnecessary. Similarly, generic advice to all physically active individuals is not appropriate due to the large individual differences in sweat losses that occur during exercise. In such a situation, this guidance would be sufficient for some individuals, but would cause some to become hypohydrated and would lead to excessive ingestion for others, with the accompanying risk of hyponatraemia. Water and electrolyte losses can be assessed during training in response to different environmental conditions, and a drinking strategy implemented to avoid reductions in body water that may negatively influence exercise performance [50]. Alternatively, individuals can drink only when thirsty [61] or ad libitum. While these last two strategies are similar, they differ in that drinking to thirst relies solely on the thirst response to drive fluid intake, whereas drinking ad libitum involves other external cues in addition to thirst response. Armstrong et al. [4] compared a drinking to thirst strategy to an ad libitum strategy in trained cyclists completing a 164-km road cycle at an ambient temperature of 36°. Although the percentage body mass lost and fluid intake were the same between the two groups, the authors concluded that an ad libitum strategy may be easier to implement as this involves less thought and consideration while exercising.

It seems clear that a single hydration strategy is not appropriate for all individuals, and numerous confounding factors need to be considered. These include the individual's water loss, individual preference of taste and familiarity with drinks, the ambient temperature, temperature of the drink and the carbohydrate and electrolyte content of the drink. An additional consideration is individual insusceptibility to gastrointestinal disturbance that may occur as a result of fluid ingestion during exercise. Relatively high-intensity exercise (>70 % VO_{2max}) reduces the rate at which fluid is emptied from the stomach [42] as does ingestion of solutions with relatively high carbohydrate concentration [81]. Consequently, ingestion of large volumes of water and/or solutions containing carbohydrate during exercise may lead to gastric discomfort in some individuals. While there is some evidence that it is possible to train the gastrointestinal system to the ingestion of these volumes [34], it is still an important consideration when planning hydration strategies during physical activity.

A body water deficit is normally present after prolonged exercise, and there is a large body of research on post-exercise rehydration. If a single exercise session is completed during the day, there is no need for an aggressive rehydration strategy to be implemented as normal regulatory processes governing fluid intake will ensure that water balance is maintained over a 24-h period. If a second exercise session is to be performed on the same day, a post-exercise rehydration strategy may be needed to ensure that euhydration is restored before the second session. The most important factors to consider are the volume and sodium content of fluid that is consumed. Shirreffs et al. [79] observed that a volume greater than that lost during exercise is required to account for obligatory urine losses. This is typically translated as 1.5 L for every 1.0 kg of body mass lost during exercise. It also seems as though the rate at which fluid is ingested is an important consideration with high rates of fluid ingestion less effective at maintaining fluid balance over a recovery period than low rates of ingestion

of the same volume [39]. The composition of a rehydration solution is likely to alter the effectiveness of that solution to maintain fluid balance over a recovery period. The most important constituent of a rehydration solution is the sodium concentration as this is the most abundance ion in the extracellular fluid and, therefore, has a significant impact on plasma osmolality. The addition of sodium to a rehydration solution has been shown to improve its effectiveness at maintaining fluid balance during a recovery period [77]. The addition of carbohydrate and protein to a solution may also be of benefit as the addition of these nutrients reduces the rate of gastric emptying and overall water absorption ensuring that water loss is minimised due to the avoidance of large changes in plasma volume [22, 33]. An additional consideration is whether food is ingested alongside a solution. Water is not considered to be an adequate rehydration solution when ingested on its own as it is rapidly absorbed into the blood and causes a relatively large reduction in plasma volume and a diuresis [64], but when ingested alongside a meal containing adequate electrolytes, water may be a suitable rehydration solution [48].

Hydration for Recreational Activity

Regular, moderate physical activity is known to induce numerous health benefits, and cardiorespiratory fitness is considered an independent risk factor for a number of disease states [12]. Physical activity is therefore often prescribed to improve health. While physical activity alone does not appear to induce large degrees of weight loss, it does have beneficial effects on body composition, and the incorporation of physical activity into a weight loss strategy that also includes restriction of energy intake is likely to produce the greatest degree of body mass loss [87]. In addition, the extent of weight regain after a period of weight loss is likely to be lower if physical activity is incorporated into the initial weight loss strategy [82]. An individual's rating of perceived exertion (RPE) during physical activity is a key regulator of self-selected exercise intensity and will likely determine whether that individual continues or terminates an exercise session. Consequently, factors that alter an individual's RPE during exercise are of interest to insure compliance with exercise strategies used to promote health.

The sensation of effort during exercise is a complex phenomenon involving numerous physiological, psychological and social factors [80] and is typically assessed using a Borg scale [11]. Briefly, the perception of effort during exercise involves integration of physiological mediators (such as ventilation, availability of energy substrate and skin temperature) with psychological factors (such as motivation, mood state and experience), exertional symptoms (such as heavy breathing and sweat rate) and, in the case of elite athletes, performance considerations, by the sensory cortex. This results in a perceptual response that will determine the perceived effort of exercise [70]. The strongest physiological mediators of RPE are respiratory rate, sensation of strain in muscles and joints, perception of body temperature and limb movement speed [70]. Given the close relationship between core body temperate, sweat rate, skin temperature and hydration status, it is no surprise that there is interest in determining whether an individual's hydration status influences their perception of effort during exercise.

Few, if any, studies have been carried out specifically to study the effect of dehydration on RPE during exercise. Instead, RPE has been measured as a secondary outcome measure in studies examining the effect of dehydration on other markers of physiological function. In some of these studies, subjects began exercise in a dehydrated state, while in others dehydration was induced by withholding fluids during exercise. Cheuvront et al. [15] induced dehydration via heat 3 h of heat exposure that resulted in a 3% reduction in body mass prior to undertaking a 30-min exercise trial at 50% VO_{2max} and a 30-min time trial. No differences in RPE were observed between the euhydrated or dehydrated states. Similarly, Kenefick et al. [37] reduced body water by 1.7–1.8 L via 75 min of exercise in hot conditions before a 75-min heat tolerance test involving running exercise. During this heat tolerance test, there was little difference in RPE when participants were euhydrated than when dehydrated. In

contrast, Riebe et al. [67] induced a level of 4% dehydration via exercise in the heat before a period of rehydration or no fluid ingestion. During a subsequent exercise test, RPE was significantly higher in the dehydrated state than in the euhydrated condition. Similar findings of an increase in RPE when previously dehydrated compared to a euhydrated condition have been observed by Fleming and James [24] and Gonzalez-Alonso et al. [27]. On balance, it would seem that beginning exercise in a dehydrated state is likely to lead to a small but meaningful increase in RPE.

Montain and Coyle [57] showed an association between the extent of dehydration accrued during prolonged exercise in a warm environment and the RPE. Watson et al. [84] reported a significant increase in RPE when participants undertook an intermittent exercise trial at 55 % VO_{2max} in hot conditions without fluid ingestion compared to when plain water was ingested to match sweat losses, that is, dehydration was allowed to accrue. Similarly, Baker et al. [5] observed that 3 h of treadmill walking without fluid replacement led to an increase in RPE in comparison to when fluid replacement was allowed. Ishijima et al. [32] reported that 90 min of cycle exercise at 55 % VO_{2max} without fluid ingestion led to greater RPE than when exercise was undertaken with fluid ingestion. These observations would suggest that dehydration accrued while exercising for longer than 30 min may also lead to a small but meaningful increase in RPE.

Several mechanisms may explain why hypohydration may lead to an increase in RPE, including both physiological and psychological factors. As discussed previously, hypohydration leads to a reduction in stroke volume and an increase in heart rate [28] as well as a reduction in skin blood flow [26]. In addition, a reduction in body water leads to a reduction in VO_{2max} with greater losses resulting in greater effects. Blood–brain barrier permeability may be altered [84] and cerebral perfusion reduced [14] in response to hypohydration resulting in a direct effect on the central nervous system. Hypohydration also leads to dryness of the mouth, thirst and headaches which could ultimately affect mood state and psychological factors that alter the perception of effort [78].

A substantial body of evidence therefore exists to suggest that starting exercise in a hypohydrated state or allowing hypohydration to occur during exercise results in an increased perception of effort. The effect of hypohydration on RPE is an important consideration for situations of occupational activity as well as the prescription of physical activity for health and well-being. Consequently, in these situations, individuals should be provided with the opportunity and education to ensure they are adequately hydrated prior to beginning work or an exercise session so that the effect of hydration status on RPE is not negatively influencing the activity in question.

Hydration as Part of a Healthy Lifestyle

As outlined previously, moderate reductions in body water are common as a result of physically active lifestyles, and these reductions may lead to changes in cardiovascular function as well as impairing cognitive processes and mood. These observations are important for physical performance in elite athletes as well as physically active members of the general public and for those with occupations that involve manual work. What is less clear, perhaps, is the effect of acute and chronic reductions in body water on health. The majority of work in this area has focussed on elderly populations.

Few studies have adequately examined the effects of hydration status on chronic disease, but there are some suggestions that a low fluid intake (and, therefore, hypohydration) may be linked to urinary stones (urolithiasis), constipation, bladder and colon cancer, hypertension and diabetic ketoacidosis [46]. These observations should be treated with caution, however, due to the lack of studies demonstrating causality.

The ageing process results in a reduction in fat-free mass, reduction in bone mass and a reduction in total body water as well as a blunted thirst response leading to an increased risk of hypohydration in older individuals. Hydration status is known to be a factor in disease progression for a number of medical disorders. Hypohydration is thought to predispose to infection which, in elderly populations, can be fatal in up to 50% of cases if not diagnosed early [23]. In an analysis of UK death certificates between 2005 and 2009, it was observed that 667 deaths were due to dehydration compared to 157 as a result of malnutrition: It is, however, difficult to determine whether dehydration was the causative factors in these deaths [47].

Leiper et al. [44] observed that water turnover, as assessed via a deuterium oxide tracer, was significantly slower in elderly individuals living in a residential care home than those who lived at home. Similarly, Wolff et al. [86] reported that 12% of elderly patients admitted to hospital from a residential care home were hypernatraemic upon admission compared to 1.3% of age-matched patients admitted from home. The extent of hypernatremia upon admission is related to likelihood of in-hospital mortality [86], and, consequently, patients from residential care homes had a greater likelihood of in-hospital death than those who lived at home. Similar results were reported by El-Sharkawy et al. [20]. These observations indicate that elderly individuals, particularly those in care homes, should be targeted by care workers to ensure adequate water ingestion and avoidance of hypohydration.

Quality of life is of primary importance in elderly individuals and is often assessed as a profile of physical, psychological, independence, social relationships, environmental and spiritual domains. In a study of 82 residents of Australian care homes, Courtney et al. [17] reported that quality of life was positively correlated with clinical care indicators. The three main indicators that affected quality of life were poor hydration status, number of fall incidents and depression. These areas may be targeted to improve quality of life in residential care, and improvements in hydration may be one of the easiest and most cost-effective ways to achieve this.

It seems clear from this evidence that certain populations, such as the elderly, are more susceptible to the impact that reductions in body water can have on health. Further evidence of this can be seen when examining the effects of changes in environmental temperature on morbidity and mortality. Sardon [73] reported that temperatures throughout Europe were several degrees higher between June and September 2003 compared to other years. In an analysis of 2003 heatwave days compared to the years from 1990 to 2004, D'Ippoliti et al. [19] observed that there were 8% more deaths in Munich, 10% more deaths in London, 27% more deaths in Rome and 34% more deaths in Milan. In Mediterranean cities, where the heatwave had the largest impact on temperature, the percentage increase in all-cause mortality compared to other years was greatest in those aged 85+ years and was higher in females than males. Similar observations have been made in Australia. Nitschke et al. [60] analysed daily ambulance transports, hospital admissions and mortality over a 13-year period from 1993 and compared heatwaves to non-heatwave periods. Total mortal was unaffected during heatwaves, but ambulance transports and hospital admissions increased by 4% and 7%, respectively. Total mental health and renal health hospital admissions were increased by 7% and 13%, respectively. Similarly, Khalaj et al. [38] surveyed 1,497,655 emergency admissions to hospital in sites across New South Wales. It was observed that on days of extreme heat, hospital admissions for heat illness, dehydration and electrolyte disturbance were increased, whereas hospital admissions from other causes were not. Individuals with underlying medical conditions affecting the nervous, circulatory, respiratory and/or renal systems were particularly affected by the high environmental temperatures. It seems clear that certain populations, particularly the elderly and those with underlying medical conditions, are most at risk of high environmental temperatures and that this is likely to be at least partly due to the effect on hydration status.

Warren et al. [83] examined the economic impact of hospital admissions due to hypohydration in US elderly adults. Almost 7% of hospitalisations in 1991 had dehydration listed as a diagnosis with 1.4% listed as the primary diagnosis. This resulted in US\$446 million (at 1991 prices) in Medicare claims, suggesting that hypohydration-related hospital admissions have a significant economic impact. From these observations, it seems that the avoidance of hypohydration in these situations is a relatively straightforward but potentially effective intervention in certain populations for improving public health.

Much attention has been given to the acute and chronic effects of hypohydration, but large acute increases in body water, though less common than hypohydration, can also be hazardous. Overhydration can occur as a result of the ingestion of water in excess of the amount needed to maintain euhydration, an electrolyte deficit and/or an inability of the renal system to compensate for these changes by appropriate adjustments of renal function [30]. As with hypohydration, over-hydration is unlikely to lead to serious health consequences in healthy individuals, but occasionally it can be fatal. If the concentration of sodium in the extracellular space falls, water moves from the interstitial space into the cellular compartment, leading to swelling of cells. In most tissues, this is of little consequence, though it may have implications for a number of cellular functions [40]. If sufficiently severe, however, an increase in intra-cranial pressure will result and symptoms associated with "water intoxication", including headache, nausea, confusion and changes in behaviour, may appear. If intra-cranial pressure continues to increase, this can lead to central nervous system dysfunction, coma and death. Exercise-associated hyponatremia, brought about by ingesting a greater volume of water than is lost via sweat, has been associated with several deaths and was reported in 13% of finishers of the Boston marathon in 2002 despite these individuals showing no clinical symptoms [2]. A number of nonexercise cases of "water intoxication", brought on due to excessive fluid ingestion, have also been described. These include cases from fraternity initiation practices, co-ingestion of large volumes of fluid with recreational drugs such as 3,4-methylenedioxymethamphetamine (MDMA), water ingestion during weight loss plans and social competitions involving large volumes of fluid intake [30].

Conclusion

Despite the abundance of water in the human body, it is important to maintain levels within narrow limits. This is achieved by matching water intake and water output. An imbalance between water intake and output leads to either hypohydration or over-hydration. Chronic water imbalances are usually relatively mild: Homeostasis will be restored due to normal regulatory processes. Acute imbalances can, if sufficiently severe, lead to changes in physiological function that can affect performance of physical activity and, in extreme cases, can lead to serious health consequences. Some elite athletes, exercisers and manual workers have been shown to begin activities in a hypohydrated state, even when there is a high risk of potentially harmful hypohydration. Sweat rates and fluid intake during physical activity both vary greatly between individuals. Undertaking physical activity in a hypohydrated state leads to an increase in subjective effort which may have an impact on exercise performance as well as self-selected exercise intensity. Some populations, such as the elderly, are more likely to become hypohydrated, and they, and those who care for them, may need specific hydration advice in order to avoid potential adverse health consequences.

References

- 1. Adolph EF, Barker JP, Hoy PA. Multiple factors in thirst. Am J Physiol. 1954;178:538-62.
- Almond CSD, Shin AY, Fortescue EB, Mannix RC, Wypij D, Binstadt BA, Duncan CN, Olson DP, Salerno AE, Newburger JW, Greenes DS. Hyponatremia among runners in the Boston marathon. New Eng J Med. 2005;352:1550–6.
- Armstrong LE, Costill DL, Fink WJ. Influence of diuretic-induced dehydration on competitive running performance. Med Sci Sports Exerc. 1985;17:456–61.
- Armstrong LE, Johnson EC, Kunces LJ, Ganio MS, Judelson DA, Kupchak BR, Vingren JL, Munoz CX, Huggins RA, Hydren JR, Moyen NE, Williamson KH. Drinking to thirst versus drinking ad libitum during road cycling. J Athl Train. 2014;49:624–31.

- Baker LB, Dougherty KA, Chow M, Kenney WL. Progressive dehydration causes a progressive decline in basketball skill performance. Med Sci Sports Exerc. 2007;39:1114–23.
- Barr SI, Costill DL, Fink WJ. Fluid replacement during prolonged exercise: effects of water, saline, or no fluid. Med Sci Sports Exerc. 1991;23(7):811–7.
- Bates GP, Miller VS, Joubert DM. Hydration status of expatriate manual workers during summer in the middle East. Ann Occup Hyg. 2010;54:137–43.
- 8. Baylis PH. Osmoregulation and control of vasopressin secretion in healthy humans. Am J Physiol. 1987;253:R671-8.
- 9. Below PR, Mora-Rodríguez R, González-Alonso J, Coyle EF. Fluid and carbohydrate ingestion independently improve performance during 1 h of intense exercise. Med Sci Sports Exerc. 1995;27:200–10.
- Biggs C, Paterson M, Maunder E. Hydration status of South African forestry workers harvesting trees in autumn and winter. Ann Occup Hyg. 2011;55:6–15.
- 11. Borg GAV. Psychophysical bases of perceived exertion. Med Sci Sports Exerc. 1982;14:377-81.
- Bouchard C, Blair SN, Katzmarzyk PT. Less sitting, more physical activity, or higher fitness. Mayo Clin Proc. 2015;90:1533–40.
- Brake R. Fluid consumption sweat rates and hydration status of thermally-stressed underground miners and the implications for heat illness and shortened shifts. 2001. Available at: http://www.qrc.org.au/conference/_dbase_ upl/2001_spk024_brake.pdf. Accessed 7 Mar 2016.
- Carter III R, Cheuvront SN, Vernieuw CR, Sawka MN. Hypohydration and prior heat stress exacerbates decreases in cerebral blood flow velocity during standing. J Appl Physiol. 2006;101:1744–50.
- Cheuvront SN, Carter III R, Castellani JW, Sawka MN. Hypohydration impairs endurance exercise performance in temperate but not cold air. J Appl Physiol. 2005;99:1972–6.
- Cheuvront SN, Kenefick RW. Dehydration: physiology, assessment, and performance effects. Compr Physiol. 2014;4:257–85.
- Courtney M, O'Reilly M, Edwards H, Hassall S. The relationship between clinical outcomes and quality of life for residents of aged care facilities. Aust J Adv Nurs. 2009;26:49–57.
- Cunniffe B, Fallan C, Yau A, Evans GH, Cardinale M. Assessment of physical demands and fluid balance in elite female handball players during a 6-day competitive tournament. Int J Sport Nutr Exerc Metab. 2015;25:78–88.
- 19. D'Ippoliti D, Michelozzi P, Marino C, de'Donato F, Menne B, Katsouyanni K, Kirchmayer U, Analitis A, Medina-Ramon M, Paldy A, Atkinson R, Kovats S, Bisanti L, Schneider A, Lefranc A, Iniguez C, Perucci CA. The impact of heat waves on mortality in 9 European cities: results from the EuroHEAT project. Env Health. 2010; 9:37.
- El-Sharkawy AM, Watson P, Neal KR, Ljungqvist O, Maughan RJ, Sahota O, Lobo DN. Hydration and outcome in older patients admitted to hospital (the HOOP prospective cohort study). Age Ageing. 2015;44:943–7.
- European Food Safety Authority (EFSA) Panel on Dietetic Products. Nutrition, and Allergies (NDA): scientific opinion on dietary reference values for water. EFSA J. 2010;8:1459–507.
- Evans GH, Shirreffs SM, Maughan RJ. Post-exercise rehydration in man: the effects of osmolality and carbohydrate content of ingested drinks. Nutrition. 2009;25:905–13.
- 23. Ferry M. Strategies for ensuring good hydration in the elderly. Nutr Rev. 2005;63:S22-9.
- 24. Fleming J, James LJ. Repeated familiarisation with hypohydration attenuates the performance decrement caused by hypohydration during treadmill running. Appl Physiol Nutr Metab. 2014;39:124–9.
- 25. Ganio MS, Armstrong LE, Casa DJ, McDermott BP, Lee EC, Yamamoto LM, Marzano S, Lopez RM, Jimenez L, Le Bellego L, Chevillotte E, Lieberman HR. Mild dehydration impairs cognitive performance and mood of men. Br J Nutr. 2011;106:1535–43.
- Gonzalez-Alonso J, Calbet JA, Nielsen B. Muscle blood flow is reduced with dehydration during prolonged exercise in humans. J Physiol. 1998;513:895–905.
- Gonzalez-Alonso J, Mora-Rodriguez R, Below PR, Coyle EF. Dehydration reduces cardiac output and increases systemic and cutaneous vascular resistance during exercise. J Appl Physiol. 1995;79:1487–96.
- Gonzalez-Alonso J, Mora-Rodriguez R, Below PR, Coyle EF. Dehydration markedly impairs cardiovascular function in hyperthermic endurance athletes during exercise. J Appl Physiol. 1997;82:1229–36.
- 29. Greenleaf JE. Problem: thirst, drinking behavior, and involuntary dehydration. Med Sci Sports Exerc. 1992;24: 645–56.
- 30. Hew-Butler T, Rosner MH, Fowkes-Godek S, et al. Statement of the 3rd international exercise-associated hyponatremia consensus development conference, Carlsbad, California, 2015. Clin J Sports Med. 2015;25:303–20.
- 31. Institute of Medicine. Panel on dietary reference intakes for electrolytes and water, dietary reference intakes for water, potassium, sodium, chloride and sulfate. Washington, DC: The National Academies Press; 2005.
- 32. Ishijima T, Hashimoto H, Satou K, Muraoka I, Suzuki K, Hiquchi M. The different effects of fluid with and without carbohydrate ingestion on subjective responses of untrained men during prolonged exercise in a hot environment. J Nutr Sci Viaminol. 2009;55:506–10.
- James LJ, Clayton D, Evans GH. Effect of milk protein addition to a carbohydrate-electrolyte solution ingested after exercise in the heat. Br J Nutr. 2011;105:393–9.

- 34. Jeukendrup AE, McLaughlin J. Carbohydrate ingestion during exercise: effects on performance, training adaptations and trainability of the gut. In: Maughan RJ, Burke LM, editors. Sports nutrition: more than just calories – triggers for adaptation. Nestle Nutrition Institute Workshop Series. 2011;69:1–17.
- Judelson DA, Maresh CM, Anderson JM, Armstrong LE, Casa DJ, Kraemer WJ, Volek JS. Hydration and muscular performance – does fluid balance effect strength, power and high-intensity endurance? Sports Med. 2007;37:907–21.
- Kenefick RW, Cheuvront SN, Leon LR, O'Brien KK. Dehydration and rehydration. In: Auerbach PS, editor. Wilderness medicine. USA: Elsevier; 2012. p. 1393–404.
- Kenefick RW, O'Moore KM, Mahood NV, Castellani JW. Rapid IV versus oral rehydration: responses to subsequent exercise heat stress. Med Sci Sports Exerc. 2006;38:2125–31.
- Khalaj B, Lloyd G, Sheppeard V, Dear K. The health impacts of heat waves in five regions of New South Wales, Australia: a case-only analysis. Int Arch Occup Envrion Health. 2010;83:833–42.
- Kovacs EMR, Schmahl RM, Senden LMG, Brouns F. Effect of high and low rates of fluid intake on post-exercise rehydration. Int J Sports Nutr Exerc Metab. 2002;12:14–23.
- 40. Lang F. Mechanisms and significance of cell volume regulation. J Am Coll Nutr. 2007;26(5 Suppl):613S-23.
- Leiper JB, Carnie A, Maughan RJ. Water turnover rates in sedentary and exercising middle aged men. Br J Sports Med. 1996;30:24–6.
- 42. Leiper JB, Broad NP, Maughan RJ. Effect of intermittent high-intensity exercise on gastric emptying in man. Med Sci Sports Exerc. 2001;33:1270–8.
- Leiper JB, Pitsiladis Y, Maughan RJ. Comparison of water turnover rates in men undertaking prolonged cycling exercise and sedentary men. Int J Sports Med. 2001;22:181–5.
- Leiper JB, Primrose CS, Primrose WR, Phillimore J, Maughan RJ. A comparison of water turnover in older people in community and institutional settings. J Nutr Health Aging. 2005;9:189–93.
- Lindseth PD, Lindseth GN, Petros TV, Jensen WC, Caspers J. Effect of hydration on cognitive function in pilots. Mil Med. 2013;178:792–8.
- 46. Manz F, Wentz A. The importance of good hydration for the prevention of chronic diseases. Nutr Rev. 2005;63:S2–5.
- 47. Maughan RJ. Hydration, morbidity and mortality in vulnerable populations. Nutr Rev. 2012;70:S152-5.
- Maughan RJ, Leiper JB, Shirreffs SM. Restoration of fluid balance after exercise-induced dehydration: effects of food and fluid intake. Eur J Appl Physiol Occup Physiol. 1996;73:317–25.
- Maughan RJ, Merson SJ, Broad NP, Shirreffs SM. Fluid and electrolyte intake and loss in elite soccer players during training. Int J Sport Nutr Exerc Metab. 2004;14:333–46.
- Maughan RJ, Shirreffs SM. Development of individual hydration strategies for athletes. Int J Sports Nutr Exerc Metab. 2008;18:457–72.
- Maughan RJ, Shirreffs SM, Merson SJ, Horswill CA. Fluid and electrolyte balance in elite male football (soccer) players training in a cool environment. J Sports Sci. 2005;23:73–9.
- Maughan RJ, Watson P, Evans GH, Broad N, Shirreffs SM. Water balance and salt losses in competitive football. Int J Sport Nutr Ex Metab. 2007;17:583–94.
- Mears SA, Shirreffs SM. Assessing hydration status and reported beverage intake in the workplace. Am J Lifestyle Med. 2015;9:157–68.
- McConell GK, Burge CM, Skinner SL, Hargreaves M. Influence of ingested fluid volume on physiological responses during prolonged exercise. Acta Physiol Scand. 1997;160:149–56.
- 55. Miller VS, Bates GP. The thermal work limit is a simple reliable heat index for the protection of workers in thermally stressful environments. Ann Occup Hyg. 2007;51:553–61.
- 56. Miller VS, Bates GP. Hydration, hydration, hydration. Ann Occup Hyg. 2010;54:134-6.
- Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. J Appl Physiol. 1992;73:1340–50.
- Murray SR, Michael TJ, McClellan PD. The influence of fluid replacement rate on heart rate and RPE during exercise in a hot, humid environment. J Strength Cond Res. 1995;9:251–4.
- Nielsen B, Sjogaard G, Bondepettersen F. Cardiovascular, hormonal and body-fluid changes during prolonged exercise. Eur J Appl Physiol Occup Physiol. 1984;53:63–70.
- 60. Nitschke M, Tucker G, Bi P. Morbidity and mortality during heatwaves in metropolitan Adelaide. Med J Aust. 2007;187:662–5.
- 61. Noakes TD. Commentary: role of hydration in health and exercise. BMJ. 2012;345:e4171.
- 62. Noakes T, Mekler J, Pedoe DT. Jim Peters' collapse in the 1954 Vancouver Empire Games marathon. S Afr Med J. 2008;98:596–600.
- 63. Noakes TD, Sharwood K, Collins M, Perkins DR. The dipsomania of great distance: water intoxication in an Ironman triathlete. Br J Sports Med. 2004;38:E16.
- 64. Nose H, Mack GW, Shi XR, Nadel ER. Role of osmolality and plasma volume during rehydration in humans. J Appl Physiol. 1988;65:325–31.

- 65. Nybo L, Jensen T, Nielsen B, Gonzalez-Alonso J. Effects of marked hyperthermia with and without dehydration on VO2 kinetics during intense exercise. J Appl Physiol. 2001;90:1057–64.
- 66. Polkinghorne BG, Gopaldasani V, Furber S, Davies B, Flood VM. Hydration status of underground miners in a temperate Australian region. BMC Public Health. 2013;13:426.
- Riebe D, Maresh CM, Armstrong LE, Kenefick RW, Castellani JW, Echegaray ME, Clark BA, Camaione DN. Effects of oral and intravenous rehydration on ratings of perceived exertion and thirst. Med Sci Sports Exerc. 1997;29: 117–24.
- Roberts DF, Donnelly S. Fluid balance and sweat rates during strenuous physical labour by timber harvesters. Med Sci Sports Exerc. 2006;38:S173–4.
- 69. Robertson GL. Vasopressin in osmotic regulation in man. Annu Rev Med. 1974;25:315–22.
- Robertson R, Noble BJ. Perception of physical exertion: methods, mediators and application. Exerc Sport Sci Res. 1997;25:407–52.
- 71. Rolls BJ, Wood RJ, Rolls ET, Lind H, Lind W, Ledingham JGG. Thirst following water deprivation in humans. Am J Physiol. 1980;239:R476–82.
- Roy BD, Green HJ, Burnett ME. Prolonged exercise following diuretic-induced hypohydration: effects on cardiovascular and thermals strain. Can J Physiol Pharmacol. 2000;78:541–7.
- 73. Sardon JP. The 2003 heat wave. Euro Surveill. 2007;12:226.
- Saunders CJ, de Milander L, Hew-Butler T, Xenophontos SL, Cariolou MA, Anastassiades LC, Noakes TD, Collins M. Dipsogenic genes associated with weight changes during Ironman triathlons. Hum Mol Genet. 2006;15: 2980–7.
- 75. Savoie FA, Kenefick RW, Ely BR, Cheuvront SN, Goulet EDB. Effect of hypohydration on muscle endurance, strength, anaerobic power and capacity and vertical jumping ability: a meta-analysis. Sports Med. 2015;45: 1207–27.
- 76. Shimamoto H, Komiya S. The turnover of body water as an indicator of health. J Physiol Anthropol Appl Human Sci. 2000;19:207–12.
- Shirreffs SM, Maughan RJ. Volume repletion following exercise-induced volume depletion in man: replacement of water and sodium losses. Am J Physiol. 1998;43:F868–75.
- Shirreffs SM, Merson SJ, Fraser SM, Archer DT. The effects of fluid restriction on hydration status and subjective feelings in man. Br J Nutr. 2004;91:951–8.
- Shirreffs SM, Taylor AJ, Leiper JB, Maughan RJ. Post-exercise rehydration in man: effects of volume consumed and drink sodium content. Med Sci Sports Exerc. 1996;28:1260–71.
- St Clair Gibson A, Baden DA, Lambert MI, Lambert V, Harley YXR, Hampson D, Russell VA, Noakes TD. The conscious perception of the sensation of fatigue. Sports Med. 2003;33:167–76.
- Vist GE, Maughan RJ. Gastric emptying of ingested solutions in man: effect of beverage glucose concentration. Med Sci Sports Exerc. 1994;26:1269–73.
- Wang XW, Lyles MF, You TJ, Berry MJ, Rejeski WJ, Nicklas BJ. Weight regain is related to decreases in physical activity during weight loss. Med Sci Sports Exerc. 2008;40:1781–8.
- Warren JL, Bacon WE, Harris T, McBean AM, Foley DJ. The burden and outcomes associated with dehydration among US elderly, 1991. Am J Public Health. 1994;84:1265–9.
- Watson P, Black KE, Clark SC, Maughan RJ. Exercise in the heat: effect of fluid ingestion on blood-brain barrier permeability. Med Sci Sports Exerc. 2006;42:2197–204.
- 85. Watson P, Whale A, Mears SA, Reyner LA, Maughan RJ. Mild hypohydration increases the frequency of driver errors during a prolonged, monotonous driving task. Physiol Behav. 2015;147:313–8.
- Wolff A, Stuckler D, McKee M. Are patients admitted to hospitals from care homes dehydrated? A retrospective analysis of hypernatremia and in-hospital mortality. J R Soc Med. 2015;108:259–65.
- Wu T, Gao X, Chen M, van Dam RM. Long-term effectiveness of diet plus exercise interventions vs diet only interventions for weight loss: a meta-analysis. Obes Rev. 2009;10:313–23.
- 88. Yau MWA, Moss A, James LJ, Gilmore W, Ashworth JJ, Evans GH. The influence of angiotensin converting enzyme and bradykinin B2 receptor gene variants on voluntary fluid intake and fluid balance in healthy men during moderate intensity exercise in the heat. Appl Physiol Nutr Metab. 2015;40:184–90.

Part V Nutrition in Specialized Populations and Conditions

Chapter 16 Nutrition for a Healthy Pregnancy

Laurie Tansman

Key Points

- Obesity presents challenges to a healthy pregnancy
- Preconception care includes nutrition strategies to prepare for a health pregnancy
- · Adequacy of calcium, folic acid, iodine, iron and magnesium intake
- Fish intake is important
- Abstinence from alcohol intake is not 100%
- Diabetes diagnosed in the first trimester is considered to be pre-existing Type II diabetes

Keywords Calcium • Diabetes • Folic acid • Fish • Hypertensive disorders • Iron • Listeria • Obesity • Weight gain

Introduction

Pregnancy in the twenty-first century is challenging because the landscape of the pregnant population over the past 50 years has changed. To begin with, many women entering pregnancy today have a weight problem, namely obesity. Because of this, more women are entering pregnancy with Type II diabetes and hypertension. Then, more women are having children at an older age, many because they were previously concentrating on their careers. With the older pregnant woman, you have increased pregnancy-related risks. Next, because of advances in medicine, more women are entering pregnancy with a history of chronic medical problems, and it is not just women with Type I diabetes or epilepsy but those with a history of cancer, multiple sclerosis and organ transplants such as renal and heart. In a review article on pregnancy in renal transplant recipients, it was stated that "pregnancies in transplant recipients are now considered commonplace" referring to the numbers

L. Tansman, MS, RD, CDN

e-mail: laurie.tansman@mountsinai.org

Department of Clinical Nutrition, The Mount Sinai Hospital, New York, NY, USA

Department of Environmental Medicine and Public Health, Icahn School of Medicine at Mount Sinai, New York, NY, USA

of such women who become pregnant [1]. Finally, women who have benefitted from advanced fertility technologies are at risk for pregnancy-related problems too. Pregnancy has thus become very complicated.

According to the Centers for Disease Control and Prevention (CDC) publication, At A Glance 2015, Safe Motherhood, since the establishment of the Pregnancy Mortality Surveillance System in 1986, reported pregnancy-related deaths have increased from 7 per 10,000 live births in 1987 to almost 17 deaths per 100,000 live births in 2010. Furthermore, the incidence of severe maternal morbidity (SMM) more than doubled during the first decade of this century with a reported 65,000 women having serious health complications as a result of pregnancy during 2010–2011 [2].

In the 2015 (16th annual) State of the World's Mothers report, the latest data from 179 countries (46 developed nations and 133 developing nations) were culled together to assess the well-being of mothers and children. These were countries that had a 2014 population over 100,000. The ranking for the United States was 33 in the *Mothers' Index.* "In the United Sates, women face a 1 in 1,800 risk of maternal death. This is the worst performance of any developed country in the world. A woman in the U.S. is more than 10 times as likely as a woman in Austria, Belarus or Poland to eventually die from a pregnancy-related cause" [3]. The index takes into account maternal health (lifetime risk of maternal health), children's well-being (under-5 mortality rate), educational status, economic status (gross national income per capita) and political status (women in national government). Sixty-one nations have a lower maternal death rate compared to the United States which ties with Armenia, Azerbaijan, China and Grenada. Forty-two nations had a lower under-5 mortality rate compared to the United States.

Explanations for this disheartening data are unclear. But because of these statistics, it is important that a woman entering pregnancy be in the best possible health. Nutrition can "play" a vital role to help achieve this goal, resulting in improved maternal and fetal outcomes [4].

Therefore, this chapter is really about how to achieve the healthiest pregnancy possible with regards to nutrition, beginning with preconception nutrition, addressing nutrition needs during pregnancy and including nutrition-related challenges that occur along the way during the 9 months.

Preconception

More attention is being paid to preconception care because so many women are entering pregnancy with risk factors that can contribute to adverse maternal and fetal outcomes [5]. Being in optimal nutrition health can help a woman prepare for a healthy pregnancy. The challenge is that because many pregnancies are unintended, 45% in 2011 [6], sexually active women in their reproductive years should consider the following nutrition-related recommendations from the CDC [7]:

- 400 µg of folic acid daily
- Reach and maintain a healthy weight
- Limit alcohol intake to less than seven drinks a week and never more than one drink on any one occasion. *If planning for pregnancy, stop drinking alcohol.*

Women should also be careful of taking preformed vitamin A supplements because of the possibility of teratogenic effects, specifically during the first trimester when a woman may not yet know she is pregnant, and especially because the pregnancy may be unintended [8].

An integral part of preconception care is to achieve and maintain a well-balanced diet.

According to the most recently released 2015–2020 Dietary Guidelines for Americans in January 2016, a healthy eating pattern includes fruits, vegetables, protein, dairy, grains and oils while limiting saturated fats, trans fats and sodium [9].

Weighty Issues

Obesity

risk of infertility. Weight loss can improve fertility [10]. For the obese woman who undergoes fertility treatments, weight loss is also recommended [11].

The American College of Obstetricians and Gynecologists (ACOG) more than a decade ago recommended that obstetricians provide preconception counseling to their obese patients to lose weight before attempting pregnancy [11] because of increased risks that include miscarriage, gestational diabetes (GDM), pregnancy-induced hypertension (PIH) disorders, venous thromboembolism, dystocia as well as caesarian section [12–15]. For the developing fetus of an obese mother, there is increased risk for birth defects, insulin resistance, preterm birth, macrosomia, increased risk for obesity and premature mortality in adulthood [12, 14, 16, 17]. In fact, obesity is such a concern that the ACOG has an obesity toolkit to assist obstetricians–gynecologists to help their patients improve their weight including lifestyle therapies [18].

To address obesity and that may be planned long before a woman becomes pregnant, bariatric surgery may be performed. As the treatment of classes 2 and 3 obesity with bariatric surgery increases and the age at which bariatric surgery is being undertaken is earlier in life, more data are surfacing on the outcomes of pregnancy after such surgery. Recently reported concerns related to micronutrient deficiencies were "weak and inconclusive" [19]. Two studies from Sweden both identified that women who had bariatric surgery had shorter gestation and an increased risk for small-for-gestational (SGA) infants [20, 21]. The former study indicated that in those who had bariatric surgery also had a reduced risk of GDM and excessive fetal growth.

Gestational Weight Gain

In 2009, the Institute of Medicine (IOM) of the National Academies updated their guidelines for maternal weight gain for a singleton pregnancy as well as provisional weight gain for twins (Table 16.1) [22]. When addressing gestational weight gain, concerns identified with obese women can also occur in the normal-weight woman who gains too much weight, particularly GDM and preeclampsia [23]. Also, amount of gestational weight gain in the normal-weight woman can be associated with childhood overweight for those who gain too much as well as too little weight [23].

For women pregnant with three or more fetuses, there are no IOM weight-gain guidelines. The best guidance is to at least gain the uppermost value in the range recommended for twins. The ultrasound growth scan that provides the estimated fetal weight (EFW) along with the growth percentile is probably the best data to judge adequacy of weight gain.

Pregravid weight	Body mass index	Singleton pregnancy recommended range of total weight gain (lb)	Twin pregnancy recommended range of total weight gain (lb) (provisional)
Underweight	<18.5	28–40	Not available
Normal weight	18.5-24.9	25–35	37–54
Overweight	25-29.9	15–25	31–50
Obese (all classes)	≥30	11–20	25-42

Table 16.1 IOM recommendations for maternal weight gain during pregnancy

Calorie and Protein Needs

Although many think that during pregnancy a woman is eating for two, this does not translate into double the amount of calories. Currently, the IOM only makes recommendations for women with a singleton pregnancy (Table 16.2).

Micronutrients

Just as Dietary Reference Intakes (DRIs) were released for calories and protein, the release of the DRIs for the micronutrients has included recommendations for pregnancy based upon a singleton pregnancy. Table 16.3 provides a summary of the Recommended Dietary Allowances (RDAs) for the micronutrients discussed in this section as well as subsequent sections.

So how does a woman know about and achieve adequate micronutrient intake? First, as of May 20, 2016, a revised Nutrition Facts food label was unveiled by the FDA [28]. A significant improvement will be that besides listing the % Daily Value (DV), the actual amount of calcium and iron (as well as vitamin D and potassium) will be listed. For foods that are good sources of other micronutrients, it is unclear if the actual amount will be listed besides the already %DV that may appear on some food labels. This revision will be a great tool to help a woman achieve optimal intake of some of these micronutrients, provided her healthcare provider educates her about her micronutrient needs. Second, one of the easiest ways to achieve an optimal micronutrient intake besides a well-balanced diet is to include at least one serving daily of a fortified cold breakfast cereal. Finally, for a comprehensive analysis of most foods and for which the nutrient information is not available on the Nutrition Facts label, it can be recommended to go to the US Department of Agriculture's (USDA) SuperTracker website that has a Food-A-Pedia [29] where the nutrient analysis of over 8,000 foods is comprehensive, except for iodine. Limited information for the iodine content of foods is available from the Office of Dietary Supplements (ODS) [30].

Folate

Folate is a B vitamin that is important in pregnancy for multiple reasons including red blood cell formation and cell division. Adequate intake prior to conception and especially during the first trimester is important to decrease the incidence of neural tube defects (NTD). Because the bioavailability of

Trimester	Additional daily calories	Additional daily protein (g)	
First	None	None	
Second	340	25	
Third	452	25	

Table 16.2 IOM recommendations for additional calorie and protein needs for a singleton pregnancy [24]

Table 16.3 Sel	ect RDAs for	micronutrients	for a singleton	pregnancy [8.	25 - 271	
----------------	--------------	----------------	-----------------	-------------	----	----------	--

Life stage (years)	Calcium (mg/d)	Folate (µg/d) ^a	Iodine (μg/d)	Iron (mg/d)	Magnesium (mg/d)	Vitamin C (mg/d)
14–18	1,300	600	220	27	400	80
19–30	1,000	600	220	27	350	85
31-50	1,000	600	220	27	360	85

^aAs dietary folate equivalents

folate from food is nearly half that of synthetic folic acid, it is recommended that women of childbearing age who are sexually active meet the RDA for folate from folic acid found in fortified foods and supplements. This is the reason for the recommendation to take 400 μ g/day of folic acid. When a woman enters pregnancy, the RDA for folate increases by 50%.

The fortification of foods with folic acid that became mandatory in the United States by January 1, 1998, was intended to reduce the incidence of NTDs. What was interesting is that this FDA requirement provided only for the fortification of those grain products that were enriched. Therefore, whole grain products may not be fortified with folic acid such as some of the popular whole grain organic cereals that the health-conscious young woman may often purchase over usual supermarket brands. And the breads they purchase may often be fresh ground whole grain artisanal breads that have not been fortified with folic acid. What is interesting is that since the release of the 1995 DGAs, consumption of whole grains is encouraged. Thus, the female for whom the mandatory folic acid fortification was intended and who consumes only whole grains that may not be fortified with folic acid may therefore not be consuming adequate folic acid. With the recent release of the 2015–2020 DGAs [9], it states in the full report that at least half of grains consumed be whole grain and then it is stated:

Those who consume all of their grains as whole grains should include some grains, such as whole-grain readyto-eat breakfast cereals, that have been fortified with folic acid. This is particularly important for women who are capable of becoming pregnant...

Finally, mandatory fortification of certain grains with folic acid has been controversial, particularly because of the concern that an increased intake might mask vitamin B12 deficiency [31]. As a matter of fact, there are many countries that do not have this mandatory requirement including none in the European Union, although the United Kingdom continues to consider this fortification. Now, here is where things get interesting. At a symposium in 2006 in Wageningen, the Netherlands, that was attended by mostly Dutch scientists, the attendees were asked to identify the greatest nutrition discoveries and challenges from 1976 to 2006 [32]. And guess what? The number one discovery since 1976 was folic acid prevents birth defects!

Iodine

To assure that the pregnant mother consumes adequate iodine, the American Thyroid Association recommends that such women consume 250 μ g iodine daily from diet and supplements with 150 μ g iodine coming from a supplement such as the prenatal vitamin [33]. While at one time many prenatal vitamins did not contain this mineral, the American Thyroid Association now recommends that all contain 150 μ g of iodine.

Iron

Iron deficiency anemia is considered to be the number one nutritional deficiency worldwide, and pregnant women are one of the high-risk populations [34, 35]. Anemia during pregnancy can contribute to poor gestational weight gain with low birth weight, preterm birth, neuro-behavioral and cognitive deficits [36]. To prevent deficiency, oral intake from fortified foods and supplements is suggested [37]. Most prenatal vitamin preparations have iron included. When additional supplementation is needed, ascorbic acid is often prescribed to optimize absorption [35].

Although pregnant women are among the most vulnerable populations to have iron-deficient anemia, the United States Preventive Services Task Force (USPSTF) recently released a Recommendation Statement [37] regarding the screening for iron deficiency anemia and iron supplementation to improve maternal health and birth outcomes. The recommendations are as follows:

The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of screening for iron deficiency anemia in pregnant women to prevent adverse maternal health and birth outcomes (I statement)

The USPSTF concludes that the current evidence is insufficient to assess the balance of benefits and harms of routine iron supplementation for pregnant women to prevent adverse maternal health and birth outcomes (I statement)

Regarding screening, the inadequate evidence for screening for iron-deficient anemia pertains to *asymptomatic* pregnant women as elaborated upon in the paper.

Regarding treatment, the recommendation made was based on the fact that no recent studies "were generalizable to the general US population." It was pointed out that there is a "critical gap in the evidence [37]."

Considerations for Specific Populations

Nutrition for Twins and Beyond

Unfortunately, there are minimal *official* recommendations available such as from the IOM. This can be very challenging when trying to help a woman pregnant with twins or more. Luke [38, 39] as well as Goodnight and Newman [40] have provided much valuable information.

Probably the best way to judge if a woman pregnant with twins or more is eating enough calories and protein is by trending weight gain and the ultrasound growth scan. It has been recommended that in the first trimester, it may be optimal to consume an additional 500 calories daily because of the increased probability that such pregnancies will result in preterm births [41].

Finally, while a woman pregnant with two or more presumably has increased micronutrients needs too, it is probably best to not exceed the Tolerable Upper Intake Levels (UL) for the discussed micronutrients in this chapter unless there is a need for therapeutic doses (Table 16.4).

Vegans

If a vegan woman does not have any identified nutrition deficiencies prior to becoming pregnant, she will probably be able to obtain adequate nutrition during pregnancy to assure optimal fetal growth resulting in a healthy baby. Today, many plant-based foods are fortified with a wide range of micronutrients that may be of concern in the vegan diet, especially vitamin B12. Probably the biggest challenge is adequate protein intake, especially if pregnant with twins or more. *Author's personal*

Life stage (years)	Calcium (mg/d)	Folate (µg/d) ^a	Iodine (µg/d)	Iron (mg/d)	Magnesium (mg/d) ^b	Vitamin C (mg/d)
14–18	3,000	800	900	45	350	1,800
19–30	2,500	1,000	1,100	45	350	2,000
31-50	2,500	1,000	1,100	45	350	2,000

Table 16.4UL for select micronutrients [8, 25–27]

^aAs dietary folate equivalents

^bFrom pharmacologic agents only

comment: Based upon personal experience, when a vegan is pregnant with more than one, she many times will choose to add some animal protein to her diet, such as whey protein powder added to a smoothie or choosing to eat fish.

Food Safety

There are several government websites that have guidelines for food safety during pregnancy. One website, in particular, Foodsafety.gov states on its homepage: Your Gateway to Food Safety Information. This is a collaborative effort from federal government agencies, such as the CDC, FDA and USDA. The materials for the pregnant woman are excellent, including "Checklist of Food to Avoid during Pregnancy" [42].

Foodborne Pathogens

Campylobacter, E. Coli, Listeria monocytogenes (hereafter, referred to as listeria), Toxoplasma gondii and Salmonella are considered the top five foodborne pathogens. While each of these can have severe consequences for both the mother and fetus, only listeria will be discussed here as the pregnant woman is reportedly ten times more likely to get listeria infection compared to the general public. Infection with listeria can increase risk for miscarriage, preterm labor, low birth weight and infant death. Furthermore, the baby who is infected *in utero* with listeria may develop problems later in life, including intellectual disability, paralysis, seizures, blindness and impairment of the brain, heart and kidney according to Foodsafety.gov.

To prevent listeria infection, the pregnant woman should avoid:

- · Raw fish.
- Refrigerated smoked fish unless reheated to 165 °F smoked fish that is canned, shelf stable or part of a casserole or other cooked dish is permissible.
- Unpasteurized milk and soft cheeses made from such milk including Brie, Camembert, Feta, Roquefort, quesco balance and quesco fresco. Such soft cheeses are permissible if the label indicates that they were made from pasteurized milk.
- Luncheon meats and frankfurters unless they are reheated until steaming hot or 165 °F.
- Premade meat or seafood salad such as chicken or shrimp salad. These are OK to eat if prepared at home following food safety basics.
- Refrigerated pates or meat spreads. Canned versions are acceptable.

In addition to the above recommendations, listeria can be found in many other foods, although this is because of an accidental contamination. During the writing of this chapter in 2016, a multistate outbreak (limited as of May 2016 to California, Maryland and Washington) was identified in March 2016 and that is currently being linked to packaged frozen vegetable products manufactured or processed at the CRF Frozen Foods plant in Pasco, Washington [43]. Approximately 358 consumer products sold under 42 brand names are involved in the voluntary recall that is precautionary to contain the outbreak. Fortunately, there have only been two reported cases of listeriosis in 2016 as of the end of May that are related to this outbreak and that were in older adults, not pregnant women.

So what is a pregnant woman to do to stay abreast of food recalls that may pose a risk to herself and unborn child? If she has an e-mail address, she can sign up for automatic notification of recalls and alerts at the FDA or Foodsafety.gov websites [44, 45].

Methylmercury

Found in fish, a high intake of methylmercury can be harmful to brain development in the fetus through childhood. Yet, fish intake is a key source of docosahexaenoic acid (DHA) that is important in the neurologic development of infants [46, 47]. Because of the concern about methylmercury, there has been a lot of confusion about what to do and that may be a contributing factor to inadequate fish intake in the pregnant population [48]. This prompted the FDA and Environmental Protection Agency (EPA) to issue a draft of updated advice for the pregnant woman [49]. So here are the facts as per that most recent draft of advice by the FDA and EPA:

Avoid: tilefish (from the Gulf of Mexico), shark, swordfish and king mackerel

Limit: white (albacore) tuna intake to 6 ounces weekly

Eat: a variety of low-mercury fish up to 12 ounces weekly. This includes salmon, shrimp, pollack, light tuna, tilapia, catfish and cod.

Compounding the confusion surrounding the methylmercury content of fish is the concern of mislabeling of fish that may unknowingly lead to an intake of high-mercury fish. In a 2013 report released by Oceana, tilefish was sold as halibut and red snapper in a store in New York City, and king mackerel was sold as grouper in a store in South Florida [50]. It is therefore understandable that pregnant women may be confused and a contributing reason to why some may be avoiding fish, preferring fish oil supplements as an alternative to get an adequate intake of DHA [48].

Lead

While many food safety materials geared for the pregnant woman do not address lead exposure, this can be a serious concern increasing risk for miscarriage, preterm birth and SGA age [51, 52]. Furthermore, lead exposure *in utero* can have harmful effects on neurodevelopment in childhood [51–53]. The CDC has a comprehensive publication addressing lead exposure targeted to the professional audience [51] as well as a handout for the pregnant woman [53].

Strategies to reduce lead exposure include:

- Optimizing calcium, iron and vitamin C intake can decrease lead absorption
- · Avoid using imported lead-glazed ceramic pottery
- · Avoid leaded crystal for serving or storing beverages

More detailed information is available in the CDC publication for the professional.

Another source of lead as per the CDC can be from contaminated soil and water [51]. This was a food safety concern that came to public attention in 2015 during the initial writing of this chapter that occurred in Flint, Michigan [54]. The problem is so severe that residents have to go to local fire stations to retrieve bottled water for drinking and cooking. Some residents are so concerned that they also use bottle water for everything from dishwashing to laundering and daily self-bathing [55].

Non-nutritive Substances

Alcohol

It is a *sine qua non* that pregnant women should abstain from alcohol. As stated earlier in this chapter, women planning pregnancy should also abstain from alcohol. In a recent report released from the American Academy of Pediatrics, "Prenatal exposure to alcohol can damage the developing fetus and is the leading preventable cause of birth effects and intellectual and neurodevelopmental disabilities"

[56]. Yet, there are women who still do not abstain [57]. In fact, it has been reported that during a 2011–2013 study, 1 in 10 women drank alcohol during pregnancy during the prior 30-day period [58].

Artificial Sweeteners

Controversy reigns regarding the safe use of such sweeteners, especially in regards to preterm labor [59–61]. In a Position Paper from the Academy of Nutrition and Dietetics, nothing is said one way or the other, except that there is limited research to address the safety of such sweeteners [62]. So, it is understandable when the risks for preterm delivery are high that the pregnant woman may sometimes be told to avoid such sweeteners.

Caffeine

Another controversial topic. However, in August 2010, the ACOG released a Committee Opinion, "Moderate caffeine consumption (less than 200 mg per day) does not appear to be a major contributing factor in miscarriage or preterm birth" [63]. Prior research addressed the concern of caffeine as a teratogen and that there was no evidence of a correlation [64].

Nutrition-Related Complaints During Pregnancy

Nausea and Vomiting

The most recent Practice Bulletin from ACOG on nausea and vomiting states that the estimated incidence of nausea and vomiting is 50% and that approximately 25% only experience nausea [65]. The most benign is the woman who may experience nausea upon awakening, hence referred to as "morning sickness." To the extreme is the woman who is intolerant to oral intake, experiencing relentless nausea and vomiting, referred to as hyperemesis gravidarum (HG). Most women probably experience something in the middle of these two extremes.

In the most benign scenario, a woman may be advised to take some plain crackers upon arising in the morning. Ginger snap cookies is another good choice as well as flat ginger ale.

As nausea leads to vomiting, restrictive nutrition interventions may help along with the healthcare provider recommending vitamin B6 or vitamin B6 plus doxylamine as "first-line pharmacotherapy" [65]. Such intervention may include avoiding high-fat and spicy foods, eating solids apart from liquids and sitting up for at least an hour or more after eating. Some women may tolerate cold and room temperature foods best as these do not have the strong odor of warm and hot foods. Often, the more bland the food, the better the tolerance, such as unadorned cold pasta and cold hard-cooked egg whites. Anything ginger, except raw ginger and ginger supplements, has also been suggested [65, 66]. Staying out of the kitchen and, sometimes, no cooking or heating of foods in the home may be helpful too.

For the woman with HG, administration of intravenous fluid (IVF) with dextrose (to provide the RDA for carbohydrates of 175 g/day) and vitamins may be considered [65]. It is further suggested by ACOG that thiamine be administered before IVF with dextrose to prevent Wernicke encephalopathy.

With prolonged HG, consideration needs to be given to nutrition support. While enteral nutrition (EN) support should be first attempted as it poses the least amount of risk to the pregnant woman, this is not always pursued because of the resistance on the part of the woman and that includes discomfort

associated with a feeding tube [66]. A report from almost 20 years ago describes success in using EN support via a naso-gastric tube in a small group of pregnant women [67]. Parenteral nutrition (PN) is only recommended when everything else has failed to treat the pregnant woman with HG [65].

Constipation

This is attributed to the effect of progesterone that slows gut motility during pregnancy. The pressure of the growing fetus(es) against the gut can also contribute to constipation too. Finally, the need for an iron and/or calcium supplement can cause constipation. It is sometimes automatic that when a woman is prescribed an iron supplement that she is also started on a bowel regimen. Diet strategies begin with the avoidance of constipating foods such as bananas [68]. Chocolate has also been suggested to be constipating, although it is doubtful that a woman will want to give up chocolate. Next, increasing intake of fruits and vegetables to provide more fiber may be helpful too. Finally, high-fiber breakfast cereals are often a great solution, sometimes even better than a bowel regimen.

Gastro-Esophageal Reflux (GERD)

This can be very painful for some women, and medication may often need to be prescribed. While this is often due to the pressure of the fetus against the gut, there are nutrition strategies that can be helpful including sitting up for as long as possible after eating. In fact, women with such a complaint should not eat anything for at least 3–4 h prior to going to sleep. Foods to limit include high-fat foods, caffeine, carbonated beverages, chocolate, peppermint, citrus fruits, tomato and tomato products as well as spices. It is also advisable to avoid large meals.

Complications

Gestational Diabetes

At one time, GDM was defined as glucose intolerance identified at any time during pregnancy. However, with so many women entering pregnancy with obesity and thus may have undiagnosed Type II diabetes, this definition was revised several years ago. GDM is diabetes diagnosed in the second or third trimester that is not thought to be Type II diabetes [69]. For example, a pregnant woman with a pregravid BMI of 38 who did not go to seek prenatal care until week 20 and with glucose intolerance would probably be diagnosed with Type II diabetes. Because more pregnant women have diabetes, a new section, Management of Diabetes in Pregnancy, was added to the American Diabetes Association's annual Standards of Medical Care in Diabetes in 2015 and that is published annually as a supplement to the January edition of Diabetes Care.

Current recommendations for the diagnosis of GDM are to test for undiagnosed diabetes at the first prenatal visit in those with risk factors. If a woman is not found to have Type II diabetes at that first prenatal visit or not deemed to be at risk for Type II diabetes, then screening should be done for those women at weeks 24–28 [69]. Risk factors for GDM include prior history of GDM, advanced maternal age, obesity and family history of diabetes [69].

The optimal treatment of any type of diabetes during pregnancy is of critical importance because uncontrolled diabetes during this time includes increased risk for spontaneous abortion, fetal anomalies, preeclampsia, fetal demise, macrosomia and neonatal hypoglycemia [70].

Diet is usually the cornerstone for treatment of Type II diabetes and GDM and, if uncontrolled, may require the use of an oral agent and/or insulin. Diet therapy should be individualized and can range from making one simple diet change such as discontinuing the drinking of fruit juice to multiple changes that may necessitate carbohydrate counting.

GDM often resolves following delivery, although up to 10% may be found to have Type II diabetes [71]. It is currently recommended that an oral glucose tolerance test (OGTT) be administered at the 6- to 12-week postpartum visit to identify if GDM has resolved or converted to Type II diabetes. However, because GDM is a risk factor for diabetes, lifelong screening for diabetes is recommended every 1–3 years depending on risk factors [70].

Hypertensive Disorders of Pregnancy

This can be divided into four sub-types [72, 73]:

Chronic hypertension (cHTN), preeclampsia and eclampsia, preeclampsia superimposed on cHTN and gestational hypertension.

Women with PIH are at increased risk for maternal as well as fetal mortality, preterm birth and intrauterine growth restriction (retardation) (IUGR) [74].

Women who are at increased risk for these disorders are those who exceed gestational weight-gain guidelines, so weight control is an important strategy [72].

Can diet manipulation be helpful? While a moderate restriction of dietary sodium may be indicated, it has further been suggested that adequate intake of calcium and magnesium from dietary sources could reduce the incidence of PIH [74]. This appears to be similar to the foundations of the Dietary Approaches to Stop Hypertension (DASH) diet that was developed, trialed and promoted during the 1990s at the National Heart, Blood and Lung Institute of the National Institutes of Health. Over the years, it has been revised and updated, but the foundation of the diet remains the same: fruits and vegetables as well as non-fat/low-fat dairy products to optimize dietary intake of calcium, potassium and magnesium.

The important role of adequate calcium intake to reduce the severity of preeclampsia for those who have inadequate calcium intake has been recommended by ACOG [73]. In fact, in the American Heart Association/American Stroke Association (ASA) guidelines for the prevention of stroke in women released in 2014, it was recommended that for the prevention of preeclampsia, calcium supplementation of ≥ 1 g/day should be considered for a woman who consumes <600 mg/day of calcium [75].

Conclusion

Pregnancy should be a wonderful time in a woman's life, but depending on her health, it can be punctuated with multiple problems that are sometimes nutrition-related, especially obesity and diabetes. Pregnancy is one of the rare opportunities in life to literally "wipe the slate clean" because a woman is bringing a new life into the world. This presents an optimal time for the healthcare provider to help a woman make positive changes in her diet that will not only impact on her future health but equally as important, the future health of her baby as well as generations to come.

References

- 1. McKay DB, Josephson M. Pregnancy in renal transplant recipients. Dial Transpl. 2010;39:472-5.
- Division of Reproductive. National Center for Chronic Disease Prevention. Centers for Disease Control and Prevention. At A Glance 2015. Safe Motherhood. Advancing the health of mothers in the 21st century. Available at: https://www.cdc.gov/chronicdisease/resources/publications/aag/pdf/2015/safe-motherhood-aag-2015.pdf. Accessed 22 May 2016.
- 3. Save the Children. State of the world's mothers 2015. The urban disadvantage. Fairfield: Save the Children; 2015.
- Stang J, Kent H. Women's health, prenatal nutrition outcomes: a public health perspective. Women Health Rep. 2016;1:3–5.
- 5. Mitchell EW, Verbiest S. Effective strategies for promoting preconception health from research to practice. Am J Health Promot. 2013;27(3 Suppl):S1–3.
- 6. Finer LB, Zoina MR. Declines in unintended pregnancy in the United States, 2008-2011. N Engl J Med. 2016;374:843–52.
- Centers for Disease Control and Prevention. Preconception health and healthcare. http://www.cdc.gov/preconception/showyourlove/documents/Healthier_Me_NonPlan.pdf. Accessed 29 May 2016.
- Food and Nutrition Board, Institute of Medicine. Dietary reference intakes for vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. Washington, DC: National Academies Press; 2001.
- U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015 2020 dietary guidelines for Americans. 8th ed, Dec 2015.
- 10. Best D, Bhattacharya S. Obesity and fertility. Horm Mol Biol Clin Investig. 2015;24:5-10.
- American College of Obstetricians and Gynecologists. Obesity in pregnancy. Committee opinion no. 315. Obstet Gynecol. 2005;106:671–5.
- 12. Catalano PM. Management of obesity in pregnancy. Obstet Gynecol. 2007;109:419-33.
- Catalano PM, Presely L, Minium J, Hauguel-de Mouzon S. Fetuses of obese mothers develop insulin resistance in utero. Diabetes Care. 2009;32:1076–80.
- Kabiru W, Raynor BD. Obstetric outcomes associated with increase in BMI category during pregnancy. Am J Obstet Gynecol. 2004;191:928–32.
- 15. Ryan D. Obesity in women: a life cycle of medical risk. Int J Obes. 2007;31 Suppl 2:S3-7.
- Reynolds RM, Allan KM, Raja EA, et al. Maternal obesity during pregnancy and premature mortality from cardiovascular event in adult offspring: follow-up of 1 323 275 person years. BMJ. 2013;347:f4539. doi:10.1136/bmj.f4539.
- 17. Watkins ML, Rasmussen SA, Honein MA, Botto LD, Moore CA. Maternal obesity and risk for birth defects. Pediatrics. 2003;111(5 Pt 2):1152-8.
- American College of Obstetricians and Gynecologists. Obesity toolkit. Available at: http://www.acog.org/About-ACOG/ACOG-Departments/Toolkits-for-Health-Care-Providers/Obesity-Toolkit. Accessed 29 May 2016.
- Jans G, Matthys C, Bogaerts A, et al. Maternal micronutrient deficiencies and related adverse neonatal outcomes after bariatric surgery: a systemic review. Adv Nutr. 2015;6:420–9.
- 20. Johansson K, Cnattingius S, Näslund I, et al. Outcomes of pregnancy after bariatric surgery. N Engl J Med. 2015;372:814–24.
- Roos N, Neovius M, Cnattingus S. Preinatal outcomes after bariatric surgery: nationwide population based matched cohort study. BMJ. 2013;347:f6460. doi:10.1136/bmj.f6460.
- 22. Committee to Reexamine IOM Pregnancy Weight Guidelines, Food and Nutrition Board and Board on Children, Youth and Families, Rasmussen KM, Yaktine AL, editors. Weight gain during pregnancy: reexamining the guidelines. Washington, DC: National Academies Press; 2009.
- Sridhar SB, Darbinian J, Ehrlich SF, et al. Maternal gestational weight gain and offspring risk for childhood overweight or obesity. Am J Obstet Gynecol. 2014;211(3):259. doi:10.1016/j.ajog.2014.02.030.e1-8.
- 24. Food and Nutrition Board, Institute of Medicine. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (macronutrients). Washington, DC: National Academies Press; 2005.
- Food and Nutrition Board, Institute of Medicine. Dietary reference intakes for calcium and vitamin D. Washington, DC: National Academies Press; 2010.
- 26. Food and Nutrition Board, Institute of Medicine. Dietary reference intakes for vitamin C, vitamin E, selenium, and carotenoids. Washington, DC: National Academies Press; 2000.
- Food and Nutrition Board, Institute of Medicine. Dietary reference intakes for thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin, and choline. Washington, DC: National Academies Press; 1998.
- U.S. Food and Drug Administration. Changes to the nutrition facts label. May 20, 2016. Available at: http://www. fda.gov/Food/GuidanceRegulation/GuidanceDocumentsRegulatoryInformation/LabelingNutrition/ucm385663. htm. Accessed 22 May 2016.

- U.S. Department of Agriculture. SuperTracker. Food-A-Pedia. Available at: https://www.supertracker.usda.gov/ foodapedia.aspx. Accessed 22 May 2016.
- Office of Dietary Supplements. National Institutes of Health. Iodine. Fact sheet for health professionals. Available at: https://ods.od.nih.gov/factsheets/Iodine-HealthProfessional/. Accessed 22 May 2016.
- 31. European Food Safety Authority. Folic acid: an update on scientific developments. 2010. Available at: http://www.efsa.europa.eu/en/home/publication/efsafolicacid.pdf. Accessed 14 Feb 2016.
- 32. Katan MB, Borkschoten MV, Connor WE, et al. Which are the greatest discoveries and the future challenges in nutrition? Eur J Clin Nutr. 2009;63:2–10.
- 33. Stagnaro-Green A, Abalovich M, Alexander e E, et al. Guidelines of the American Thyroid Association for the diagnosis and management of thyroid disease during pregnancy and postpartum. Thyroid. 2011;21:1081–125.
- Centers for Disease Control and Prevention. Recommendations to prevent and control iron deficiency in the United States. Morb Mortal Wkly Rep. 1998;47(No. RR-3):1–36.
- 35. Lopez A, Cacoub P, Macdougall IC, Peyrin-Biroulet L. Iron deficiency anemia. Lancet. 2016;387:907-16.
- Menon KC, Ferguson EL, Thomson CD, et al. Effects of anemia at different stages of gestation on infant outcomes. Nutrition. 2016;32:61–5.
- 37. Siu AL, US Preventive Services Task Force. Screening for iron deficiency anemia and iron supplementation in pregnant women to improve maternal health and birth outcomes: US Preventive Services Task Force Recommendation Statement. Ann Intern Med. 2015;163:529–36.
- 38. Luke B. Nutrition for multiples. Clin Obstet Gynecol. 2015;58:585-610.
- 39. Luke B. Nutrition and multiple gestation. Semin Perinatol. 2005;29:349-54.
- 40. Goodnight W, Newman R. Optimal nutrition for improved twin pregnancy outcome. Obstet Gynecol. 2009;114:1121–34.
- 41. Marcason W. Question of the month. What are the calorie requirements for women having twins. J Am Diet Assoc. 2006;106:1292.
- 42. Checklist of foods to avoid during pregnancy. Available at: http://www.foodsafety.gov/risk/pregnant/chklist_pregnancy.html. Accessed 30 Mar 2016.
- 43. Recalls of frozen vegetables, frozen fruit, and other products related to investigation of listeria illnesses. Available at: http://www.foodsafety.gov/recalls/recent/listeria.html. Accessed 29 May 2016.
- Food and drug administration: to sign-up for recalls, market withdrawals, and safety alerts. http://www.fda.gov/ Safety/Recalls/default.htm. Accessed 29 May 2016.
- 45. Foodsafety.gov. To sign up for food recalls and alerts. http://www.foodsafety.gov/recalls/alerts/index.html. Accessed 29 May 2016.
- 46. Torpy JM. JAMA patient page. Eating fish: health benefits and risks. JAMA. 2006;296:1926.
- 47. U.S. Food and Drug Administration. Consumer Health Information. New advice: pregnant women and young children should eat more fish. June, 2014. Available at: http://www.fda.gov/downloads/Food/ FoodborneIllnessContaminants/Metals/UCM400358.pdf. Accessed 27 May 2016.
- 48. Oken E, Belfort MB. Fish, fish oil, and pregnancy. JAMA. 2010;304:1717.
- 49. U.S. Food and Drug Administration and United States Environmental Protection Agency. Fish: what every pregnant and parents should know. Draft Updated Advice by FDA and EPA, June, 2014.
- Oceana Study reveals seafood fraud nationwide. Available at: http://oceana.org/sites/default/files/National_ Seafood_Fraud_Testing_Results_Highlights_FINAL.pdf. Accessed 27 Mar 2016.
- 51. Division of Emergency and Environmental Health Services, National Center for Environmental Health, Centers for Disease Control and Prevention. Guidelines for the identification and management of lead exposure in pregnant and lactating women. Atlanta: US Department of Health and Human Services; 2010.
- 52. Shah-Kulkarni S, Ha M, Kim BM, et al. Neurodevelopment in early childhood affected by prenatal lead exposure and iron intake. Medicine. 2016;95:e2508.
- Centers for Disease Control and Prevention. Lead poisoning. Are your pregnant. Available at: https://www.cdc.gov/ nceh/lead/tools/are_you_pregnant.pdf.
- 54. Bellinger DC. Lead contamination in Flint an abject failure to protect public health. N Engl J Med. 2016;374:1101–3.
- 55. Flint's Most Vulnerable. The New York Times. 5 Apr 2016.
- 56. Williams JF, Smith VC. Committee on substance abuse. Pediatrics. 2015;136:e1395–406.
- Green PP, McKnight-Eily LR, Tan CH, Mejia R, Denny CH. Vital signs: alcohol-exposed pregnancies United States, 2011–2013. Morb Mortal Wkly Rep. 2016;65:91–7.
- Tan CH, Denny CH, Cheal NE, Sniezek JE, Kanny D. Alcohol use and binge drinking among women of childbearing ag Initd States, 2011–2013. Morb Mortal Wkly Rep. 2015;64:1042–6.
- Halldorsson TI, Storm M, Olsen SF. Intake of artificially sweetened soft drinks and risk of preterm delivery: a prospective cohort study in 59,334 Dansh pregnant women. Am J Clin Nutr. 2010;92:626–33.
- Bursey RG, Watson ML. Intake of artificially sweetened soft drinks and risk of preterm delivery. Am J Clin Nutr. 2010;92:1277–8.

- 61. La Vecchia C. Intake of artificially sweetened soft drinks and risk of preterm delivery. Am J Clin Nutr. 2010;92:1540.
- Procter SB, Campbell CG. Position of the Academy of Nutrition and Dietetics: nutrition and lifetyle for a health pregnancy outcome. J Acad Nutr Diet. 2014;114:1099–103.
- 63. American College of Obstetricians and Gynecologists. Moderate caffeine consumption during pregnancy. Committee opinion no. 462. Obstet Gynecol. 2010;116:467–8.
- Browne ML. Maternal exposure to caffeine and risk of congenital anomalies. A systematic review. Epidemiology. 2006;17:324–31.
- 65. Committee on Practice Bulletins. American College of Obstetricians and Gynecologists. Nausea and vomiting of pregnancy. Number 153. Obstet Gynecol. 2015;126:e12–24.
- Wagner BA, Worthington P, Russo-Stieglitz KE, Levine AB, Armenti VT. Nutritional management of hyperemesis gravidarum. Nutr Clin Pract. 2000;15:65–76.
- Hsu JJ, Clark-Glena R, Nelson DK, Kim CH. Nasogastric enteral feeing in the management of hyperemesis gravidarum. Obstet Gynecol. 1996;88:343–6.
- Müller-Lissner SA, Kaatz V, Brandt W, Keller J, Layer P. The perceived effect of various foods and beverages on stool consistency. Eur J Gastroenterol Hepatol. 2005;17:109–12.
- 69. American Diabetes Association. Classification and diagnosis of diabetes. Diabetes Care. 2016;39 Suppl 1:S13–22.
- 70. American Diabetes Association. Management of diabetes in pregnancy. Diabetes Care. 2016;39 Suppl 1:S94–8.
- Centers for Disease Control and Prevention. National Diabetes Statistics Report, 2014. Available at: http://www. cdc.gov/diabetes/pubs/statsreport14/national-diabetes-report-web.pdf, http://www.cdc.gov/diabetes/pubs/statsreport14/national-diabetes-report-web.pdf. Accessed 20 May 2016.
- Masho SW, Urban P, Cha S, Ramus R. Body mass index, weight gain, and hypertensive disorders in pregnancy. Am J Hypertens. 2016;29:763–71.
- 73. Task force on Hypertension in Pregnancy. American College of Obstetricians and Gynecologists. Hypertension in pregnancy. Executive summary. Obstet Gynecol. 2013;122:1122–31.
- 74. Indumati V, Kodliwdmath MV, Sheela MK. J Clin Diagn Res. 2011;5:66-9.
- Bushnell C, McCullough, Awad IA. Guidelines for the prevention of stroke in women. A statement for healthcare
 professionals from the American Heart Association/American Stroke Association. Stroke. 2014;45:1545–88.

Chapter 17 Promoting Nutrition in Men's Health

Simon Rowlands and Brendan Gough

Key Points

- In an increasingly neoliberal context, men's nutritional health has largely assumed to be the cumulative product of individual choices. As a result, attention to the influence of contextual and social factors has frequently been underestimated or overlooked.
- Gender, as something one does socially, rather than something one is individually, plays an important role in defining what choices are available. Food is used in a number of ways to signify gender.
- Food has a social as well as a nutritional value, and health promotion efforts that fail to recognise this may be limited in their salience and effectiveness.
- Many of the practices that are associated with currently dominant forms of masculinities are incongruent with healthy eating advice.
- Effectively promoting nutritional health in men may require a combination of working within and exploiting some masculine norms, whilst simultaneously challenging those which impact negatively on health

Keywords Masculinity • Men • Nutrition • Health promotion • Gender

Introduction

Food preferences, dieting practices and healthy eating are all interlinked and can be considered as socially constructed and gendered phenomena. As this chapter will discuss, men and women are thought to have different approaches to nutrition – although sex differences are often overstated and changing gender ideals suggest that the relationship between gender and food is complex, dynamic and context-bound. In this chapter, we look closely at 'masculinity' (or 'masculinities') and its

S. Rowlands, PhD

Public Health/Health Promotion, Leeds Beckett University, 518 Calverley Building, City Campus, Leeds, West Yorkshire, LS1 9HE, UK e-mail: s.rowlands@leedsbeckett.ac.uk

B. Gough, PhD (⊠) School of Social, Psychological & Communication Sciences, Leeds Beckett University, Calverley Building, Rm 902b Leeds LS1 3HE, UK e-mail: b.gough@leedsbeckett.ac.uk

important influence on men's health generally and on nutrition specifically. We look beyond medical and psychological perspectives to social science theory and research of relevance to men's foodrelated activities and highlight concepts which could inform recommendations for improving men's engagement with healthy eating.

Masculinity and Men's Health

As far back as 15 years ago, Watson [155] described a long established and entrenched male disadvantage with regard to health. More recent epidemiological studies map a widening gender disparity that has resulted in men currently dying on average 5-6 years earlier than women [9, 99, 162]. Men suffer from higher mortality rates for the majority of leading causes of death within all age ranges, including those in which diet plays a significant role in managing such as cardiovascular disease, diabetes, obesity and various forms of cancer [161]. This situation is in some ways surprising as it has come about in a global context where commitment to redressing inequalities in health has the appearance at least to be increasing overall [91], with a particular emphasis in some areas on those which impact on gender specifically [70, 164]. However, some are critical that the rhetoric of gender mainstreaming has become synonymous with women's health, and that the significant and multiple issues facing men go largely unrecognised within the health promotion debate [90, 159]. There currently exists a paradox where the unhealthy male appears simultaneously ubiquitous within Western media and culture, while at the same time invisible at the level of health policy, planning and research [6, 71, 77, 97, 123]. Not only are men rarely targeted as a distinct group in need of intervention in their own right, they are also frequently homogenised into a single-sex category that overlooks any potential for difference resulting from class, ethnicity, age, etc. This lack of success in finding a place for men on the health promotion agenda can in part be viewed as a consequence of an increasing neoliberal policy context [76, 85] that frames food consumption solely in terms of choice, while neglecting powerful social structures and influences. Here, men have tended to become regarded as nutritionally and 'naturally' feckless, wreckless or disinterested. Food ignorance, alongside a more general and widespread ambivalence towards health, becomes an expected, inherent, incurable and an all but inevitable male characteristic. In this scenario, it is men's faulty decision making that is at the root of the poor nutritional health, and they have no one to blame but themselves. Social factors become minimalised or lost altogether.

It is important to note early in the discussion, when building a rationale for an increased interest in men's health, that taking such a line does not necessarily argue against a justifiable concern over the many disadvantages suffered by women in a patriarchal society. The observation instead is that men's health may suffer in the pursuit of power and dominance [36], and that the health of men and women is inextricably linked and hold less meaning when considered in isolation [5, 98].

The figure of the stereotypical unhealthy male, epitomised in popular culture by the beer drinking, lazy and helplessly childlike Homer Simpson, has been tied to the notion of a masculine 'type' behaviour. Such narratives, commonly viewed as taken for granted social truisms, often have their foundations in essentialist ideas about 'what men are' and 'what men do'. This has in turn created a persistent lay perception of gender as a singular, biologically determined category within a kind of 'pop' psychology in which men are assumed to possess stable, universal and immutable traits. Examples can be heard every day in culturally embedded and socially rehearsed scripts that might, for example, presume a masculine tendency to refuse to ask for help, whether that be from the doctor in health matters, an instruction book for technical failures or a road map for direction. It is in this socially constructed, as opposed to biologically certain, context that 'real' men are expected and required to be independent, rational, unemotional or stoic. Extending this thinking of the ways in which society expects men to behave allows the consideration and understanding of why men may make a range of unhealthy

lifestyle 'choices', including those that pertain to food. This is discussed further below. However, in order to do this in a meaningful way, it is first useful to reflect on what might be meant by the terms gender and masculinity and how this relates to health.

Gender and Health

Interest in gender and health gained momentum in 1930s and 40s with studies that began to recognise significant disparity in disease and death rates between men and women from early epidemiological data. Much of the work at this time centred on analysis of differences between the sexes [29]. Influenced by the emergence of key sociological schools and theorists at that time, this led to the development of predominantly functionalist theories of gender, and the popularisation of the notion of socialised traits or 'sex roles'. Early authors began to converge around a set of common, core, masculine values which included independence, autonomy, dominance over others and the suppression of emotion [40, 149]. Masculine stereotypes were largely constructed in binary opposition to what might be considered feminine, where for example, David and Brannon [40, page 49] identify a masculine requirement to engage in 'no sissy stuff - the stigma of anything vaguely feminine'. Such theoretical positions help to explain why food provision and preparation may be assigned feminine sex role status and consequently pose a threat to masculine identity. In focusing on what might be different between biological sex categories, the academic literature developed and, arguably, has continued to develop a tendency to reinforce strict gender dualisms. Early sex roles that presumed women's 'nature' to be nurturing and health-conscious laid the foundations for defining masculine behaviour predominantly in terms of health-depleting or risk-taking behaviours. Indeed, innumerable papers can be found which link violence, drug taking and the use of fast cars and motorcycles to performances of masculinity. The culturally ingrained nature of these symbolic displays of manhood has again been reflected in media representations, where the 'Marlboro Man' epitomised masculine cool throughout the 1950s, and where film and sport idols were either strong and silent (John Wayne, Clint Eastwood), or risk-taking rebels (James Dean, Marlon Brando etc.) [81].

In 1978, Harrison claimed that the 'male sex role is bad for your health' (Page 65). Later, Courtenay [36] offered a theory of gender and health that proposed risk-taking or health-eroding behaviours as important signifiers of masculine identity. The overall result of this narrow interest in unhealthy or risky enactments of male gender has been a gradual pathologisation and medicalisation of masculinities, and by association men [124] to the point where being male itself might be considered a risk factor for disease. Riska's [119, 120] psychological personality 'types' provide one illustration of this process, where 'Type A' aggressive, competitive characteristics have become widely regarded as a 'natural male' type.

Unhealthy male type behaviours have been explored in relation to men's food-related practices where excessive alcohol consumption [59, 153, 154, 165] or a preference for larger, 'man sized portions' [15, 68, 152] are utilised for impression management. In this sense, the idiom 'you are what you eat' appears to hold true for gender at least:

It behoves a man to eat more, drink more, and to eat and drink stronger things (Bourdieu [18], page 192).

Sex role theory (SRT) and the notion of an inflexible collection of masculine traits fell subject to growing criticism in the 1970s and 1980s from feminist academics for its failure to explain the importance of (male) power, the potential for change and the countless occasions where normative gendered roles might be rejected or subverted [46, 112]. It was in response to these conceptual conundrums that the academic field of men's health emerged and Connell [32] began to develop what is now considered to be the dominant theory of masculinity (or more accurately masculinities).

Connell [33] convincingly argued that SRT cannot adequately account for homosexuality or explain the differences between men in any way other than eccentricity or idiosyncrasy. Problems exist too in a conceptual landscape of SRT where male dominance over women can be legitimated, or conceived as 'natural' in the sense of being biologically predetermined [26]. Added to this, debate over the relative influence of agency or free will over structure is left largely untheorised in SRT. Employing a nutritional example to illustrate this point, the recent changes in men's cooking, caring responsibilities and body image ideals discussed later cannot be reconciled within an inflexible, static SRT framework which proposes historical stability within which masculinity becomes a singular and consistently defined construct (see [137]). Over recent years, a more nuanced literature on masculinities has emerged based on Connell's [34] concept of multiple, dynamic and situated expressions of gender. These challenge the idea of singularly harmful masculinities by proposing that different situations may expect, value and warrant different social practices. This not only allows for a variety of masculinities across men, but also different presentations within the same man at different times and places. Robertson [122] discusses how this might create a tension between old and new masculinities in his 'don't care, should care' dilemma for modern men. Here, the changing expectation (the 'crisis of masculinity') is one of moral responsibility to maintain health in a neoliberal, capitalist and consumerist society, but where traditional ideas that men should be seen not to care still hold some traction and import. For Robertson, the resulting compromise is a cycle of control and release where, for example, the hard labour of the working week is rewarded by 'letting go' at the weekend. Similarly, others [38, 39] discuss the influence of 'healthism' and a consumer-fuelled aesthetic health ethic of late modernity, which impacts on and sometimes conflicts with changing expectations of gender. In relation to food practices, this dissonance might become manifest in an emerging climate where the increasing prevalence of sexualised images of male bodies in the print media increases the pressure on young men to strive for 'six pack' abdominal muscles, while traditional values hold that vanity, excessive or overt concern over appearance encroaches into female terrain [118]. Here, the tricky balance for men is to care while maintaining the impression of ambivalence.

Connell [34] outlines a hierarchical structure of masculinities containing four configurations of practice. Briefly, hegemonic versions dominate over all other masculinities (and women). Hegemonic masculinities provide the exemplar for maleness, modelling valued ways in which 'real men' are expected to behave. Since few men are able to meet such exacting normative standards, many more exhibit 'complicit' masculinities, that is, they benefit from, support, compliment and contribute towards the patriarchal advantage (male power). 'Subordinated' masculinities hold the least power, most directly contrast and therefore threaten or challenge hegemonic ideals, and as a result are frequently stigmatised. Gay masculinities offer an illustrative example of this category. Finally, 'marginalised' masculinities may also suffer discrimination and disadvantage with respect to social position and power, but typically in more subtle forms. Men living with disability might be described in this way.

Connell builds on West and Zimmerman's [158] seminal idea of 'doing gender', where performing masculinity becomes a situated social practice and an ongoing achievement; something one does rather than something one is; a verb as opposed to a noun. It is in this repeated, everyday 'doing' of masculinity that men's health behaviour and decision making can be observed, explored and explained. It is through the inherently social nature of gender identity construction, and the idea that masculinity must be constantly and consistently achieved and socially reinforced, that seemingly intransigent masculine attitudes toward food can begin to be understood. As some writers in gender studies suggest, by *not* 'doing' health, cooking, dieting, slimming, etc., some men may in some situations be 'doing masculinity' (see [56, 89, 129]). However, there is strong evidence to suggest that the context for shaping modern masculinities is changing. An ever more pervasive media saturated environment, the moralisation and commercialism of health and the relatively unquestioned acceptance of a neoliberal political context that prioritises individual choice and responsibility combine to make it increasingly difficult to sustain irresponsible or unhealthy masculinities. These changing social expectations

have increased the requirement for men to attend to their health and appearance, adding a growing pressure to reconcile being masculine with being healthy. This creates new issues for men and new opportunities for the health promoter.

So what does this all mean for food practices and nutritional health? The following section explores these and other ideas using examples from the literature and concludes that food 'choices' for men, far from being a product of rational and linear decision making, are strongly directed by the ways in which men are required to behave in social situations. Further, the notion is put forward that many aspects of healthy eating are incongruent with prevailing ideals of masculinity. The concept of risk can be defined and calculated for both health and for gendered identity, where threat may not be measured or valued equally. The consequences of losing masculine capital are significant, immediate and social, where health is gained or lost individually, distally, privately and over much longer periods. It should come as little surprise then, when the 'rational' decision is not always the healthiest.

Men, Food and Health

Men's diets are typically worse than those of women. Men tend to be more ignorant of the cost and nutritional properties of food, spending little time in the kitchen and generally positioning themselves as consumers rather than caterers. Those who take an interest in the sociology of food may see this as an inevitable consequence of traditional gendered divisions of labour within the home, where duties and responsibilities represent status. The point here is that food has significant social meaning beyond its nutritional value and application as a means for improving health [35, 54, 111]. Health promotion interventions that neglect this consideration and take a purely educational or behavioural approach may fall short in their expectations of success [41]. Food is in many ways gendered and gendering; food establishes and secures social position and cements relationships; food is used to express a broad range of emotions and moods; food signals acceptance, membership, inclusion and exclusion from families, groups and colleagues. The utility of food as a symbol of gender can be categorised into three main areas: Who does the food work, what is eaten and how much is eaten?

Much has been written in the critical sociological literature of the feminisation of food practices, such as shopping, preparation and cooking [24, 79, 152]. Feminist authors put forward a convincing argument that these largely unquestioned social practices act to reproduce particular forms of economic and social inequalities under the banner of 'women's special relationship to food' [43, 79, 110]. Quantitative studies offer considerable support for these theoretical claims, demonstrating that, notwithstanding some small recent improvements in terms of balance, women still undertake the vast majority of food work [10, 113, 147]. Even in an age where equality is becoming all but assumed, it is the wives or female family members that are most often called upon to provide for the men in the household [86, 121]. This is most commonly justified by appealing to a discourse of women's superior competence, skill and time [84]. Reluctance to engage with food work is most common in older and married men, and with those who hold more strongly to traditional ideas of gender [45, 92]. Thankfully, this is not the whole picture. There are emerging indications of newer constructions of masculinity with regard to food preparation inspired in part by the modern media trend for celebrity male chefs such as Jamie Oliver and Gordon Ramsey. However, these developments have been criticised for aligning to, or subtly and superficially changing the presentation of oppressive hegemonic masculinities, rather than rejecting them outright. For example, in this context, cooking is often cast as a male hobby or leisure pursuit, while the day to day drudgery of food work remains a woman's responsibility. This might constitute 'redoing' in new ways, rather than 'undoing' unequal gender relations [69, 148]. Similarly, other sites of resistance to the feminisation of food practices exist where, for example, food work is read as independence and self-reliance in single men who reject the

stereotype of incompetent bachelorhood [56, 136]; where certain professional or class expectations override gender norms such as when fire-fighters actively and strongly discourage female presence in the kitchen as part of a site-specific masculine identity [42]. Equally, men in lower social classes may favour traditional gendered relationships with food, whilst middle-class men might adopt more egalitarian practices [128]. Such occasions point to the folly of homogenising men into a single social category, the importance of changing social attitudes over time, context and intersecting identity factors on food practices.

This social gendering of food roles often works to reinforce traditional ideas about women's place and power, and there may be occasions where they themselves become complicit in reproducing patriarchal dominance through what Connell [34] calls 'emphasised femininities'. The provision of food within the family unit has long been synonymous with a stereotypical view of 'natural, nurturing femininities' [28, 43], where male encroachment may be viewed as a challenge to established gender relations, even a threat to femininities. Mroz et al. [107, 108] illustrate the practical implications of this in a study of men with prostate cancer whose attempts to influence female partner's cooking and provision of food are actively resisted by some women. One analysis of such gendered food practices is that the kitchen domain exists as one of the few places where women can acceptably exert power and control over men or, alternatively, where pleasure might be taken in providing for others [52]. Such assertions help to explain this potential arena of conflict where men's 'choices' with regard to food might be limited (as may women's opportunities to express and exert power). Is it possible, practical or ethical to ask men to put important relationships in jeopardy in prioritising nutritional over social health? Mroz et al. [107, 108] suggest that in such circumstances, the pragmatic solution might be to work within established gendered frameworks rather than attempt disruption.

Food as (feminine) affection might also manifest in different ways and in different conflicts which further problematise the notion that men's choice is derived purely from individual cognitive risk calculation, unrestricted by structural or social factors. For example, food in quantity or as personalised treats (usually unhealthy) become difficult to refuse for fear of rejecting kindness, as an interviewee from Rowland's [130] study of male weight management observes:

...but when I look at what my Mum serves me up... when you watch these documentaries on channel five about... incredibly obese people and they're being killed by kindness...It's absolutely beautiful and there's almost this sense that I don't want to not, let her down by not eating it. (Tony)

Murcott [110] establishes that knowledge of, and catering for, personal preferences within the home signifies the woman's status within *this particular family rather than any other*. Given this context, refusal or rejection presents immediate social risks to important, intimate relationships. Similar findings from Rowlands [130] show that rejecting food offered to men in kindness is more often taken as a sign of illness or a slight on culinary competence than a proactive attempt to eat more healthily:

It's like I went to me daughters last night for me tea and everybody says are you feeling alright? Cause I'd left some potatoes on me plate. (Eric)

Equally, where food is used to celebrate, commiserate or strengthen family and friendship bonds, refusal on physical or nutritional health grounds may come at the expense of social health, as another participant from the same study notes:

But this is what the problem was though that two weeks ago my Mum and Dad came back from Pakistan and the baby boy was new and so we were going over to their house every bloody three days and we just eating none stop, celebrating and what not...It's like, you know, hard to stop. It's not straightforward. It's bloody hard. (Ikram)

This raises issues of deference and asks, on balance, who concedes to whom with regard to how decisions about food are made in this important domestic setting? Much of the literature suggests that

it is women who put the needs and tastes of children and male family members before their own [10, 16, 21, 37, 110]. This, and the notion that women have greater expertise and applied knowledge of healthy eating, is borne out in studies that point to the significant improvements in men's diets after marriage, and again after starting a family [127, 135]. Such work cannot be ignored, but at the same time, very few studies have asked the same questions of men, leaving us bereft of one side of a relational dialogue. Indeed, the few researchers that have broached this area suggest there is a perception from men that it is they who defer to others in nutritional matters [45, 130]. DeBourdeaudhuij and Van Oost [16] helpfully remind us not to take at family value, as reflections of the absolute truth, subjective statements about family food decisions. While a more equal balance in whose opinions are sought in the research may not bring us any closer to knowing accurately where the power lies, it may help us to understand better how specific food practices are contested or reproduced.

Moving on from provision, there is a general consensus that individual food items can also be highly gendered [114], and so it follows that some men may enact their maleness through the things they eat, and sometimes through the things they do not. Potatoes and red meat are the clearest examples of this. Again, attention to the import of these symbolic foods within the sociological literature [1, 80, 129, 131, 142] is justified and consolidated by large-scale quantitative surveys' which repeatedly show that women proportionally eat less of these 'masculine foods', replacing them instead with fish, fruit and vegetables [11, 49]. This has been linked to a perception of the strength and size building properties of masculine food choices [28, 145], which is discussed further in relation to portion size and the valorisation of large male bodies. The propensity to adhere to 'masculine' foods may be more pronounced in lower social classes [128]. In addition, feminist writers have explored a link between the sexual politics of a male rejection of vegetarianism and wider environmental issues as a mechanism for maintaining patriarchy through symbolic dominance and violence [1, 131]. The modern media in general have been shown to play into a narrative that 'genders' specific food items (Fernstein 1982; [69]), and male-targeted magazines in particular display a tendency to sanction 'feminine food choices' and trivialise men's efforts to eat more healthily [56, 145]. As Feirstein [48] points out in his guide to 'all that is truly masculine', 'real men don't eat quiche'. Rothgerber [129] takes this further in identifying vegetable, as opposed to any other type of quiche, as being most likely to offend ones sense of masculinity.

The third way in which food might be used by men for impression management relates to portion size, volume and food quantity [15, 68, 105, 152]. The popular appeal of television shows like 'Man versus Food' and 'Hairy Bikers' orients towards a traditional view of the need to distinguish 'man sized meals' from those of women and children. Food in volume has been historically justified as 'fuel' for demanding outdoor masculine occupations - in binary opposition to the more sedentary, domestic indoor activities of women [24]. Similar forms of socialisation with regard to food volume are evident at each stage of the life course beginning in the early years where lusty appetites are viewed as healthful in boys, while 'greediness' is met with disapproval in girls. Here, young femininity is embodied at the dining table through 'eating lightly' [105, 114], and as women age, there is an expectation that their needs in terms of quantity may be sacrificed in order to cater for the requirements of the working men and growing children within the family [28, 43, 61]. In her seminal study of the significance of the cooked dinner in British (Welsh) culture, Ann Murcott [110] recognises the rituals that surround these shared eating occasions as fundamental to the physical and social health of the family. Murcott also eloquently describes the appropriate share each member is given, measured universally as 'a plateful', but distinct in that a man's quota has the largest proportion of meat and vegetables (usually potatoes), and the shortfall for women and children is compensated for with gravy. These familial practices are compounded by a wider social pressure that dictates an expectation for women to restrict and control their calorie intake more generally [30, 115, 134, 146]. Later studies draw similar conclusions where healthy portions for men are described as robust (substantial) and women's as virtuous (small and moralised) (Beardsworth 2002).

Men, Dieting and Slimming

Attempts by the health professional to encourage healthier eating habits can often be perceived by men as focusing on excluding many or all of the food products and practices that signify masculinity (traditionally conceived), and replacing them with less satisfying and tasteless substitutes. Dieting then becomes essentially reduced to eating feminine things in feminine ways [56].

There is some evidence to suggest that in general men take an interest in their nutritional health in ways that are distinct from the motivations expressed by many women. Studies show that men frequently place greater emphasis on improving functional fitness or performance, either in a sporting or occupational context, as opposed to harbouring aspirations of health improvement per se. In addition, it is suggested repeatedly in the research that a more complex, often bidirectional relationship exists when male bodies are utilised as an expression of gender than the more straightforward associations between femininity and slenderness. Put plainly: men tend to not want to be too large or too little.

Subtle signs of these masculine notions of functional weight management (as distinct from weight loss) can be seen in health promotion resources geared towards the male market. A series of publications from the UK's Men's Health Forum illustrate how masculinising sporting, militaristic and mechanical metaphors have been adopted in order to reframe nutrition in ways that appeal more to hegemonic ideals and values. For example, 'HGV Man' [8] adopted the familiar publishing format of the Haynes car workshop manual in order to 'reduce all large sizes' for 'all shapes and colours'. Chapter headings discuss the 'right fuel', encourage men to 'Go for an MOT' and 'understand what's under your bonnet'. Content includes sections covering work-related performance and nutrition for football (soccer). These themes are employed consistently throughout the series, linking strongly to ideals of masculine independence, autonomy and scientific rigour (contrasting feminine/emotional weight management). Rigorous individual commitment and will power ('plan your attack', section 3.23), along with rational decision making, are encouraged as being all that is necessary to achieve your goals: 'fight the insulin resistance' (section 4.12) and win 'the war of terror on fat' (section 4.14). While such approaches undoubtedly appeal to some men, there are wider debates concerning the harmful effects of reproducing and reinforcing stoic, dominating or aggressive stereotypes. Here, the influence of social context can be disregarded as unimportant and victims blamed for poor choices. Already marginalised men or those who do not conform to sporting ideals or car worship can be further alienated. Emotional male responses become increasingly socially sanctioned and repressed, while specific forms of (strong and silent) masculinity become valued and promoted above health [57]. The risk here is of further emphasising gender difference and dichotomy, reinforcing narrow constructions and limiting further the possibilities for choice.

This brings the discussion around to the ideas of dieting and slimming, concepts which have been writ large as 'something that women do' and men do not [104, 133, 144]. Yet again hard statistics confirm the pervasiveness of these socially constructed gender binaries, where despite their higher rates of overweight than women [73, 74], men are outnumbered in weight management services at a ratio of 5 to 1 on average [121, 167]. In addition, weight management research is also dominated to a similar degree by studies that either fail to recruit or fail to disaggregate data for men. This has resulted in services based on research on women, built on the preferences of women [116, 159]. Men's reluctance to reach out to services that promote *weight loss* is hardly surprising given the differential treatment of men and women by health professionals, who it seems are not immune from reflecting the social norms that frame fat as a feminine (sic) issue. Doctors spend 9 minutes less with overweight men than overweight women (Hebl et al. 2003); men's BMI is recorded less frequently (Gibbs et al. 2001), and when intervention is indicated, men are less likely to receive advice, information or discussion of weight issues [14, 143, 156].

Beyond the health professional it might also be informative to consider the impact of the attitudes of men themselves towards the management of their weight in order to build a more rounded explanatory picture. Weight perception tends to vary between the sexes where men most often underestimate and women over estimate their weight [22, 27, 31, 74, 75, 82, 93, 94, 156]. As the population BMI average increases, so does both the accuracy and threshold at which individuals self-assess as overweight, and this effect tends to be greatest in lower SES men [78]. Wardle and Johnson [156] argue that men's self-expressed ideal weight is often a reflection of the statistical, cultural and social norm rather than medical guidelines – that is, men compare and judge themselves against those around them. More sociological understandings posit larger male bodies as a literal representation of power, strength and masculinity, and this is discussed below in relation to male body image ideals. As a result, men show less dissatisfaction with being overweight than do women [50, 83, 109], exhibit lower levels of related depression when overweight [25], assess the risk of obesity more positively and inaccurately than women and therefore are less likely to attempt remedial action [63, 82]. However, there is emerging evidence that such attitudes and perceptions of men and in men are changing. As Connell [34, page 45] notes:

Bodies do matter' and 'true masculinity is almost always thought to proceed from men's bodies...

In a modern cultural context, the importance of image, presentation and appearance dominates. In this sense, male bodies are important as much for what they represent as what they can achieve and how they perform the functions of everyday life. Chris Shilling [139] describes how in a growing capitalist, consumerist society bodies have become commodified, identity projects to be worked on, whether that be in the gym, through food supplementation, cosmetic or bariatric surgery, etc. Turner [151] similarly considers the implications of a '*somatic society*', while Crawshaw [39] and Crawford [38] discuss disciplined consumer-savvy 'aesthetic health' in which health, beauty and identity become conflated into a single embodied concept. Pierre Bourdieu [18] argues that class too can be written into the way identity is presented through the body.

When it comes to appearance, men frequently express a preference for bodies different to the ideals of women and those of medical or weight management professionals. This has implications for promoting nutritional health in men. Most researchers in the field agree that men's concern over body image is increasing [95], and that as many men wish to gain weight as to lose it [17, 44, 51, 96, 100]. The increasing extent of male concern, and its relative lack of consideration in the female-dominated body image literature [7, 12], is reflected in statistics that show a recent increase to 3% of men in the USA who admit to using anabolic steroids as a way of enhancing their appearance, while anorexia nervosa in women is stable at around 1 % [23, 53, 141]. Here, size is directly associated with masculinity, and healthy male bodies are conceived as being both big and strong. Ideals represent the combination of what is considered healthy and what is considered masculine. Such abstract conceptions can present practically in clinical situations as a commonplace male rejection of medically focused BMI measures, where both salience and science can be questioned [102], often creating a polarising and ultimately counterproductive tension between medical and masculine discourses. This highlights a gap in men's body size between the statistical and cultural norms. Epidemiology tells us that fat men represent the former, but are paradoxically and morally judged against the latter as deviant, where soft fatness is coded as weak willed and feminine in comparison to hard and masculine muscularity [17, 62, 104]. A general, but not universal desire for lean and muscular ideals reached a peak of hyper muscular bodybuilding proportions in the mid-1990s, with less extreme, more athletic physiques being more in vogue currently [87, 117]. It is interesting that this pattern coincided with a similar high point in the depiction of muscular and sexualised male body images in film and the media [20], and had less impact in those countries where such representations of masculine bodies have been less common [166]. Such evidence illustrates a positive potential for change over time, which may provide avenues to pursue for the health promoter. Equally, extensive studies of body image in gay men give some cause for optimism in terms of going beyond a single, narrowly defined ideal, offering instead an interesting and helpful variety of idealised body shapes. These range from extreme slenderness in 'twinks' (slim gay men), often feminised and ascribed vacuous characteristics (much like the female 'bimbo' stereotype), to the hyper muscularity and exaggerated machismo favoured by some in the 1980s and 90s [96]. An interesting subculture relating to body weight and size within the gay community is that of 'bears' - sexually valorised as larger, more rugged, often hirsute men who mirror what might be considered strongly traditional heterosexual male attributes [60, 67, 101, 125, 163]. Simultaneously critiqued for reproducing a version of hegemonic masculinity and lauded for subverting and reconfiguring heteronormative maleness in the context of HIV associated weight loss, bigger, softer (fatter) bear bodies become, in this specific and situated milieu, the embodied epitome of healthy masculinity [67, 163]. This is important because it shows that multiple conceptions of healthy male bodies are possible and context-bound. As a result, those interested in promoting nutritional health might be able to develop both a broader palate in terms of what might be considered as the embodiment of masculinity and health, and also emphasis might shift away from (reducing) size towards a more direct consideration of health in its broadest sense. Thus, an appreciation of the ways in which some men might have different corporeal aspirations from women, from the medical profession and sometimes from other men is important for the practice of promoting nutritional health, as it must logically follow that each of these distinctly valued outcomes will require different approaches. One question is whether such approaches should attempt to correct lay perspectives or cultural influences that contradict the medical evidence and advice and thereby risk alienation and ineffective or irrelevant intervention - or work within existing value frameworks to increase the salience of health promotion messages. One example of the latter might be the 'health at every size movement' [2, 126], which questions the legitimacy and focus of purely weight-based programmes and concentrates instead on improving markers of health and fitness.

Promoting Nutrition in Men's Health

After considering the ways in which food and male bodies play a large part in constructing gender identity, thoughts may well turn to 'what works' with regard to promoting nutritional health. The research does not always give a consistent or conclusive answer to this question, and perhaps suggests two seemingly contradictory approaches. The first of these is to work within normative cultural frameworks and utilise the common markers of masculinity as a 'hook' to draw men in. This technique, often based around a simplistic semantic reframing, is exemplified in the mechanistic metaphors of the HGV manuals already mentioned but also includes the sporting language employed within sportbased weight management programmes, such as Premier League Fitness (Pringle et al. 2011; Zwolinsky et al. 2012), Fit Fans [13], Football Fans in Training [72] and Motivate [132].

Tackle unhealthy habits. Avoid the **Penalties** of high sugar... **Convert** to a healthier lifestyle. Excerpt from Fit Fans marketing leaflet (emphasis in the original).

Equally, the use of 'male-friendly' language and humour within weight management services that have successfully 'targeted' men takes a similar approach, as illustrated in the POWER project (Preventing Obesity Without Eating like a Rabbit) [106], and less subtlety in 'Gutbusters' [47] and 'The Fat Bastards Club' [65]. Such interventions aim to overcome male scepticism over health promotion food messages [58], a reluctance to seek help and resistance to being told what to do, by playing on masculine themes such as independence and autonomy. Deploying these tactics makes sense from the point of view of appealing to gender norms, whilst also having the tendency to reduce nutritional health to solitary decision making and neglecting the powerful social and structural influences discussed earlier.

Weight management is just a matter of choosing the right foods (Banks [8], section 4.11)

Knowing how powerful your thoughts are will give you another tool for your kitbag (Banks [8], section 2.6)

Another related approach that builds on existing gendered social structures and institutions, and has met with some moderate success in engaging men in nutritional health, is the use of particular delivery settings beyond feminised healthcare that appeal as masculine spaces. For example, the workplace has shown some ability to attract men for nutritional intervention in places as diverse as Australia, Japan and the USA (Ferguson et al. 2009; Iriyama and Murayama 2014; Morgan 2011). Harrsion [66] is critical however of the potential to miss the most vulnerable unemployed, long-term sick or disabled men by adopting this strategy, while most studies concentrate on blue or white collar occupations, leaving a gap in our understanding of how the workplace influences lower paid working men. In recent years in the UK, professional sport settings have increasingly provided an acceptable social arena for men to show a legitimate interest in health and nutrition. Here, masculine capital can be enhanced just through 'symbolic proximity' to the highly valued position of professional sport, and masculine value is conveyed, even to behaviours that are feminised in other contexts (Hunt et al. 2006). This can be usefully explored through Bourdieu's concepts of field, analogous to setting in many ways as a space in which social actors are located; and habitus as the conventional social practices within a field, or 'rules of the game' [18]. Within this framework, different fields offer distinct rules, traditions and etiquettes, while habitus is gendered and can result in behaviours being sanctioned or supported differently for men and women within a single field. This can help to explain, for example, why the firemen mentioned earlier might aggressively defend their communal cooking role in their workplace, but not their family setting [42]. Similarly, the group support eschewed as emotional and feminine talking shops within a slimming setting [138] might be revised as team loyalty and acceptable homosocial support in the sport setting [130]. In this scenario, behaviours are contextualised, and health promotion moves away from a focus on static traits and immutable behaviours, locating primacy instead on place and practice. Smith [140] describes sport as a 'Building site for working class masculinity'. (Page 186). While Connell [32] argues:

Sport is, all considered, astonishingly important. It is the central experience of the school years for many boys, and something which even the most determined swots have to work out their attitude to. What is learned by constant informal practice, and taught by formal coaching, is for each sport a specific combination of force and skill... the combination of the two is power. (Page 18).

Such a historical legacy brings with it a risk of reinforcing unequal power structures and further alienating the most marginalised men and women with the least power and poorest health. The result may bring about an invocation of the inverse care law which states that those who most need intervention will be least likely to receive it, and inequality is exacerbated [150]. Indeed, studies suggest some difficulties in recruiting BME, disabled, gay and 'non-sporty' men through these settings [72]. There are however notable exceptions that indicate when proactive measures are taken more marginalised men can be recruited through sport settings [168], which can protect against promoting masculinity over health [57]. The important lesson to be learnt from these exceptions is that in order to choose a strategy that does not aim to harvest the low hanging fruit, instead focusing on need over numbers and thereby addressing issues of equity, it is likely that some conscious targeting and marketing effort is required that does not occur 'naturally', at least when these popular masculinised settings are used. Again, this can be read through a Bourdieusian lens, where the concept of various forms of coping resources or 'capital' (economic, cultural, masculine, social) can impact on the range of 'choice' available. For example, well-educated, able-bodied, working men within strong social networks may have the greatest time, physical capacity, finances and emotional/social support to act on advice given.

The acceptability of these settings resonates with the literature that shows it is common for men to favour a pragmatic, action-orientated approach where emphasis is placed on what can be done (e.g. exercise) over what must be avoided (e.g. restrictive dieting). They offer a tangible connection between food and improved performance in work or sport, which in turn supports and reproduces a discourse of gender difference, and the contention that men's interest in food is different to that of women. Watson [155] offers a convincing theory of male embodiment in which men express a

preference for pragmatic bodies that meet the demands of everyday tasks, and place less value on 'visceral' aspects of the unseen biological body favoured in medical and health promotion discourse. This might go some way towards understanding a common rejection of (visceral) BMI by men in favour of larger (strong, pragmatic) body weights. Orienting towards the pragmatic and de-emphasising the visceral may again increase the salience of, and ultimately men's adherence to, health promotion food advice.

It could also be useful to consider health promotion settings theory, and how this has or has not been applied to nutritional practice. Whitelaw (2001) describes a continuum of settings approaches, starting from a 'neutral vehicle', or 'empty vessel' model in which fairly standard, individually and educationally focused interventions are delivered in exotic or novel locations. Here, the responsibility to comply with advice lies with the patient, and the social environment is largely ignored. At the far end of the spectrum structural, institutional values mostly beyond the control of individuals are seen as hugely influential on social practice therein, where cultural challenge and change is the desired outcome. It could be argued that most settings are utilised at the neutral end of the scale, and might instead be employed in the second, more radical approach to promoting nutritional health in men which challenges, rather than works within contemporary rules of gender. For example, the methods by which sport has been used as a vehicle to challenge racism, sexism and homophobia may be replicated to disrupt gendered ideas about food provision, preparation and consumption.

Promoting nutrition in men clearly requires consideration of gender/masculinity. Connell [34] argues that if gender is socially constructed, then it follows that it can be reconstructed. Gorley et al. [55] suggest that issues of power are resolved by interrupting or disrupting the habitus. This might be done through critical pedagogy and by challenging gender dichotomies, rejecting dualisms and stereotypes within nutritional resources, advice and information. For instance, rather than reinforcing the notion that slimming is a feminine activity and sport is exclusively masculine in the design of ostensibly single-sex weight management services, some crossover might be encouraged and incorporated. Proactively modelling examples of men who challenge unhealthy masculine signifiers might offer alternative points of reference, such as the teetotal professional rugby player Johnny Wilkinson whose non-hegemonic masculine abstinence is compensated for by his physical prowess [154]. It might also be possible to build upon, but divert acts of caring through food from showing affection through volume or confection, to demonstrating social bonds by selecting and preparing healthful alternatives.

One practical example of disruption might capitalise on emerging research that indicates there can be particular points within a man's life course where he is more open to reflecting upon his own health. This might occur at a crisis point where health is compromised, or when the consequences of illness present a greater threat to masculinity than admitting 'vulnerability' or 'weakness', such as when performance in masculine domains such as work are impacted. Research is also beginning to identify more proactive male motivations to improve health through diet that go beyond crisis management when, for example, couples are family planning, where issues of fertility arise or imminent fatherhood approaches. Equally, studies in older men show a similar interest in improving food intake when maintaining function and independence in retirement, responding to the death or caring for illness in spouses. This problematises the stereotype of the reluctant male help seeker by again indicating a more complex and nuanced reality, whilst also offering new avenues and opportunities to raise the issue of nutritional health with men in the fertility clinic, through midwives and health visitors, at the school gates, at the barbers or in men's sheds.

This chapter has outlined some of the ways in which the nutritional health of men might be strongly influenced by social factors. The main thesis questions the current dominance of educational approaches that place emphasis on individual behaviours while neglecting the places, spaces and interactions that contribute to the construction of gender. It questions too, the propensity to regard men as an unsophisticated and homogeneous category to be targeted by singular approaches, and suggests instead a combination of working within and challenging our ideas about what men are and what men do. Most of all, it is argued that a one size fits all approach is unlikely to succeed in improving

men's nutritional health, and interventions that fail to consider the social as well as health enhancing potential of food may find themselves lacking any real relevance and salience in the day to day lives of men.

References

- 1. Adams C. The sexual politics of meat. Cambridge: Polity Press; 1990.
- Aphramor L. Validity of claims made in weight management research: a narrative review of dietetic articles. Nutr J. 2010;9:30.
- Anderson E. Openly gay athletes: contesting hegemonic masculinity in a homophobic environment. Gender Soc. 2002;16(6):860–77.
- Anderson E. Orthodox and inclusive masculinity: competing masculinities among heterosexual men in a feminized terrain. Sociol Perspect. 2005;48(3):337.
- Annandale E, Hunt K. Gender inequalities in health: research at the crossroads. In: Annandale E, Hunt K, editors. Gender inequalities in health. Buckingham: Open University Press; 2000. p. 1–35.
- Baerlocher MO, Verma S. Men's health research: under researched and under appreciated? Med Sci Monit. 2008;14(3):SC5–6.
- 7. Baker L, Gringart E. Body image and self-esteem in older adulthood. Ageing Soc. 2009;29(6):977.
- 8. Banks I. The HGV man manual. Reducing all large sizes: all shapes and colours. Sparkford: Haynes; 2005.
- 9. Barford A, Dorling D, Davey Smith G, Shaw M. Life expectancy: women now on top everywhere: during 2006, even in the poorest countries, women can expect to outlive men. BMJ. 2006;332(7545):808.
- Beagan B, Chapman GE, D'Sylva A, Bassett BR. 'It's just easier for me to do it': rationalizing the family division of foodwork. Sociology. 2008;42(4):653–71.
- Beardsworth A, Bryman A, Keil T, Goode J, Haslam C, Lancashire E. Women, men and food: the significance of gender for nutritional attitudes and choices. Br Food J. 2002;104(7):470–91.
- Bennett E, Gough B. In pursuit of leanness: the management of appearance, affect and masculinities within a men's weight loss forum. Health. 2013;17(3):284–99.
- Bingham D, Parnell D, Curran K, Jones R, Richardson D. Fit fans: perspectives of a practitioner and understanding participant health needs within a health promotion programme for older men delivered within an English Premier League Football Club. Soccer Soc. 2014;15(6):883–901.
- 14. Bish CL, Blanck HM, Serdula MK, Marcus M, Kohl HW, Khan LK. Diet and physical activity behaviors among Americans trying to lose weight: 2000 behavioral risk factor surveillance system. Obes Res. 2005;13(3):596–607.
- 15. Bisogni CA, Connors M, Devine CM, Sobal J. Who we are and how we eat: a qualitative study of identities in food choice. J Nutr Educ Behav. 2002;34:128–39.
- 16. De Bourdeaudhuij I, Van Oost P. Personal and family determinants of dietary behaviour in adolescents and their parents. Psychol Health. 2000;15(6):751.
- 17. Bottamini G, Ste-Marie DM. Male voices on body image. Int J Men Health. 2006;5(2):109-32.
- 18. Bourdieu P. Distinction: a social critque of the judgement of taste. Cambridge: Havard University Press; 1984.
- 19. Bove CF, Sobal J, Rauschenbach BS. Food choices among newly married couples: convergence, conflict, individualism, and projects. Appetite. 2003;40(1):25–41.
- Boyle E, Brayton S. Ageing masculinities and "muscle work" in hollywood action film: an analysis of the expendables. Men Masc. 2012;15(5):468–85.
- Broom DH, Dixon J. The sex of slimming: mobilizing gender in weight-loss programmes and fat acceptance. Soc Theory Health. 2008;6(2):148–66.
- 22. Brug J, Wammes B, Kremers S, Giskes K, Oenema A. Underestimation and overestimation of personal weight status: associations with socio-demographic characteristics and weight maintenance intentions. J Hum Nutr Diet. 2006;19(4):253–62.
- Cafri G, van den Berg P, Thompson JK. Pursuit of muscularity in adolescent boys: relations among biopsychosocial variables and clinical outcomes. J Clin Child Adolesc Psychol. 2006;35(2):283–91.
- 24. Caplan P, Keane A, Willetts A, Williams J. Studying food choice in its social and cultural contexts: approaches from a social anthropological perspective. In: Murcott A, editor. The nation's diet: the social science of food choice. New York: Pearson Education; 1998. p. 168–96.
- Carpenter KM, Hasin DS, Allison DB, Faith MS. Relationships between obesity and DSM-IV major depressive disorder, suicide ideation, and suicide attempts: results from a general population study. Am J Public Health. 2000;90(2):251–7.

- 26. Carrigan T, Connell B, Lee J. Toward a new sociology of masculinity. Theory Soc. 1985;14(5):551–604.
- 27. Chang VW, Christakis NA. Self-perception of weight appropriateness in the United States. Am J Prev Med. 2003;24(4):332–9.
- Charles N, Kerr M. Just the way it is: gender and age differences in family food consumption. In: Brannen J, Wilson G, editors. Give and take in families: studies in resource distribution. Boston: Allen and Unwin; 1987. p. 155–74.
- 29. Charles N. Gender in modern Britain. Oxford: Oxford University Press; 2002.
- 30. Chernin K. The obsession: reflections on the tyranny of slenderness. New York: Harper Colophon; 1981.
- 31. Christensen VT. Gendered perceptions of own and partner weight-level. Health. 2012;16(4):382–99.
- 32. Connell RW. Which way is up? London: George Allen and Unwin; 1983.
- 33. Connell RW. Gender and power. Oxford: Polity Press; 1987.
- 34. Connell RW. Masculinities. 2nd ed. Cambridge: Polity; 1995.
- 35. Counihan C, Van Esterik P. Food and Culture. Counihan C, Van Esterik P. editors. London: Routledge; 1997.
- 36. Courtenay WH. Constructions of masculinity and their influence on men's well-being: a theory of gender and health. Soc Sci Med. 2000;50(10):1385–401.
- Coveney J. What does research on families and food tell us? Implications for nutrition and dietetic practice. Nutr Diet. 2002;59(2):113.
- 38. Crawford R. Health as a meaningful social practice. Health. 2006;10(4):401-20.
- Crawshaw P. Governing the healthy male citizen: men, masculinity and popular health in men's health magazine. Soc Sci Med. 2007;65(8):1606–18.
- 40. David D, Brannon R. In: David D, Brannon R, editors. The forty nine percent majority: the male sex role. London: Addison-Wesley Publishing Company; 1976.
- Delormier T, Frohlich K, Potvin L. Food and eating as social practice understanding eating patterns as social phenomena and implications for public health. Sociology of Health & Illness 2, 2009;31(2):215–28.
- 42. Deutsch J. "Please pass the chicken tits": rethinking men and cooking at an urban firehouse. Food Foodways. 2005;2(13):91–114.
- 43. DeVault M. Feeding the family. Chicago: The University of Chicago Press; 1991.
- 44. Drummond M. Reflections on the archetypal heterosexual male body. Austr Fem Stud. 2011;26(67):103–17.
- Drummond M, Smith J. Ageing men's understanding of nutrition: implications for health. J Mens Health Gend. 2006;3(1):56–60.
- 46. Edward AR. Sex roles: a problem for sociology and for women. J Sociol. 1983;19(3):385.
- 47. Egger G. Intervening in men's nutrition: lessons from the GutBuster men's 'waist loss' program. Aust J Nutr Diet. 2000;57(1):46–9.
- 48. Feirstein B. Real men don't eat quiche: a guidebook to all that is truly masculine. New York: Pocket Books; 1982.
- 49. Fraser GE, Welch A, Luben R, Bingham SA, Day NE. The effect of age, sex, and education on food consumption of a middle-aged English cohort EPIC in East Anglia. Prev Med. 2000;30(1):26–34.
- Frederick D, Forbes GB, Grigorian KE, Jarcho JM. The UCLA body project I: gender and ethnic differences in self-objectification and body satisfaction among 2,206 undergraduates. Sex Roles. 2007;57(5–6):317–27.
- Furnham A, Calnan A. Eating disturbance, self-esteem, reasons for exercising and body weight dissatisfaction in adolescent males. Eur Eat Disord Rev. 1998;6(1):58–72.
- 52. Fürst EL. Cooking and femininity. Womens Stud Int Forum. 1997;20(3):441-9.
- 53. Galli N, Reel JJ. Adonis or Hephaestus? exploring body image in male athletes. Psychol Men Masc. 2009;10(2):95–108.
- Germov J, Williams L. A sociology of food and nutrition: The social appetite. In: Germov J, Williams L, editors. Oxford: Oxford University Press; 1999.
- 55. Gorely T, Holroyd R, Kirk D. Muscularity, the habitus and the social construction of gender: towards a genderrelevant physical education. Br J Sociol Educ. 2003;24(4):429–48.
- 56. Gough B. "Real men don't diet': an analysis of contemporary newspaper representations of men, food and health. Soc Sci Med. 2007;64(2):326–37.
- 57. Gough B. Promoting 'masculinity' over health: a critical analysis of men's health promotion with particular reference to an obesity reduction 'manual'. In: Gough B, Robertson S, editors. Men, masculinities and health: critical perspectives. Basingstoke: Palgrave; 2009.
- Gough B, Conner MT. Barriers to healthy eating amongst men: a qualitative analysis. Soc Sci Med. 2006;62(2):387–95.
- Gough B, Edwards G. The beer talking: four lads, a carry out and the reproduction of masculinities. Sociol Rev. 1998;46(3):409–55.
- 60. Gough B, Flanders G. Celebrating "obese" bodies: gay "bears" talk about weight, body image and health. Int J Mens Health. 2009;8(3):235–53.
- Graham H. Being poor: perceptions and coping strategies. In: Brannen J, Wilson G, editors. Give and take in families: studies in resource distribution. London: Allen and Unwin; 1987. p. 56–75.

- 62. Grogan S, Richards H. Body image: focus groups with boys and men. Men Masc. 2002;4(3):219.
- Gregory CO, Blanck HM, Gillespie C, Maynard ML, Serdula MK. Perceived health risk of excess body weight among overweight and obese men and women: differences by sex. Prev Med. 2008;47(1):46–52.
- 64. Hargreaves J. Sport, power and culture. Oxford: Polity; 1986.
- 65. Harrison A. Weight management in the workplace. In: Conrad D, White A, editors. Men's health: how to do it. Oxford: Radcliffe Publishing Ltd; 2007.
- 66. Harrison J. Warning: the male sex role may be dangerous to your health. J Soc Issues. 1978;34(1):65-86.
- 67. Hennen P. Bear bodies, bear masculinity recuperation, resistance, or retreat? Gend Soc. 2005;19(1):25–43.
- 68. Herman CP, Roth DA, Polivy J. Effects of the presence of others on food intake: a normative interpretation. Psychol Bull. 2003;129(6):873.
- 69. Hollows J. Oliver's twist: leisure, labour and domestic masculinity in the naked chef. Int J Cult Stud. 2003;6(2):229-48.
- HM Government. Equality act. London. 2010. Available at: http://www.legislation.gov.uk/ukpga/2010/15/pdfs/ ukpga_20100015_en.pdf.
- 71. Hunt K, Annandale E. Relocating gender and morbidity: examining men's and women's health in contemporary Western societies. Introduction to special issue on gender and health. Soc Sci Med. 1999;48(1):1–5.
- 72. Hunt K, Wyke S, Gray C, Anderson A, Brady A, Bunn C, Donnan P, Fenwick E, Grieve E, Leishman J, Miller E, Mutrie N, Rauchhaus P, White A, Treweek S. A gender-sensitised weight loss and healthy living programme for overweight and obese men delivered by Scottish Premier League football clubs (FFIT): a pragmatic randomised controlled trial. Lancet. 2014;383(9924):1211–21.
- HSCIC. The health survey for England 2008: latest trends. London. 2009. Available at: http://www.hscic.gov.uk/ catalogue/PUB00455.
- HSCIC. Health survey for England 2012. London. 2012. Available at: http://www.hscic.gov.uk/catalogue/ PUB13218.
- Isomaa R, Isomaa A, Marttunen M, Kaltiala-Heino R, Bjorkqvist K. Longitudinal concomitants of incorrect weight perception in female and male adolescents. Body Image. 2011;8(1):58–63.
- Jebb SA, Aveyard PN, Hawkes C. The evolution of policy and actions to tackle obesity in England. Obes Rev. 2013;14:42–59.
- 77. Jones WK. Men's health as a public health issue. J Mens Health Gend. 2004;1(2-3):147-9.
- 78. Johnson-Taylor WL, Fisher RA, Van Hubbard S, Starke-Reed P, Eggers PS. The change in weight perception of weight status among the overweight: comparison of NHANES III (1988–1994) and 1999–2004 NHANES. Int J Behav Nutr Phys Act. 2008;5:9.
- 79. Julier A, Lindenfield L. Mapping men onto the menu: masculinities and food. Food Foodways. 2005;13:1-16.
- Kiefer I, Rathmanner T, Kunze M. Eating and dieting differences in men and women. J Mens Health Gend. 2005;2(2):194–201.
- Krauss K. Male beauty: postwar masculinity in theatre, film and physique magazines. Albany: State of New York University Press; 2013.
- Kuchler F, Variyam JN. Mistakes were made: misperception as a barrier to reducing overweight. Int J Obes (Lond). 2003;27(7):856–61.
- Kuk JL, Ardern CI, Church TS, Hebert JR, Sui X, Blair SN. Ideal weight and weight satisfaction: association with health practices. Am J Epidemiol. 2009;170(4):456–63.
- 84. Lake AA, Hyland RM, Mathers JC, Rugg-Gunn AJ, Wood CE, Adamson AJ. Food shopping and preparation among the 30-somethings: whose job is it? (the ASH30 study). Br Food J. 2006;108(6):475–86.
- Lang T, Rayner G. Overcoming policy cacophony on obesity: an ecological public health framework for policymakers. Obes Rev. 2007;8:165–81.
- Leishman J. Working with men in groups. In: White A, Pettifer M, editors. Hazardous waist: tackling male weight problems. Oxford: Radcliffe Publishing Ltd; 2007.
- Leit RA, Pope HG, Gray JJ. Cultural expectations of muscularity in men: the evolution of playgirl centerfolds. Int J Eat Disord. 2001;29(1):90–3.
- 88. Lemle R, Mishkind ME. Alcohol and masculinity. J Subst Abuse Treat. 1989;6(4):213-22.
- Levi A, Chan KK, Pence D. Real men do not read labels: the effects of masculinity and involvement on college students' food decisions. J Am Coll Health. 2006;55(2):91.
- Lohan M. How might we understand men's health better? Integrating explanations from critical studies on men and inequalities in health. Soc Sci Med. 2007;65(3):493–504.
- Marmot M. Fair society, healthy lives. Strategic review of health inequalities in England post. The Marmot review. 2010. http://www.instituteofhealthequity.org/projects/fair-society-healthy-lives-the-marmot-review. Accessed 31 Jan 2016
- 92. Mallyon A, Holmes M, Coveney J, Zadoroznyj M. I'm not dieting, 'I'm doing it for science': masculinities and the experience of dieting. Health Sociol Rev. 2010;19(3):330–42 13p. [serial on the Internet] [cited January 31, 2016].

- Martin MA, Frisco ML, May AL. Gender and race/ethnic differences in accurate weight perceptions among U.S. adolescents. Womens Health Issues. 2009;19(5):292–9.
- 94. Martin MA, May AL, Frisco ML. Equal weights but different weight perceptions among US adolescents. J Health Psychol. 2010;15(4):493–504.
- McCabe MP, Ricciardelli LA. Body image dissatisfaction among males across the lifespan. A review of past literature. J Psychosom Res. 2004;56:675–85.
- 96. McCreary DR, Hildebrandt TB, Heinberg LJ, Boroughs M, Thompson KJ. A review of body image influences on men's fitness goals and supplement use. Am J Mens Health. 2007;1(4):307–16.
- 97. Meryn S, Jadad AR. The future of men and their health: are men in danger of extinction? Br Med J. 2001;323(7320):1013.
- 98. Meryn S, Shabsigh R. Men's health: past, present and future. J Mens Health. 2009;6(3):143-6.
- 99. Miniño A, Heron M, Murphy S, Kochanek K. Deaths: final data for 2004. Natl Vital Stat Rep. 2007; 55(19):1–119.
- Mishkind ME, Rodin J, Silberstein LR, Striegelmoore RH. The embodiment of masculinity cultural, psychological and behavioural dimensions. Am Behav Sci. 1986;29(5):545–62.
- 101. Monaghan LF. Big handsome men, bears and others: virtual constructions of "fat male embodiment.". Body Soc. 2005;11(2):81–111.
- 102. Monaghan LF. Body mass index, masculinities and moral worth: men's critical understandings of "appropriate" weight-for-height. Sociol Health Illn. 2007;29(4):584–609.
- Monaghan LF. McDonaldizing men's bodies? Slimming, associated (Ir)rationalities and resistances. Body Soc. 2007;13(2):67.
- 104. Monaghan LF. Men and the war on obesity: a sociological study. New York: Routledge; 2008.
- 105. Mori D, Pliner P, Chaiken S. "Eating lightly" and the self-presentation of femininity. J Pers Soc Psychol. 1987;53(4):693.
- 106. Morgan PJ, Collins CE, Plotnikoff RC, Cook AT, Berthon B, Mitchell S, Callister R. Efficacy of a workplacebased weight loss program for overweight male shift workers: the workplace POWER (preventing obesity without eating like a rabbit) randomized controlled trial. Prev Med. 2011;52(5):317–25.
- 107. Mroz LW, Chapman GE, Oliffe JL, Bottorff JL. Men, food, and prostate cancer: gender influences on men's diets. Am J Mens Health. 2011;5(2):177–87.
- Mróz LW, Chapman GE, Oliffe JL, Bottorff JL. Gender relations, prostate cancer and diet: re-inscribing heteronormative food practices. Soc Sci Med. 2011;72:1499–506.
- 109. Muennig P, Jia H, Lee R, Lubetkin E. Think therefore I am: perceived ideal weight as a determinant of health. Am J Public Health. 2008;98(3):501.
- 110. Murcott A. On the social significance of the "cooked dinner" in South Wales. Soc Sci Inform. 1982; 21(4–5):677–96.
- 111. Murcott A. The nations diet: The social science of food choice. In: Murcott A. editor. Harlow: Addison-Wesley Longman Limited; 1998.
- 112. Oakley A. Sex, gender and society. Aldershot: Gower House Publishing Company Limited; 1972. Revised.
- 113. Oates CJ, McDonald S. Recycling and the domestic division of labour: is green pink or blue? Sociology. 2006;40(3):417-33.
- O'Doherty Jensen K, Holm L. Preferences, quantities and concerns: socio-cultural perspectives on the gendered consumption of foods. Eur J Clin Nutr. 1999;53(5):351–9.
- 115. Orbach S. Fat is a feminist issue. London: Arrow Books; 1978.
- Pagoto SL, Schneider KL, Oleski JL, Luciani JM, Bodenlos JS, Whited MC. Male inclusion in randomized controlled trials of lifestyle weight loss interventions. Obesity. 2012;20(6):1234–9.
- 117. Pope HG, Olivardia R, Gruber A, Borowiecki J. Evolving ideals of male body image as seen through action toys. Int J Eat Disord. 1999;26(1):65–72.
- 118. Pope HG, Phillips K, Olivardia R. The Adonis complex: the secret crisis of male body obsession. New York: The Free Press; 2000.
- 119. Riska E. The rise and fall of type A man. Soc Sci Med. 2000;51(11):1665–74.
- 120. Riska E. From type A man to the hardy man: masculinity and health. Sociol Health Illn. 2002;24(3):347-58.
- 121. Robertson C, Archiblad D, Avenell A, Douglas F, Hoddinott P, van Teijlingen E, Boyers D, Stewart F, Boachie C, Fioratou E, Wilkins D, Street T, Carroll P, Fowler C. Systematic reviews of and integrated report on the quantitative, qualitative and economic evidence base for the management of obesity in men. Health Technol Assess. 2014;18(35):v–vi, xxiii-xxix, 1-424.
- 122. Robertson S. Understanding men and health: masculinities, identity and well-being. Maidenhead: Open University Press; 2007.
- 123. Robertson S, White A. Tackling men's health: a research, policy and practice perspective. Public Health. 2011;125(7):399–400.

- 124. Robertson S, Williams R. Men, public health and health promotion: towards a critically structural and embodied understanding. In: Gough B, Robertson S, editors. Men, masculinities and health. Critical perspectives. Basingstoke: Palgrave Macmillan; 2010.
- 125. Robertson Textor A. Organisation, specialization, and desires in the big men's movement: preliminary research in the study of sub-culture-formation. J Gay Lesb Bisexual Identity. 1999;4(3):217–39.
- 126. Robison J. Health at every size: toward a new paradigm of weight and health. Med Gen Med. 2005;7(3):13.
- 127. Roos E, Lahelma E, Virtanen M, Prattala R, Pietinen P. Gender, socioeconomic status and family status as determinants of food behaviour. Soc Sci Med. 1998;46(12):1519–29 11p [serial on the Internet].
- 128. Roos G, Prattala R, Koski K. Men, masculinity and food: interviews with Finnish carpenters and engineers. Appetite. 2001;37(1):47–56.
- 129. Rothgerber H. Real men don't eat (vegetable) quiche: masculinity and the justification of meat consumption. Psychol Men Masc. 2013;14(4):363–75.
- Rowlands S. Men's experiences and the construction of masculinities within NHS weight management services. PhD thesis, Unpublished. 2015.
- 131. Ruby MB, Heine SJ. Meat, morals, and masculinity. Appetite. 2011;56(2):447-50.
- 132. Rutherford Z, Seymour-Smith S, Matthews C, Gough B, et al. "Motivate": the effect of a football in the community delivered weight loss programme on over 35 year old men and women's cardiovascular risk factors. Soccer Soc. 2014;15(5):951.
- Sabinsky MS, Toft U, Raben A, Holm L. Overweight men's motivations and perceived barriers towards weight loss. Eur J Clin Nutr. 2007;61(4):526–31.
- 134. Saguy A. Why fat is a feminist issue. Sex Roles. 2012;66(9-10):600-7.
- 135. Schafer RB, Schafer E, Dunbar M, Keith PM. Marital food interaction and dietary behavior. Soc Sci Med. 1999;48(6):787–96.
- 136. Sellaeg K, Chapman GE. Masculinity and food ideals of men who live alone. Appetite. 2008;51(1):120-8.
- 137. Segal L. Slow motion: changing masculinities changing men. 2nd ed. London: Virago; 1997.
- 138. Seymour-Smith S. Men's negotiations of a "legitimate" self help group identity. In: Gough B, Robertson S, editors. Men, masculinities and health. Critical perspectives. London: Palgrave Macmillan; 2010.
- 139. Shilling C. The body and social theory. London: Sage Publications Ltd; 1993.
- 140. Smith J. 'Ye've got to 'ave balls to play this game sir!' boys, peers and fears: the negative influence of schoolbased 'cultural accomplices' in constructing hegemonic masculinities. Gend Educ. 2007;19(2):179–98.
- Smolak L, Murnen SK, Thompson JK. Sociocultural influences and muscle building in adolescent boys. Psychol Men Masc. 2005;6(4):227–39.
- 142. Sobal J. Men, meat, and marriage: models of masculintiy. Food Foodways. 2005;13:135-58.
- 143. Sonntag U, Henkel J, Renneberg B, Bockelbrink A, Braun V, Heintze C. Counseling overweight patients: analysis of preventive encounters in primary care. International J Qual Health Care. 2010;22(6):486–92.
- 144. De Souza P, Ciclitira KE. Men and dieting: a qualitative analysis. J Health Psychol. 2005;10(6):793–804.
- 145. Stibbe A. Health and the social construction of masculinity in men's health magazine. Men Masc. 2004;7(1):31–51.
- Stinson K. Women and dieting culture: inside a commerical weight loss group. London: Rutgers University Press; 1991.
- 147. Sullivan O. The division of domestic labour: twenty years of change? Sociology. 2000;34(3):437.
- 148. Szabo M. Foodwork or foodplay? men's domestic cooking, privilege and leisure. Sociology. 2013;47(4):623–38.
- 149. Thompson EH, Pleck JH. The structure of male role norms. Am Behav Sci. 1986;29(5):531–43.
- 150. Tudor Hart J. Inverse care law. Lancet. 1971;1(7696):405.
- 151. Turner B. Regulating bodies: essays in medical sociology. London: Routledge; 1992.
- Vartanian LR, Herman CP, Polivy J. Consumption stereotypes and impression management: how you are what you eat. Appetite. 2007;48(3):265–77.
- 153. de Visser RO, Smith JA. Alcohol consumption and masculine identity among young men. Psychol Health. 2007;22(5):595–614.
- 154. de Visser RO, Smith JA, McDonnell EJ. "That's not masculine': masculine capital and health-related behaviour. J Health Psychol. 2009;14(7):1047–58.
- 155. Watson J. Male bodies. Health, culture and identity. Buckingham: Open University Press; 2000.
- 156. Wardle J, Johnson F. Weight and dieting: examining levels of weight concern in British adults. Int J Obes Relat Metab Disord. 2002;26(8):1144.
- 157. Wellard I. Sport, masculinities and the body. New York: Routledge; 2009.
- 158. West C, Zimmerman D. Doing gender. Gend Soc. 1987;1(2):125-51.
- 159. White A. Male obesity: policy and context. In: White A, Pettifer M, editors. Hazardous waist: tackling male weight problems. Oxford: Radcliffe Publishing Ltd; 2007.
- 160. White A, Cash K. The state of men's health in Western Europe. J Mens Health Gend. 2004;1(1):60–6.

- 161. White A, de Souza B, de Visser R, Hogston R, Madsen SA, Makara P, McKee M, Raine G, Richardson N, Clarke N, Zatonski W. The state of men's health in Europe. 2011. Available at: http://ec.europa.eu/health/population_groups/docs/men_health_extended_en.pdf.
- White A, Holmes M. Patterns of mortality across 44 countries among men and women aged 15–44 years. J Mens Health Gend. 2006;2:139.
- Whitesel J, Shuman A. Normalizing desire: stigma and the carnivalesque in gay bigmen's cultural practices. Men Masc. 2013;16(4):478–96.
- 164. UN. The millennium development goals report 2014. New York. 2014. Available at: http://www.un.org/ millenniumgoals/2014MDGreport/MDG2014Englishweb.pdf.
- 165. Uy JP, Massoth AN, Gottdiener HW. Rethinking male drinking: traditional masculine ideologies, gender-role conflict, and drinking motives. Psychol Men Masc. 2014;15(2):121–8.
- 166. Yang CFJ, Gray P, Pope HG. Male body image in Taiwan versus the West: Yanggang Zhiqi meets the adonis complex. Am J Psychiatry. 2005;162(2):263–9.
- 167. Young MD, Morgan PJ, Plotnikoff RC, Callister R, Collins C. Effectiveness of male-only weight loss and weight loss maintenance interventions: a systematic review with meta-analysis. Obes Rev. 2012;13(5):393–408.
- 168. Zwolinsky S, McKenna J, Pringle A, Daly-Smith A, Robertson S, White A. Optimizing lifestyles for men regarded as "hard-to-reach" through top-flight football/soccer clubs. Health Educ Res. 2013;28(3):405–13.

Chapter 18 Nutritional Considerations for Hispanics

Diana K. Cuy Castellanos

Key Points

- Hispanics are the largest ethnic minority group in the USA.
- Health disparities are apparent in this population, such as high rates of obesity and diabetes and low access to healthcare.
- Dietary behaviors are different across Hispanic subgroups.
- To effectively address Hispanics' undesirable health behaviors, health practitioners must increase their understanding of the range of values, beliefs, knowledge, preferences, and attitudes of each Hispanic person or subgroup.
- The traditional Hispanic diet has often been considered healthier than the Western diet. However, in the midst of the global nutrition transition, many Hispanics have adopted Western dietary habits before migrating to the USA.
- A thorough assessment of each person's or each subgroup's dietary behaviors and influencing factors is essential in order to provide culturally appropriate nutrition education or therapy.
- Several nutrition interventions have been successful in positively influencing Hispanics' dietary behavior.

Keywords Dietary acculturation • Hispanic • Latino • Health Disparities • Dietary intervention

The Hispanic Population in the USA

As of 2014, 17.4% of the US population was Hispanic, and about 40.0% of all Hispanics residing in the USA were foreign-born [107]. Between 2010 and 2014, the USA experienced a 9% increase in Hispanics. Further, the Hispanic population accounted for 49% of the population increase between these 4 years; 26% of the increase was due to migration, and 74% was accounted for by birth in the USA [107].

D.K.C. Castellanos

Health and Sport Science, University of Dayton, Dayton, OH, USA e-mail: dcuycastellanos1@udayton.edu

As defined by the US Office of Management and Budget [109], Hispanic is "a person of Cuban, Mexican, Puerto Rican, South or Central American, or other Spanish culture or origin, regardless of race." (para. 14). Of the over 55 million Hispanics in the USA, the majority are from Mexico, representing about 64.3%. Other populations represented in the USA include Puerto Rico (9.3%), Cuba (3.5%), Central American (8.1%), South America (5.9%), and other (8.9%). Females represent approximately 50.8% and males 49.2% of all Hispanics in the USA [107].

Currently, the majority of Hispanics reside in the Southwestern states. However, all states are experiencing increases in the Hispanic population. For example, New Mexico (47.3%), California (38.4%), Texas (38.4%), Arizona (30.3%), and Nevada (27.5) represent the top five states with the highest percentages of Hispanic residents [107]. In terms of the largest number of Hispanics, about 14.7 million Hispanics reside in California, and Texas experienced the highest increase in number at 213,000 since 2012 [107]. More state and county Hispanic population information can be found at www.census.gov.

Hispanic Socioeconomic Factors

When compared to other ethnic groups residing in the USA, the Hispanic population at large experiences more income and educational disparities. For instance, among ethnic groups, the Hispanic population has the highest number of non-high school graduates (35.0%) and the lowest number of people (14.5%) who have a bachelor's degree or higher [108].

Hispanics are second lowest only to Blacks in income [25]. The median annual income for a Hispanic household in the USA is approximately \$42,000, compared to the median US household's annual income of about \$53,657. In 2014, 23.6% of Hispanics lived below the national poverty line. Interestingly, the median income increased between 2013 and 2014 for foreign-born Hispanics but decreased for native-born. Moreover, because 80.1% of Hispanics in 2014 had governmental or private health insurance, 19.9% of Hispanics were uninsured – the highest percentage of uninsured among ethnic groups. Of those who were insured, native-born Hispanics at 78.6% [99]. One study reported only 10.5% of undocumented Hispanics have health insurance [74].

The unemployment rate for Hispanics is slightly higher than that of the overall US population. At the end of 2014, the Hispanic population had a 6.4% unemployment rate, compared to 5.1% of the overall US population. Of those employed, the majority of Hispanics worked in service occupations [107]. When the percentages of Hispanic employees in different occupations are examined, statistics show that a high concentration of Hispanics work in construction, agricultural forestry, farming and fisheries, and hospitality, while Hispanics are underrepresented in the financial, information, and public administration employment categories [107].

Hispanic Culture

Culture can be defined as "the attitudes, feelings, values and behavior that characterize and inform society as a whole or any social group within it" (*Collins English Dictionary* def. 2). It is a collaboration of ideas and behaviors shared and reinforced within a particular group or society. Regarded in this perspective, the Hispanic culture as a whole is complex and is comprised of various subgroups. For this chapter, however, the Hispanic culture will be discussed in a broad context and thus should not be generalized to particular Hispanic subgroups and individuals.

Cultural Characteristic

There are several inherent cultural differences between the general Hispanic and US cultures. It is important that health practitioners be aware of such differences to be able to understand and relate to a cultural context for certain behaviors. In general, the Hispanic culture is collectivistic while the US is individualistic. In a collectivistic culture, the focus is on the group; therefore, responsibilities and decision-making are shared, group activities dominate, and there is an emphasis on group harmony, not the individual. In comparison, the US cultural focus is on the individual's functionality and his or her ability to work and live independently [44]. In light of such different emphases, it would not be uncommon for a Hispanic patient not only to bring several family members to a consultation, but also to allow the family members to determine the best care plan.

The Hispanic culture is also relationship-oriented, unlike the time-oriented US culture. Relationship orientation is clearly interconnected with the collectivistic culture, placing more emphasis on relationships than on time considerations. Thus, for example, if a Hispanic male were helping a friend fix his car but was supposed to go to a doctor's appointment at say, 4:00 p.m., he would not leave the friend and the friend's car to ensure he arrived on time for the appointment. The relationship with the person he was with in that moment would take priority. In his mind, his arriving late to a party or an appointment is acceptable. Within the US culture, this may be seen as rude, disrespectful, and inconsiderate, while the Hispanic man would find it more inappropriate to leave his friend.

In fact, Hispanics' time orientation leans more toward the present moment and is more reactionary, compared to the time orientation of people within the US culture, who live in the future and reflect more proactive behavior. This reflects back on the hypothetical situation just presented. If the Hispanic male is helping his friend fix his car and he has another appointment, he is not thinking foremost of how important it is to arrive at his appointment on time; he is focusing instead on helping his friend and finishing the task at hand, living in that present moment and concentrating on that more immediate task. Within the Hispanic culture, making appointments is simply not common. For medical visits in particular, it is more common for Hispanics to go to the practitioner's office without an appointment at all, simply when needed, and then to wait until they can be seen. The culture is reactionary.

Communication is also different between the two cultures. In the US culture, direct and verbal communication is the norm and the accepted mode. In the Hispanic culture, indirect and nonverbal communication is most common. Again this may be stem from the cultural orientation toward relationships. Within a healthcare setting, the Hispanic patient may consider it rude if the practitioner does not participate in small talk for a few minutes before the appointment or the examination. When working with Hispanics, therefore, healthcare practitioners need to adjust to such cultural tendencies in order to better relate to and better care for their patients. Along the same lines, Hispanics often utilize nonverbal cues to communicate. For example, if asked a direct question, the Hispanic person may not answer directly or honestly but in a form he or she feels the person asking the question wants. And then if there is a language barrier and the Hispanic person does not understand the question, many times the Hispanic individual will just answer yes (or no) instead of asking for clarification. Thus it is advisable that health practitioners ask more openended questions to their Hispanic patients. Further, if a Hispanic person wants something, he or she may ask for it indirectly. For example, if the person needs a ride to pick up a prescription at the pharmacy, instead of directly asking a friend for a ride, the person may say, "I can't buy my medicine," hoping the friend would then ask why and then ask if the individual needs a ride. As a result of being aware of such cultural approaches, a savvy health practitioner will no longer simply ask a Hispanic patient if there are any questions or if anything is needed; a more roundabout approach is called for.

Other Cultural Characteristics and Considerations in the Context of Health Care

Cuellar et al. [17] outlined five cultural characteristics in the Hispanic culture that may affect health behaviors: machismo, folk illness, familism, fatalism, and personalismo. Table 18.1 provides definitions and context for each of these characteristics. Assessing whether an individual or subgroup reflects one of more of these characteristics is important so the practitioner can provide information or care that is appropriate. For example, if a client says, "I have diabetes because it is God's will," this comment may indicate the client does not believe his own actions contributed to the disease and is demonstrating the characteristic of fatalism. Picking up on this, a practitioner can then ask the client what specific actions (i.e., dietary changes) he or she believes might help control the disease, assessing any ambivalence in the client's answer to determine best methods for motivating the client to change negative health behavior.

Complicating the situation is the Hispanic culture's division into different subcultures. Subcultures may be influenced by such factors as nativity, country of origin, or race (white, black, or indigenous). One study showed differences in the perception of health between Hispanics who were Mexican or American-born and those who identified as Mayan. Foreign-born Mexicans and Mayans perceived health as something a person has to work for and is seen as essential to work and provide for family. In contrast, US-born Mexicans saw health as a right, as something that should be available to everyone, and as something that can be purchased [101].

It is important, of course, that health practitioners know the more particular cultural context of each patient or subgroup. Awareness of an overall Hispanic cultural context is good only to a certain extent; to avoid making overarching cultural assumptions, practitioners in the health field must always be alert for adjustments when subcultural differences come to light. Knowledge of and sensitivity to the various subgroups' cultural tendencies is required.

Characteristic	Definition	Description
Machismo	Strong adherence to manliness	Can have positive or negative associations such as bravery, courageousness, womanizer, or dominant. Males often have a strong influence on the family's decisions such as diet and types of service to seek
Folk illness	Nonscientific culture specific illnesses	Illness believed to be caused by supernatural powers. For example, "susto" is a condition where an individual's spirit is temporarily scared way post traumatic event
Fatalism	The belief that an individual's fate is out of his or her control	Since individuals believe that they have no personal control over their fate such as which illnesses he or she may experience, it can affect the degree of health care they seek
Famalism	Core value of Hispanic culture that requires family members to support on each other	Family members have an obligation to support each other emotionally and physically and to abide by all of the family practices and tendencies. This impacts who/ when individuals seek health information
Personalismo	Friendly way to relate to an individual	Respond better to human/personal contact than impersonal contact, for example health services that involve therapists instead of reading literature are often more effective

Table 18.1 Characteristics associated with the Hispanic culture

332

Cuellar et al. [17]

Acculturation

After examining different aspects of the Hispanic culture, we must then explore the acculturation process of Hispanics residing in the USA. *Acculturation* is a term used for the "psychological and social changes that groups and individuals experience when they enter a new and different cultural context" ([11], p.128). The acculturation process includes the adoption of and interaction with the host culture's attitudes, beliefs, values, and behaviors. Berry [9] suggested that acculturation was a process involving individuals and groups and was not stagnant. In such a view, each Hispanic individual, foreign- or native-born, has a different acculturation experience and adopts different attributes of the host culture (in this case, the US culture) at differing degrees. Figure 18.1 outlines individual, group, and mediating factors involved in the acculturation process.

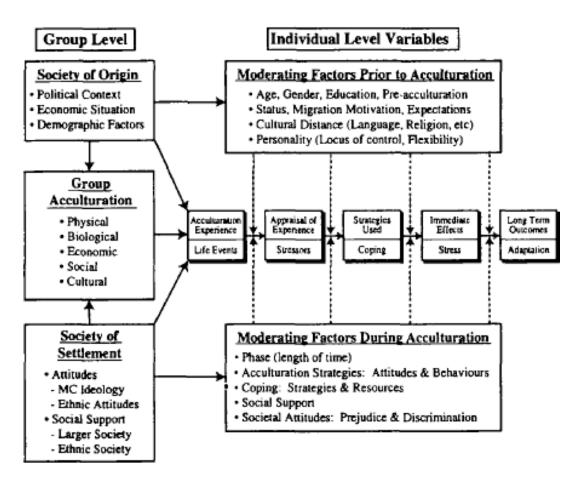


Fig. 18.1 A framework for acculturation research (Berry [9])

Acculturation Measures

Acculturation is not a linear process, even though is it often measured as such. Proxies such as language ability (written, spoken, and/or read), language use at an interview or an interview with friends, birth place, media preference, generation, length of time in the USA, age of immigration, and length of residence have all been used to assess acculturation in the nutrition and health literature [104]. Other multi-dimensional acculturation scales have also been developed to measure the process nonlinearly, and these researchers argue that the nonlinear measurement provides a more comprehensive view of acculturation [9, 11, 17], warranting further research on the measurement of acculturation [104]. The challenge is in developing an instrument that truly reflects all attributes of the process. Figure 18.2 provides different measurements that are currently used in research and practice. More information relating to acculturation measurements in health research and practice can be found at Thomson and Hoffman-Goetz [104].

Acculturation Related to Behavior Change

Acculturation is related to different health behaviors. For instance, Marin [62], suggested three levels associated with the acculturation process. Level one is the superficial level and includes influenced

Acculturation scales (n=87)

Authors	Scale	Scale type	Theory/ model	' n (modified) ^b	Numbe of items	er Domains s	Reliability Cronbach's alpha	Validity	Development/ testing
Cuellar et al. (1980)	Acculturation Rating Scale for Mexican Americans (ARSMA)	Unidimensional	No	29(23)	20	Language use, language preference, ethnic identity, ethnic classification, cultural heritage, ethnic behaviors ethnic interaction	Single scale (0.81–0.88) Generation status	5 week test-retest (0.8 non-clinical)	Mexican American
Cuellar et al. (1995)	Acculturation Rating Scale for Mexican Americans II (ARSMA II)	Multidimensional	Yes	10(3)	48	ethnic classification, cultural heritage, ethnic behaviors, ethnic interaction	Mexican American Orientation (0.86) anglo Orientation (0.88) Marginality (0.87)	1 Week test-retest (0.72–0.96), relation to ARSMA, generational status	Mexican American
Marin & Sabogal (1987)	Short Acculturation Scale for Hispanics	Unidimensional	No	24(4)	12	Language use, media, ethnic social relations	Single scale (0.92)	Generational status, length residence, age at arrival, perceived acculturation level	Mexican American Central American
Marin & Gamba (1996)	Bidimensional Acculturation Scale	Bidimensional	No	5(3)	24	Language, language proficiency, electronic media	Hispanic domain (0.87–0.97) Non-Hispanic domain (0.97)	Generational status, length of residence, age at arrival Proportion of life in US., ethnic identification	Mexican American Central American
Hazuda et al (1988)	. Hazuda Scale	Multidimensional	Yes	4(2)	31	Language, values, attitudes, ethnic interaction	Acculturation and structural assimilation (0.75–0.77)	Generational status, differentiate between Hispanic and Non-Hispanic white	Mexican American
Coronado et al. (2005)	Coronado Scale	Unidimensional	No	1(0)	4	Language, ethnic identification, nativity	Single scale ²	None reported	Mexican American
caetano (1987)	Caetano scale	Unidimensional	No	7(1)	17	Language, ethnic interaction, ethnic values and attitudes, media	Single scale (0.91)	Nativity, number of years lived in the U.S., age	Hispanic
Balcazar et al. (1995)	General Acculturation Index	Unidimensional	No	5(0)	5	Language, ethnic interaction, cultural pride	Single scale (0.82)	Education in Latin America education in U.S.	Mexican American
Anderson et al. (2004)	Anderson Scale	Unidimensional	No	1(0)	37	Language, media, nativity, ethnic values and beliefs, ethnic interaction		Language preference, television channel preference	Puerto Rican
Mikolajezyk et al. (2007)	Mikolajezyk Scale	Unidimensional	No	1(0)	5	Language, citizenship status, years in U.S.	Single scale None reported	None reported	Latino

^a Reliability testing completed using principal factor analysis.

^b In this case "modified" refers to scales that were changed from the original version.

Fig. 18.2 Acculturation measurements used in research and practice (Thomson and Hoffman-Goetz [104])

behaviors such as diet and media. Within this level, practitioners may see dietary changes in people who still possess most of their traditional attributes. The second level, the intermediate level of the process, incorporates behaviors such as language use and social networks. The third level, referred to as the significant level, includes the adoption of norms and values of the host culture.

In Hispanics, health-related factors have been associated with acculturation. For instance, higher acculturation, as measured by language use, has been positively associated with higher income, employment, exercise, and education and negatively related to tobacco use and dietary intake [77]. Extensive research using various acculturation proxies has been completed to examine different diet and health behaviors and outcomes and their association with acculturation. Following parts of this chapter will thus discuss acculturation as operationalized by various measurements and definitions, as well as its association with certain behaviors and outcomes.

Health in the Hispanic Population

Diabetes

Hispanics in the USA face multiple health disparities. For example, Hispanic Americans had about a 1.7 times higher prevalence of diabetes compared to non-Hispanic Whites in 2014 [14]. Diabetes in the Hispanic male population is currently 12.8%, with the highest prevalence observed in Mexican males [14]. Moreover, it is estimated that more than 2 million Hispanics have diabetes but are undiagnosed and that a 111% increase in prevalence will occur between 2011 and 2025 [51]. Research suggests factors such as acculturation status and country of origin may affect prevalence and disease-related morbidity. For example, Hispanics from South America and Cuba have a lower prevalence compared to those from Central America, Mexico, the Dominicas, and Puerto Rico [94]. Acculturation may account for differences in disease risk factors among Hispanics; however, their relationship to disease is unclear [2, 22, 111]. This will be discussed later in this chapter.

Overweight and Obesity

In comparison to all other ethnic groups, Hispanic children and men have the highest percent of overweight and obesity. According to Ogden and colleague [79], 77.1% of Hispanics, males and females, are overweight or obese; the highest among non-Hispanic Whites, Blacks, and Asians. Hispanic children and adolescents ages 2–19 had the highest rate of overweight or obesity, at 38.9%, compared to non-Hispanic groups [79]. Research suggests overweight and obesity is positively related to time spent in the USA and nativity and negatively associated with age of migration. A recent study indicated that Hispanics from lower income levels did not differ in terms of moderate obesity from Hispanics with higher income levels, but that they did have a higher rate of extreme obesity from those with higher income levels [53]. Another study showed income to be negatively associated with BMI in children; however, in Hispanic immigrant families income was positively associated with BMI in children. The lower-income families had a lower incidence of overweight and obese children, whereas higher-income immigrant families had a higher incidence. Culture may be a factor in the high incidence of overweight and obesity within this population. For instance, New et al. [77] found Hispanics of lower acculturation, independent of income, perceived overweight and obesity as more accepted. Studies suggest Hispanic parents lack understanding of short-term consequences or health issues associated with overweight or obese children and are less likely than parents of other ethnicities to perceive their children as overweight or obese. Further, these parents see "skinny" as undesirable and related to illness [87, 100]. As foreign-born Hispanics are more exposed to unhealthy behaviors in the USA, such as less physical activity and poorer diets, the risk of overweight and obesity increases and is possibly confounded by cultural acceptance.

Other Chronic Diseases

In 2012–2013, 8.0% of Hispanics in the USA had heart disease, compared to 11.0% of non-Hispanics. For stroke, the prevalence is similar to non-Hispanic Whites but lower than non-Hispanic Blacks [75], with a lower rate of hypertension compared to non-Hispanics Whites and Blacks. Although this population has a lower rate of hypertension and heart disease compared to other ethnic groups, Hispanics experience high rates of diabetic complications such as heart disease, stroke, kidney disease, blindness, amputations, and mortality [47, 110].

In terms of cancer, Hispanics have a lower risk compared to all other ethnic groups except Asians. About 3.6% of Hispanics over 18 were diagnosed with cancer between 2012 and 2013 [75]. The most prevalent cancers in the Hispanic population are of the prostate, breast, lung, and liver; the lowest prevalent cancers are skin, testicular, and ovarian [97].

Healthcare Access and Health Literacy

Acculturation is positively associated with health literacy in Hispanics, but Hispanics have the lowest health literacy among ethnic groups in the USA [40, 56]. Health literacy and numeracy literacy show the degree that an individual can access, process, and understand basic health and numerical information [52]. Specifically, Hispanics with limited English ability have lower health literacy and are less likely to communicate with health professionals, providing a barrier to healthcare access [40]. In terms of dietary literacy, increased use of food labels and nutrition knowledge has been associated with better dietary intake in this Hispanic population [38, 95].

Other Hispanic-related health disparities include discrimination, legal status, language use, and lack of healthcare access or insurance [35]. For example, research indicates Hispanics who lack English ability report a lack of healthcare access and preventative care and perceive their health as worse [28]. Health disparities may be associated with acculturation or legal status. For instance, New and colleagues [77] reported a positive association between acculturation and the following: health insurance, routine place of care, visits to healthcare provider, self-rated health status, and obesity-related comorbidities. A study by [74] reported that 89.5% of the undocumented Hispanic participants had health insurance and that only 36.5% had access to a regular healthcare provider.

Positive and negative associations between acculturation and health are documented. Research reports Hispanics who are more acculturated have more access to health care and do more structured physical activity but have unhealthier diets, smoke more, and have more negative pregnancy outcomes compared to their lower acculturated counterparts [57]. Further, obesity is positively associated to the time an immigrant has spent in the USA or being US-born [23].

Hispanic Paradox

The previous information delivers the stark statistics relating to factors affecting Hispanics' health and mortality. In the USA, this population has a low educational attainment and income, a higher unemployment rate when compared to the overall population, an almost two-fold higher prevalence of diabetes when compared to non-Hispanic Whites, and the highest rate of obesity when compared to all other ethnic groups. In further comparisons, Hispanics show a lower health literacy, a lower access to health care, and a lower perception of overall health.

Yet in spite of these factors, Hispanics overall have a lower mortality rate compared to non-Hispanic whites and a lower rate of death from cancer and cardiovascular disease [63, 71]. This phenomenon is referred to as the "Hispanic Paradox." Possible reasons for this paradox are an increase in social support within the Hispanic society, a decreased prevalence of smoking, and better overall dietary behaviors. Other reasons may be due what is called the "Salmon bias". The Salmon bias suggests immigrants migrate back to their country of origin and are not represented in the US mortality statistics. Another possible reason for the paradox is the health migrant effect. The healthy migrant effect states data may be missing or undocumented for many immigrants and again to total population and thus are not reflected within the overall health and mortality data [3], [12].

Teruya and Bazargan-Hejazi [103] examined recent literature on this paradox. They found the phenomenon very complex, with factors such as birthplace, documentation status, and nationality confounding the paradox still more. For example, there are differences in mortality rate when compared to non-Hispanic Whites dependent on nationality and gender. Older Mexican American women have a higher mortality age compared to non-Hispanic Whites, but contrarily Mexican American men had a higher death rate. Younger non-Mexican Hispanics and Cubans had a lower mortality rate compared to non-Hispanic whites. Clearly, the paradox is complex and not completely understood.

Dietary Behavior and the Hispanic Population

Dietary Attributes of the Hispanic Diet

The Hispanic diet is diverse due to the various regions and cultures of the people within the population. For example, the foods in Mexico and Central America are heavily influenced by native indigenous and Spanish ingredients. The traditional Latino diet, specifically in Mexico and Central America, consists of chili, lard, cactus, coffee, rice, poultry, fish, meat, beans, cocoa, tomatoes, corn, peas, and squash, and it is typically high in fiber, fruits, and vegetables [42, 55, 68], [60].

In South America, there may be areas influenced by other European states. In Argentina, for example, there is an Italian influence in the dishes, and Chile is influenced by German cuisine. Then, too, there are vast differences not only between countries, but also between the regions within countries: indigenous versus Spanish and urban versus rural. Table 18.2 provides information regarding traditional foods from each Latin American region.

The traditional Hispanic diet has been correlated with lower mortality from chronic diseases, including heart disease, breast cancer and other cancers, and a lower prevalence of obesity when compared to the diet of US-born Hispanics [50, 73]. However, diets throughout Latin America continue to change and be influenced by other cultures.

The Nutrition Transition

Over the past 20 years, countries throughout Latin America have been influenced by foods and ingredients from the USA. More processed and refined foods and beverages and more fast food chains are entering Latin American countries due to the increase in foreign-direct investment and transnational food companies. This change from a traditional diet to a more Western diet is referred to in the field as the *nutrition transition* [85]. The nutrition transition is defined as a shift in dietary patterns from

Region	Stable foods	Common ingredients	Common dishes	Traditional beliefs	Religion
Mexico	Maize Beans	Chili Peppers Cocoa Corn Tomatoes Plantains	Hearty soups and stews Casseroles Stuffed foods	Elements of Indian supernatural rituals European folk medicine Health is a gift from God Illness is almost always due to outside forces Health care is usually advised by mothers, grandmothers, or other older women Home remedies are tried before outside help is sought Curanderos are consulted for serious illnesses	Roman Catholic, Protestant
Central America	Rice Beans Corn Cassava	Cabbage Beets Carrots Flowers Tomatoes Avocado	Tortillas Enchiladas Soups and stews	Fruits and vegetables are important for maintaining good health Health is a balance between spiritual and social worlds Hot-cold theory	Roman Catholic, Protestant
Caribbean	Cassava Avocado Legumes	Lard Salt Onions Sweet peas Tomatoes Coconut Milk Sugar	Rice and peas Starchy vegetables with meat	Prayer important for good health Guardian angels Healing practices closely related to African beliefs Mild conditions treated by older women Serious conditions treated with supernatural powers Hygiene is important	Roman Catholic Protestant Voodoo Santaria
South America	Corn Chili Beef Beans Fish Potatoes	Tomatoes Pumpkins Bananas Plantains White root Peanuts Cashews Peppers	Grill meats Slow cooked organ meats Stuff foods	Bad health attributed to liver problems Hot and cold theory Health advice from mothers/ friends Faith associated with health Patron saints for medical help Herbal teas for health care Plants used for treatment	Roman Catholic, Protestant

Table 18.2 Traditional food and beliefs in Latin America

Kittler and Sucher [55]

traditional diets to a diet that mimics the western diet as a result of changes in environmental and social factors [85]. The nutrition transition has led to a shift in disease, such that chronic diseases have increased and are now the primary causes of mortality in many developing countries, such as in Mexico [85]. The diet of many Mexicans and Central Americans have seen a transition from a diet high in corn, tortillas, and beans to one high in fast food, processed foods, and high-calorie beverages over the past 10–40 years [7, 8, 86]. With this transition, there has been a change in disease rates for chronic diseases such as heart disease, cancer, and diabetes – on the rise throughout Latin America. Martínez [65] argues that, when assessing dietary behaviors in Hispanics residing in the USA, it is important to explore pre-migration dietary practices to establish whether Western food intake was already part of the daily diet and not only acculturation.

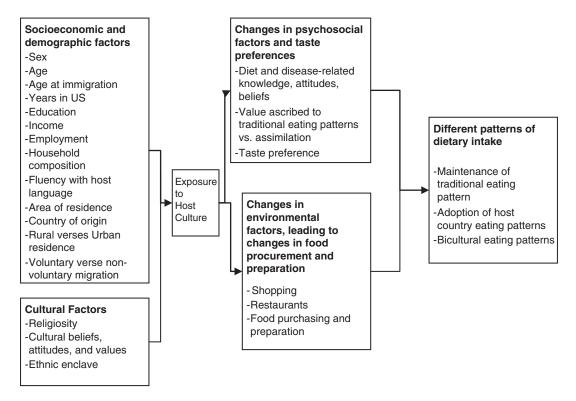


Fig. 18.3 A dietary acculturation model (Satia-Abouta [91])

Dietary Attributes of the Western Diet

The Western diet is defined as a diet high in refined and/or processed sugar, grains and oils, fatty meats, dairy, and sodium. Usually foods that are part of the western diet are relatively inexpensive and convenient. And the Western diet is associated with diet-related chronic diseases, such as obesity, diabetes, cancer, and heart disease [16]. The industrial age throughout the 1900s influenced the rapid development of the Western diet [16].

Dietary Acculturation

The process a person goes through when adopting the diet of his or her new culture is referred to as *dietary acculturation* [91]. Satia-Abouta [91] outlined factors that influence the degree to which a person of a different culture adopts to the dietary practices of his or her new culture in a dietary acculturation model (Fig. 18.3). In general, studies show acculturation is negatively associated with dietary behavior within the Hispanic population [27, 32, 72, 76]. Higher consumption of whole milk, fried foods, fruit, and vegetables and lower intakes of sugar, desserts, snacks, and eating outside the home is associated with lower acculturated Hispanics [32, 76, 78, 82–84].

Recently, Van Hook and colleagues [112] developed a dietary acculturation scale. The scale compares foods consumed to those common in a Western diet. The scale can be utilized to assess to what extent a person is consuming or has adapted a Western diet.

Dietary Intake in Hispanics

Fruit and Vegetable Intake

Recent studies suggest Hispanics have a higher intake of fruits and vegetables compared to non-Hispanic Whites and Blacks [48]. In general, Hispanics have a high intake of tomatoes and potatoes [96]. However, there are differences between Hispanics from different Latin American regions in terms of their fruit and vegetable intake. For example, comparisons between people from the Dominican Republic, Cuba, Puerto Rico, Mexican, Central America, and South America showed Dominican Americans and South Americans had the highest consumption of fruits between groups. Puerto Rican Americans had the lowest. Further, Cuban Americans had the highest vegetable consumption and Dominican Americans the lowest.

Other factors such as socioeconomic status (SES) and acculturation contribute to differences in fruit and vegetable intake. Acculturation has consistently been negatively related to consumption [20, 21, 29–31, 43, 76]. Proxies for acculturation in these studies included time in the USA, nativity, and language use. Acculturation confounds the relationship between SES and intake of fruits and vegetables. In lower SES households, lower acculturation is protective (higher fruit and vegetable intake), but in higher SES households, higher acculturation is protective [21, 67]. Hispanic children show a fruit-and-vegetable intake that is lower than recommended [21, 36]; however, their intake is higher compared to other ethnic groups' intake [26].

Dairy

Hispanics have a high rate of lactose intolerance, at an estimated 50 % of the population [93]. However, true intolerance is not known and is thought to be overestimated [6]. Intake of dairy products is below the recommended level of about three servings/day as set by the Dietary Guidelines for Americans. Data from the 2009–2010 National Health and Nutrition Examination Survey (NHANES) reported Hispanics averaging about 1.5 servings of dairy per day. Furthermore, research suggests Hispanics less acculturated or who have immigrated to the USA have higher intakes of whole milk and less of low-fat dairy compared to native born or more acculturated Hispanics [24, 32].

Dairy intake is associated with calcium intake, bone density, obesity, and chronic disease [6]. Overall, within the Hispanic population, calcium intake is just below the daily recommendation at 992 mg/day (NHANES, 2009–2010). Bone density is similar to that of non-Hispanic Whites and other ethnic groups. Research suggests that about 18.5% of Hispanic have osteopenia [114]. Other factors may contribute to calcium intake, such as vegetables, beans, and tortillas made with calcium. Although dairy intake has been associated with obesity and higher blood pressure, limited research has been specific to the Hispanic population.

Processed Foods, Meats, and Sugar

Research consistently associates consumption of processed foods and refined sugar with a more highly acculturated Hispanic population, a population that has lived in the USA for a longer time, nativity, and those with lower socioeconomic status and nutrition knowledge [32, 76, 78]. As previously pointed out, the availability of such foods is becoming more common throughout Latin America due to the nutrition transition. Therefore, understanding the pre-migration diet is important to assess dietary acculturation post-migration more correctly.

Meat consumption is limited in a traditional Hispanic diet; however, in the USA Hispanics have a meat consumption lower than non-Hispanic Blacks but similar to non-Hispanic Whites [33]. Unlike other

ethnic groups, the majority of their meat intake comes from beef, with poultry as the second-highest source. As reported, Hispanics view meat as more affordable in the USA, and its consumption is associated with social status [19]. Average consumption is approximately 6.1 ounces per day [96].

Research suggests a link between consumption of processed meat and diabetes as well as heart disease [5]; however, the connection between red meat and such diseases is uncertain [4, 58, 69]. Correlations between certain types of cancer and meat consumption are apparent, but certain cooking methods and processing may increase the risk still more [89, 117]

Sugar-Sweetened Beverages

Compared to all other ethnic groups, the Hispanic population shows the highest consumption of sugar-sweetened beverage (SSB) [54]. Further, when examined within the population, Hispanics who have resided in the USA longer and who speak Spanish have a higher consumption [80]. Unlike the research on other dietary factors, research shows low-acculturated Hispanics have a higher intake of sugar-sweetened beverages [113]. This possibly may be due to the nutrition transition, for intake of sugar-sweetened beverages is common throughout Latin America and higher compared to US consumption [98]. SSBs are associated with obesity and diabetes [61, 116]. Therefore, this is an important dietary factor to address with Hispanic clients and groups with high consumption.

Alcohol Consumption

Alcohol consumption is common in the Hispanic population and may be linked to the rate of liver disease and cancer in the population. However, use varies among subpopulations. For example, a recent study showed that Mexican Americans and Puerto Rican Americans were two to three times more likely to have an alcohol-use disorder compared to other Hispanic subpopulations and non-Hispanic Whites [88]. Another study showed that among Spanish-speaking Hispanics, 20% indicated drinking beyond control and were at risk for alcohol disorder [59]. Hispanic men reported drinking more than Hispanic women, and the effect of acculturation here is unclear [10, 64, 90, 118].

Eating Outside the Home

Consumption of fast food is now common in urban areas throughout Latin America [66] and therefore may be a normal part of the diet of an immigrant or native-born Hispanic. Furthermore, research does indicate acculturation is positively associated with eating outside the home and with Hispanic adolescents ([78, 106]; Ayala et al. 2005). Other factors that are associated with eating outside of the home are work schedule, female employment, and living structure [19]. Further, in a sample of Hispanic children from different subgroups, between 70.2 and 72.6% indicated consuming fast food at least once per week [45]. In fact, children's preference for fast food may drive family consumption [19].

Dietary Influencing Factors

Psychosocial Factors

Within the Hispanic population, multiple factors influence dietary intake such as the aforementioned income, education, and acculturation status. In addition, psychosocial factors such as dietary-related beliefs, attitudes, values, knowledge, perceptions, and preferences can influence intake. Within the traditional Hispanic culture, family meals and fresh and organic foods are valued, traditional foods are preferred, and foods can be seen as medicinal [15, 19, 81, 92, 115]. Moreover, social status is positively associated with foods such as meat and with eating at a restaurant [18, 19, 81, 105].

Lack of acculturation plays a role in psychosocial factors relating to dietary intake. For instance, a Hispanic person reflecting more of the traditional culture may express characteristics such as machismo, fatalism, and familismo within his or her dietary behavior. These cultural characteristics may appear within the beliefs, attitudes, and values about diet. For example, a more traditional Hispanic may expect the woman to procure and prepare the food (attitude, machismo), believe disease is not related to food intake but to natural and supernatural factors and powers (belief, fatalism), and value eating meals in a social setting with friends and family (value, familismo). And a more traditional Hispanic may have a higher preference and intake of traditional foods (preference, intake).

In the USA, studies suggest Hispanic males have the lowest diet-related knowledge among ethnic and gender groups [95]. Nutrition interventions appropriate for this male subpopulation are limited yet warranted due to not only to the high rates of obesity, diabetes, and associated negative dietary behaviors found in Hispanic males (i.e. high consumption of meat and SSBs), but also to the overall male influence within the household.

Environmental Factors

Environmental factors that influence diet include food cost, food availability, living structure, social interaction, time, and market style. Hispanics attempting to continue a traditional diet indicate it is hard to find fresh, traditional ingredients. More canned and frozen ingredients and foods are available, compromising the taste and flavor of the food. However, food availability may be dependent on the area of residence within the USA. There are higher Hispanic populations in the Southwestern USA, and therefore traditional food items may be easier to find in these areas. The cost of certain foods also affects intake. For example, the higher cost of fresh produce in the USA is seen as a barrier to Hispanics' consumption of fruits and vegetables. But, in a contrasting example, the cheaper cost of meats increases post-migration consumption of meats by foreignborn Hispanics [13, 19, 81, 92].

Other environmental factors affecting consumption patterns include living structure and time allotted for food procurement and preparation. Women working outside of the home influence dietary intake, particularly traditional food consumption [19, 46, 105, 115]. Many male immigrants may come to the USA without their spouses and/or without their parents and children, and they are not accustomed to procuring and preparing food [19]. Another influential change in the USA is a different work schedule. The altered living structure and work schedule may affect dietary intake and meal patterns. The traditional patterns of eating three meals a day and of limited snacking changes over to more snacking, more consumption of food outside of the home, more consumption of convenience foods, and more meals eaten alone [13, 19, 39, 105, 115].

Another important element to recognize is the role that children play in dietary behavior. Children's exposure to food associated with a Western diet, as well as their increased physiological desire for foods high in fat and sugar, influence their preference for nontraditional, processed foods [19, 34, 41, 49, 102]. Children are exposed to more foods associated with the Western diet at school, at daycare facilities, through other social occasions, and through food advertising [1, 19, 37].

Nutritional Considerations and Practical Implications

Assessing and Addressing Dietary Behaviors

After gaining a fuller understanding of the Hispanic population in the USA and their health and dietary attributes, healthcare professionals better appreciate the complexity addressing dietary issues and thus can better intervene with appropriate responses. Grasping the overall Hispanic culture and then the respective cultures of subgroups (i.e. country of origin, nativity, acculturation) leads to more careful assessment of idiosyncratic health behaviors and diet and more effective treatments.

The guide in Box 18.1 outlines steps for completing an initial dietary assessment for a Hispanic client or groups (Box 18.1). When working with the Hispanic population, a healthcare worker needs to ascertain current and past intake as well as diet-related influencing factors, recognizing the importance of understanding the individual differences within the group. Hispanics residing in the USA come from such a variety of backgrounds (i.e. country of origin, religion, race, educational level), and this variety in turn demands more thorough study on the part of those in the healthcare profession. But assessing each individual's diet (see Box 18.2) or a random number's diet from each subgroup within the larger group can inform culturally appropriate intervention, adaptation, and development.

Box 18.1 Indicators for Assessment

The following provides a guide to better understanding dietary behavior in a Hispanic individual.

- Social indicators: Assess nativity, socioeconomic status, primary language, and employment status. If an immigrant is foreign-born, inquire regarding the type of area – urban or rural – in which that person resided in the country of origin and time spent in the USA. Determine the client or groups interaction with the host culture and maintenance of traditional Hispanic norms and behaviors.
- Dietary behavior: Determine what natural protection has accrued from traditional dietary behaviors that have been retained, and then encourage the continuation of these behaviors. Such protective behaviors include consumption of fresh fruits and vegetables, consumption of beans, and consumption of homemade foods. Identify negative dietary behaviors such as consumption of high-fat dairy products or cooking with lard. Next, find out what dietary behaviors are occurring, such as increased consumption of refined sugars and processed foods, high intake of meat intake, high intake of fast food, increased consumption of sugar-sweetened beverages. Dietary evaluation methods include food screeners, 24-h recall or a dietary acculturation scale.
- Dietary-influencing factors: Identify different psychosocial and environmental factors that
 may be related to positive or negative dietary acculturation behaviors. In the psychosocial
 arena, discover the person's food beliefs, attitudes, preferences, values, and knowledge. For
 the environmental factors, determine the individual's living and social structure, food access
 and availability, time/schedule, and abilities in food procurement and preparation.

An assessment tool such as the Psychosocial and Environmental Dietary Questionnaire can help to determine relevant factors [18]. Once the practitioner has a complete view of

influencing factors and their relationships to one another, he or she can better understand the connection between diet and health outcomes and determine the best route to address dietary influencing factors that may be detrimental to one's health. Some routes may be to link the individual with a nutrition course or intervention offered in the community that addresses an identified influencing factor, refer him or her to a registered dietitian for nutrition counseling or contact a social worker to help provide and identify certain resources. Cuy Castellanos [132].

Box 18.2 Examining Dietary Factors and Behavior in Hispanic Individuals and Groups Cuy Castellanos [132]

Social indicators	Dietary behavior	Dietary-related environmental factors	Dietary-related psychosocial factors
Nativity, socioeconomic factors, language, time in the USA, employment, acculturation	Traditional vs. nontraditional	Food access and availability, cooking skills, social interaction, living structure, time/schedule	Beliefs, attitudes, preferences, values, and knowledge

Current Dietary Interventions

Multiple dietary interventions for various Hispanic subpopulations have been developed to address different dietary behaviors and health-related outcomes. Table 18.3 provides an overview of nutrition interventions developed for and completed within a Hispanic population. The majority of the interventions have been conducted with Hispanic women and/or children, have targeted a specific disease, and were completed in the southwestern USA or New England [70, 83]. Several interventions used trained lay *promotores de salud* (health promoters) or *abuelas* (grandmothers) to provide the nutrition intervention. Other factors incorporated in the interventions to ensure cultural appropriateness included the following: interviews and information that was provided in both Spanish and English, inclusion of family members, friends, and children for social support, and use of hands-on activities. Such culturally tailored interventions have been successful in positively affecting dietary behavior and health outcomes. Of course, if used with different Hispanic subgroups, such interventions should be adapted to ensure cultural appropriateness.

Martinez et al. [119] Foreign-born Latino men +19 yo, non-diabetic Kieffer et al. [120] Pregnant Latinas, +18 yo, less than 20 week gestations gestations Rocha-Goldberg Hispanic/Latino men and et al. [121] BP>120/88 or taking antihypertension meds	ien <i>N</i> =16 ic		Kesults	CONCIUSION
Pregnant Latinas, +18 less than 20 week gestations Hispanic/Latino men a women +18 yo wit BP > 120/88 or tak antihypertension m		3 Focus groups – Social Ecological Model used as framework;Groups discussed participants' perspectives on weight, diet, physical activity, perceived risk from, and susceptibility to obesity and related illness. Participants also discussed what role their spouse played in home nutrition	3 main themes: (1) description of general perceptions of weight, diet, and physical activity. (2) and (3) perceived barriers and perceived facilitators to healthy living	Barriers are inflexible work setting, influence of the socially constructed role of women in the household, and threat to traditional family structure at different levels of society
His	yo, <i>N</i> =139/arm	2-arm randomized control trial with parallel-group design, MOMs intervention- 11 week period with 2 home visits and 9 group meeting. Topics included general pregnancy info and healthy habits	Significant intervention effect of increased vegetable intake and a decrease in added sugars, total fat, and saturated fat	Supported that a community planned, community health worker-led lifestyle intervention can improve dietary habits of low-income pregnant Latinas
	h N=17 ng ieds	Culturally adapted behavioral lifestyle intervention aimed at decreasing hypertension based on social cognitive theory and techniques of behavioral self-management was conducted using the Stage of Change model and motivational enhancement approaches. Consisted of 6 weekly group sessions	Systolic BP was reduced and every other outcome except fat intake was improved. The largest impact on behavior- related variables was in change in weight and amount of exercise	It is feasible to implement lifestyle interventions for lowering BP in Hispanic/Latino adults. Participants felt that they were likely to change given the new knowledge. It is very important to use culturally appropriate interventions
Rothschild et al. T2D being treated with 1 [122] or more hypoglycemic agents and family Mexican origin	mic $N=121$	CHWs led behavioral self- management training during 36 home visits over 2 years. Self-management theory. Program emphasized knowledge and skills relating to diabetes self-management and goal setting	After 2 years, the intervention participants had significantly lower HBA1c levels, higher levels or self-reported physical activity, and lower body weight	CHWs can help individuals with diabetes achieve and maintain improvements in glycemic control

345

Table 18.3 (continued)	ed)				
Reference	Population	Sample	Intervention	Results	Conclusion
Taylor et al. [123]	Hispanic grandmothers and grandmother figures	<i>N</i> =36 abuelas	Stage of Change Model use abuelas as peer educators; 12 separate focus group discussions were conducted, which helped develop "The Healthy Kitchen". The abuelas were used to help educate younger mothers with young kids	Abuela peer educators improved their understanding of the taught nutrition topics and retained it	Training sessions were effective. Longer times are needed for nutrition information to be understood, put into practice, and succeed enough for significant improvements
Sorkin et al. [124]	Mexican American women with T2D with overweight daughters	N=89 dyads	 16 weeks with 4 group meetings, 8 home visits, and 4 booster telephone calls and was modeled after Diabetes Prevention Program's Lifestyle Change Program 	After 16 weeks, intervention participants had lower body weights, lower glycemic loads, and lower saturated fat intake. The intervention group had a greater health-related social support and persuasion and less undermining. Intervention daughters consumed more fruit and vegetables than control daughters	This program is more efficacious than usual care designed with educational materials for weight loss
Ockene et al. [125]	Latinos (Dominican & and Puerto Rican) with high risk of T2D	and $N = 321$	Social Cognitive Theory was used in 3 individual and 13 group sessions over a 12 month period with the goal of increasing intake of whole grains, physical activity, and nonstarchy vegetables. Also, to reduce sodium, fat, portion size, and refined carbohydrates	Inte	This lifestyle intervention was found to have a modest but significantly positive effect

Intervention may help reduce the decrease in PA once becoming pregnant	This was a successful intervention despite all of the participant barriers. Finding suggest that including family members in educational interventions may provide support to develop healthy family behaviors	Migrant farmer mothers want to learn and it is feasible to implement an intervention similar to the one conducted in this study	(continued)
No significant results	Significant improvements in systolic BP, diabetes self-efficacy, general diet, blood sugar, foot care, diabetes knowledge, physical components, and mental components	Intervention mothers had significantly higher self-efficacy scores but lower acculturation levels Intervention mothers' nutrition knowledge significantly improved. Mothers with less acculturation had greater improvements. There was a significant tend of intervention children moving into the normal BMI category. Mothers living alone were more likely to attend intervention classes	
Transtheoretical Model and Social Cognitive Theory used for 6-monthly in person behavioral counseling sessions and 5 telephone sessions. The goal was to encourage pregnant women to achieve the ACOG guidelines for PA and to decrease the intake of saturated fat and increase fiber intake	2 family sessions and 8 weekly group sessions which were conducted by a nurse practitioner. Sessions discussed healthy lifestyles and habits	Conducted during summer (~3 months) 2 group pre/post quasi experiment; Intervention group met 5 times (3 instructional and 2 data collection sessions) each lasting an hour. Instructional sessions discussed the food pyramid, portion sizes, physical activity, and goal achieving skills	
N=68	<i>N</i> =36 participants and 37 family members	N=59 mothers, 82 children (34 intervention mothers)	
Overweight Hispanic women 18–40 yo	T2D Hispanic individuals <i>N</i> =36 with at least 1 family part member; +18 yo fam fam	Migrant mothers and their children (2–12 yo) that live on agricultural work camps	
Hawkins et al. [126]	Hu et al. [127]	Kilanowski and Lin [128]	

Table 18.3 (continued)	(pa				
Reference	Population	Sample	Intervention	Results	Conclusion
Vincent et al. [129]	Spanish-speaking adults of Mexican descent	N=91	5 month intervention with 1 attention control group and 1 intervention group with an intensive phase consisting of 8 weekly 2-h sessions and maintenance phase of 3 monthly 1 h sessions. The goal was to promote healthy eating and physical activity. Participants were invited to bring a support family member to sessions	The intervention group improved weight, waist circumference, BMI, and diet self-efficacy	Suggests that 6 months or more of intervention is needed to obtain significant improvements
Pérez-Escamilla et al. [130]	Latino adults with T2D	N=211	18 month community based randomized control trial with a culturally appropriate intervention aiming to promote skills in nutrition, food access, physical activity, blood glucose monitoring, medication adherence, and appointment attendance. This was conducted during 17 home visits from a CHW	Intervention group had significantly lower HbA1c levels and fasting glucose. Fewer participants dropped out of the intervention group	There was a strong impact on glycemic control in this home-based model
Greenlee et al. [131]	Hispanic women who survived breast cancer	N=70	Randomized control trial investigating the effect of a 12 week dietary intervention. 9 sessions were conducted over 12 weeks which primarily focused on the maintenance of nutrition- related guidelines for cancer survivors	The intervention group had significantly greater increase in fruit/vegetable consumption and decrease in percent calories from total fat. Both of these were maintained at the 6 month mark. The more sessions attended led to greater improvements	The results indicated that the hands-on skills and knowledge building approach was effective in this population

Conclusions

The Hispanic population is the largest minority population in the USA, but it consists of people from various cultural, religious, geographical, acculturation, and socioeconomic backgrounds. Overall, this Hispanic population has lower SES, lower health and nutrition literacy, and lower access to health care compared to non-Hispanic Whites. The population has high rates of nutrition-related diseases, such as diabetes and obesity, but lower rates of heart disease and cancer. To meet the spectrum of dietary behaviors within the Hispanic population, healthcare professionals must become more familiar with the various dietary-related psychosocial and environmental factors. Understanding these factors in all their permutations is important for addressing poor dietary behaviors that lead to negative health outcomes.

References

- Abbatangelo-Gray J, Byrd-Bredbenner C, Austin SB. Health and nutrient content claims in food advertisements on Hispanic and mainstream prime-time television. J Nutr Educ Behav. 2008;40(6):348–54.
- Afable-Munsuz A, Gregorich SE, Markides KS, Pérez-Stable EJ. Diabetes risk in older Mexican Americans: effects of language acculturation, generation and socioeconomic status. J Cross Cult Gerontol. 2013;28(3): 359–73.
- 3. Arandia G, Nalty C, Sharkey JR, Dean WR. Diet and acculturation among Hispanic/Latino older adults in the United States: a review of literature and recommendations. J Nutr Gerontol Geriatr. 2012;31(1):16–37.
- 4. Ashaye A, Gaziano J, Djoussé L. Red meat consumption and risk of heart failure in male physicians. Nutr Metab Cardiovasc Dis. 2011;21(12):941–6.
- Aune D, De Stefani E, Ronco A, Boffetta P, Deneo-Pellegrini H, Acosta G, et al. Meat consumption and cancer risk: a case–control study in Uruguay. Asian Pac J Cancer Prev. 2009;10(3):429–36.
- Bailey RK, Fileti CP, Keith J, Tropez-Sims S, Price W, Allison-Ottey SD. Lactose intolerance and health disparities among African Americans and Hispanic Americans: an updated consensus statement. J Natl Med Assoc. 2013;105(2):112–27.
- Barquera S, Hernandez-Barrera L, Tolentino ML, Espinosa J, Ng SW, Rivera JA, et al. Energy intake from beverages is increasing among Mexican adolescents and adults. J Nutr. 2008;138(12):2454–61.
- Bermudez OI, Tucker KL. Trends in dietary patterns of Latin American populations. Cad Saude Publica. 2003;19:S87–99.
- 9. Berry JW. Immigration, acculturation, and adaptation. Appl Psychol An Int Rev. 1997;46(1):55-68.
- 10. Bryant AN, Kim G. The relation between acculturation and alcohol consumption patterns among older Asian and Hispanic immigrants. Aging Ment Health. 2013;17(2):147–56.
- 11. Cabassa LJ. Measuring acculturation: where we are and where we need to go. Hisp J Behav Sci. 2003;25(2):127–46.
- Carter-Pokras O, Zambrana RE, Yankelvich G, Estrada M, Castillo-Salgado C, Ortega AN. Health status of Mexican-origin persons: Do proxy measures of acculturation advance our understanding of health disparities? Journal of Immigrant and Minority Health. 2008;10(6):475–88.
- Cason K, Nieto-Montenegro S, Chavez-Martinez A. Food choices, food sufficiency practices, and nutrition education needs of Hispanic migrant workers in Pennsylvania. Top Clin Nutr. 2006;21(2):144–58.
- Centers for Disease Control and Prevention. National diabetes statistics report: estimates of diabetes and its burden in the United States. Atlanta: U.S. Department of Health and Human Services; 2014. Accessed 9 Nov 2015.
- Chavez-Martinez A, Cason KL, Mayo R, Nieto-Montenegro S, Williams JE, Haley-Zitin V. Assessment of predisposing, enabling, and reinforcing factors toward food choices and healthy eating among Hispanics in South Carolina. Top Clin Nutr. 2010;25(1):47–59.
- Cordain L, Eaton SB, Sebastian A, Mann N, Lindeberg S, Watkins BA, et al. Origins and evolution of the Western diet: health implications for the 21st century. Am J Clin Nutr. 2005;81(2):341–54.
- 17. Cuellar I, Arnold B, Maldonado R. Acculturation rating scale for Mexican Americans-II: a revision of the original ARSMA scale. Hisp J Behav Sci. 1995;17(3):275–304.
- 18. Cuy Castellanos D, Abrahamsen K. Using the PRECEDE-PROCEED model to assess dietary needs in the Hispanic population in northeastern Pennsylvania. Hisp Health Care Int. 2014;12(1):43–55.

- Cuy Castellanos D, Downey L, Graham-Kresge S, Yadrick K, Zoellner J, Connell CL. examining the diet of postmigrant Hispanic males using the precede-proceed model: predisposing, reinforcing, and enabling dietary factors. J Nutr Educ Behav. 2013;45(2):109–18.
- Dave JM, Evans AE, Pfeiffer KA, Watkins KW, Saunders RP. Correlates of availability and accessibility of fruits and vegetables in homes of low-income Hispanic families. Health Educ Res. 2010;25(1):97–108.
- Dave JM, Evans AE, Saunders RP, Watkins KW, Pfeiffer KA. Associations among food insecurity, acculturation, demographic factors, and fruit and vegetable intake at home in Hispanic children. J Am Diet Assoc. 2009;109(4):697–701.
- Daviglus ML, Pirzada A, Talavera GA. Cardiovascular disease risk factors in the Hispanic/Latino population: lessons from the Hispanic Community Health Study/Study of Latinos (HCHS/SOL). Prog Cardiovasc Dis. 2014;57(3):230–6.
- Delavari M, Sonderlund AL, Swinburn B, Mellor D, Renzaho A. Acculturation and obesity among migrant populations in high income countries a systematic review. BMC Public Health. 2013;13:458–69.
- de Hoog MLA, Kleinman KP, Gillman MW, Vrijkotte TGM, van Eijsden M, Taveras EM. Racial/ethnic and immigrant differences in early childhood diet quality. Public Health Nutr. 2014;17(6):1308–17. 10p.
- 25. DeNavas-Walt C, Proctor B. U.S. Census Bureau, Current Population Reports, P60-249, Income and Poverty in the United States: 2013. Washington, DC: U.S. Government Printing Office; 2014.
- Di Noia J, Byrd-Bredbenner C. Determinants of fruit and vegetable intake in low-income children and adolescents. Nutr Rev. 2014;72(9):575–90.
- Dixon LB, Sundquist J, Winkleby M. Differences in energy, nutrient, and food intakes in a US sample of Mexican-American women and men: findings from the Third National Health and Nutrition Examination Survey, 1988– 1994. Am J Epidemiol. 2000;152(6):548–57.
- DuBard CA, Gizlice Z. Language spoken and differences in health status, access to care, and receipt of preventive services among US Hispanics. Am J Public Health. 2008;98(11):2021–8.
- 29. Dubowitz T, Acevedo-Garcia D, Salkeld J, Lindsay AC, Subramanian SV, Peterson KE. Lifecourse, immigrant status and acculturation in food purchasing and preparation among low-income mothers. Public Health Nutr. 2007;10(4):396–404.
- Dubowitz T, Heron M, Bird CE, Lurie N, Finch BK, Basurto-Dávila R, et al. Neighborhood socioeconomic status and fruit and vegetable intake among whites, blacks, and Mexican Americans in the United States. Am J Clin Nutr. 2008;87(6):1883–91.
- Dubowitz T, Subramanian SV, Acevedo-Garcia D, Osypuk TL, Peterson KE. Individual and neighborhood differences in diet among low-income foreign and U.S.-born women. Womens Health Issues. 2008;18(3):181–90.
- Duffey KJ, Gordon-Larsen P, Ayala GX, Popkin BM. Birthplace is associated with more adverse dietary profiles for US-born than for foreign-born Latino adults. J Nutr. 2008;138(12):2428–35.
- Economic Research Service. Commodity consumption by population characteristics. NHANES data. United States Department of Agricultre. http://www.ers.usda.gov/data-products/commodity-consumption-by-populationcharacteristics.aspx. Access 6 Nov 2015.
- 34. Edmonds VM. The nutritional patterns of recently immigrated Honduran women. J Transcult Nurs. 2005;16(3):226-35.
- 35. Elder JP, Ayala GX, Parra Medina D, Talavera GA. Health promotion in the Latino community: communication issues and approaches. Annu Rev Public Health. 2009;30:227–51.
- 36. Erinosho TO, Berrigan D, Thompson FE, Moser RP, Nebeling LC, Yaroch AL. Dietary intakes of preschool-aged children in relation to caregivers' race/ethnicity, acculturation, and demographic characteristics: results from the 2007 California Health Interview Survey. Matern Child Health J. 2012;16(9):1844–53.
- Fleming-Milici F, Harris JL, Sarda V, Schwartz MB. Amount of Hispanic youth exposure to food and beverage advertising on Spanish-and English-language television. JAMA Pediatr. 2013;167(8):723–30.
- Fitzgerald N, Damio G, Segura-Pérez S, Pérez-Escamilla R. Nutrition knowledge, food label use, and food intake patterns among Latinas with and without type 2 diabetes. J Am Diet Assoc. 2008;108(6):960–7. doi: 10.1016/j. jada.2008.03.016.
- Gerchow L, Tagliaferro B, Squires A, Nicholson J, Savarimuthu SM, Gutnick D, et al. Latina food patterns in the United States. Nurs Res. 2014;63(3):182–93.
- 40. Ginde AA, Clark S, Goldstein JN, Camargo Jr CA. Demographic disparities in numeracy among emergency department patients: evidence from two multicenter studies. Patient Educ Couns. 2008;72(2):350–6.
- 41. Glassman ME, Figueroa M, Irigoyen M. Latino parents' perceptions of their ability to prevent obesity in their children. Fam Community Health. 2011;34(1):4–16. 13p.
- 42. Goody CM, Drago L. Cultural food practices. Diabetes Care and Education. Dietetic Practice Group: American Dietetic Association, Chicago, Il 2010.
- 43. Gregory-Mercado K, Staten LK, Ranger-Moore J, Thomson CA, Will JC, Ford ES, et al. Fruit and vegetable consumption of older Mexican-American women is associated with their acculturation level. Ethn Dis. 2006;16(1):89–95.
- 44. Gudykunst WB. Bridging differences: effective intergroup communication. Newbury Park: SagePublishers; 1998.

- 45. Guerrero AD, Ponce NA, Chung PJ. Obesogenic dietary practices of Latino and Asian subgroups of children in California: an analysis of the California Health Interview Survey, 2007–2012. Am J Public Health. 2015;105(8): e105–12.
- Hartweg DL, Isabelli-García C. Health perceptions of low-income, immigrant Spanish-speaking Latinas in the United States. Hisp Health Care Int. 2007;5(2):53–63.
- 47. Hazel-Fernandez L, Li Y, Nero D, Moretz C, Slabaugh L, Meah Y, et al. Racial/ethnic and gender differences in severity of diabetes-related complications, healthcare resource use, and costs in a medicare population. Popul Health Manag. 2014. http://online.liebertpub.com/doi/pdf/10.1089/pop.2014.0038. Accessed 16 Feb 2015.
- Hiza HAB, Casavale KO, Guenther PM, Davis CA. Diet quality of Americans differs by age, sex, race/ethnicity, income, and education level. J Acad Nutr Diet. 2013;113(2):297–306.
- Hoke MM, Timmerman GM, Robbins LK. Explanatory models of eating, weight, and health in rural Mexican American women. Hisp Health Care Int. 2006;4(3):143–51.
- Huh J, Prause JA, Dooley CD. The impact of nativity on chronic diseases, self-rated health and comorbidity status of Asian and Hispanic immigrants. J Immigr Minor Health. 2008;10(2):103–18.
- Institute for Alternative Futures. Diabetes 2025 forecasting model. 2010. http://www.altfutures.org/diabetes2025. Accessed 4 Nov 2015.
- Institute of Medicine. Health literacy: a prescription to end confusion. Washington, DC: National Academies Press; 2004.
- 53. Isasi CR, Ayala GX, Sotres-Alvarez D, Madanat H, Pedendo F, Loria C, et al. Is acculturation related to obesity in Hispanic/Latino adults? results from the Hispanic community health study/study of Latinos. J Obes. 2015. doi:10.1155/2015/186276.
- Kit BK, Fakhouri TH, Park S, Nielsen SJ, Ogden CL. Trends in sugar-sweetened beverage consumption among youth and adults in the United States: 1999–2010. Am J Clin Nutr. 2013;98(1):180–8.
- 55. Kittler PG, Sucher KP. Food and culture. 5th ed. Florence: Cengage; 1998. Learning.
- 56. Kutner M, Greenberg E, Jin Y, Paulsen C. The health literacy of America's adults: results from the 2003 National Assessment of adult literacy, Washington, DC, US 2006.
- 57. Lara M, Gamboa C, Kahramanian MI, Morales LS, Bautista DEH. Acculturation and Latino health in the United States: a review of the literature and its sociopolitical context. Annu Rev Public Health. 2005;26:367–97.
- Lippi G, Mattiuzzi C, Sanchis-Gomar F. Red meat consumption and ischemic heart disease. A systematic literature review. Meat Sci. 2015;108:32–6.
- Lotfipour S, Cisneros V, Anderson CL, Roumani S, Hoonpongsimanont W, Weiss J, et al. Assessment of alcohol use patterns among Spanish-speaking patients. Subst Abus. 2013;34(2):155–61.
- Loftas T, Ross J. Dimensions of need: An atlas of food and agriculture. Food and Agriculture Organization of the United Nations, Rome, Italy c1995.
- 61. Malik V, Popkin B, Bray G, Després J, Hu F. Sugar-sweetened beverages, obesity, type 2 diabetes mellitus, and cardiovascular disease risk. Circulation. 2010;121(11):1356–64.
- 62. Marin G. Issues in the measurement of acculturation among Hispanics. In: Geisinger KF, editor. Psychological testing of Hispanics. Washington, DC: American: Psychological Association; 1992. p. 235–51.
- Markides KS, Eschbach K. Aging, migration, and mortality: current status of research on the Hispanic paradox. J Gerontol B Psychol Sci Soc Sci. 2005;60(Special Issue 2):S68–75.
- 64. Markides KS, Snih SA, Walsh T, Cutchin M, Ju H, Goodwin JS. Problem drinking among Mexican-Americans: the influence of nativity and neighborhood context? Am J Health Promot. 2012;26(4):225–9.
- 65. Martínez AD. Reconsidering acculturation in dietary change research among Latino immigrants: challenging the preconditions of US migration. Ethn Health. 2013;18(2):115–35.
- 66. Martinez J, Powell J, Agne A, Scarinci I, Cherrington A. A Focus Group Study of Mexican Immigrant Men's Perceptions of Weight and Lifestyle. Public Health Nurs. 2012; 29(6):490–498.
- Mazur RE, Marquis GS, Jensen HH. Diet and food insufficiency among Hispanic youths: acculturation and socioeconomic factors in the third National Health and Nutrition Examination. Am J Clin Nutr. 2003;78(6):1120–7.
- 68. McArthur LH, Anguiano RP, Nocetti D. Maintenance and change in the diet of Hispanic immigrants in Eastern North Carolina. Fam Consum Sci Res J. 2001;29:309–35.
- Micha R, Wallace SK, Mozaffarian D. Red and processed meat consumption and risk of incident coronary heart disease, stroke, and diabetes mellitus: a systematic review and meta-analysis. Circulation. 2010;121(21):2271–83.
- Mier N, Ory MG, Medina AA. Anatomy of culturally sensitive interventions promoting nutrition and exercise in Hispanics: a critical examination of existing literature. Health Promot Pract. 2010;11(4):541–54.
- 71. Minino AM. Death in the United States, 2011. NCHS Data Brief 2013;115:1-8.
- Montez JK, Eschbach K. Country of birth and language are uniquely associated with intakes of fat, fiber, and fruits and vegetables among Mexican-American women in the United States. J Am Diet Assoc. 2008;108(3):473–80.
- Murtaugh MA, Sweeney C, Giuliano AR, Herrick JS, Hines L, Byers T, et al. Diet patterns and breast cancer risk in Hispanic and non-Hispanic white women: the Four-Corners Breast Cancer Study. Am J Clin Nutr. 2008;87(4):978–84.

- 74. Nandi A, Galea S, Lopez G, Nandi V, Strongarone S, Ompad DC. Access to and use of health services among undocumented Mexican immigrants in a US urban area. Am J Public Health. 2008;98(11):2011–20.
- National Center for Health Statistics. Health, United States, 2014: Center of Disease Control and Prevention with special feature on adults aged 55–64. Hyattsville; 2015.
- Neuhouser ML, Thompson B, Coronado GD, Solomon CC. Higher fat intake and lower fruit and vegetables intakes are associated with greater acculturation among Mexicans living in Washington State. J Am Diet Assoc. 2004;104(1):51–7.
- New C, Xiao L, Ma J. Acculturation and overweight-related attitudes and behavior among obese Hispanic adults in the United States. Obesity. 2013;21(11):2396–404.
- Norman S, Castro C, Albright C, King A. Comparing acculturation models in evaluating dietary habits among low-income Hispanic women. Ethn Dis. 2004;14(3):399–404.
- Ogden C, Carroll M, Flegal K. Prevalence of childhood and adult obesity in the United States, 2011–2012. JAMA. 2014;311(8):806–14.
- Park S, Blanck HM, Dooyema CA, Ayala GX. Association between sugar-sweetened beverage intake and proxies of acculturation among U.S. Hispanic and non-Hispanic white adults. Am J Health Promot. 2016;30:357–64.
- Park Y, Quinn J, Florez K, Jacobson J, Neckerman K, Rundle A. Hispanic immigrant women's perspective on healthy foods and the New York City retail food environment: a mixed-method study. Soc Sci Med. 2011;73(1):13–21.
- Pérez-Escamilla R. Acculturation, nutrition, and health disparities in Latinos. Am J Clin Nutr. 2011;93(5):1163S–7.
- Perez-Escamilla R, Putnik P. The role of acculturation in nutrition, lifestyle, and incidence of type 2 diabetes among Latinos. J Nutr. 2007;137(4):860–70.
- 84. Pérez-Escamilla R. Dietary quality among Latinos: is acculturation making us sick? J Am Diet Assoc. 2009;109(6):988–91.
- 85. Popkin BM. Nutritional patterns and transitions. Popul Dev Rev. 1993;19(1):138-57.
- 86. Ramirez Mayans JA, Garcia Campos M, Cervantes Bustamante R, Mata Rivera N, Zarate Mondragon F, Mason Cordero T, et al. Dietary changes in Mexico. An Pediatr (Barc). 2003;58(6):568–73.
- Reifsnider E, Flores-Vela AR, Beckman-Mendez D, Nguyen H, Keller C, Dowdall-Smith S. Perceptions of children's body sizes among mothers living on the Texas-Mexico border (La Frontera). Public Health Nurs. 2006;23:488–95.
- Ríos-Bedoya CF, Freile-Salinas D. Incidence of alcohol use disorders among Hispanic subgroups in the USA. Alcohol. 2014;49(5):549–56. 8p.
- Rohrmann S, Linseisen J, Overvad K, Lund Würtz AM, Roswall N, Tjonneland A, et al. Meat and fish consumption and the risk of renal cell carcinoma in the European prospective investigation into cancer and nutrition. Int J Cancer. 2015;136(5):E423–31.
- Rote SM, Brown RL. Gender differences in alcohol and drug use among Hispanic adults: the influence of family processes and acculturation. J Addict Dis. 2013;32(4):354–64.
- 91. Satia-Abouta J. Dietary acculturation: definition, process, assessment, and implications. Int J Hum Ecol. 2003;4(1):71-86.
- 92. Schmied EA, Parada H, Horton LA, Madanat H, Ayala GX. Family support is associated with behavioral strategies for healthy eating among Latinas. Health Educ Behav. 2014;41(1):34–41.
- 93. Scrimshaw N, Murray E. Prevalence of lactose maldigestion. Am J Clin Nutr. 1988;48:1086–98.
- Schneiderman N, Llabre M, Cowie CC, Barnhart J, Carnethon M, Gallo LC, et al. Prevalence of diabetes among Hispanics/Latinos from diverse backgrounds: the Hispanic Community Health Study/Study of Latinos (HCHS/ SOL). Diabetes Care. 2014;37(8):2233–9.
- 95. Sharma SV, Gernand AD, Day RS. Nutrition knowledge predicts eating behavior of all food groups and fruits and vegetables among adults in the Paso del Norte region: Qué Sabrosa Vida. J Nutr Educ Behav. 2008;40(6):361–8.
- 96. Siega-Riz A, Sotres-Alvarez D, Ayala GX, Ginsberg M, Himes JH, Liu K, et al. Food-group and nutrient-density intakes by Hispanic and Latino backgrounds in the Hispanic Community Health Study/Study of Latinos. Am J Clin Nutr. 2014;99(6):1487–98.
- Siegel R, Fedewa S, Miller K, Goding-Sauer A, Pinheiro P, Jemal A, et al. Cancer statistics for Hispanics/Latinos, 2015. CA Cancer J Clin. 2015;65(6):457–80.
- 98. Singh R, Coyne LS, Wallace LS. Brief screening items to identify spanish-speaking adults with limited health literacy and numeracy skills. BMC Health Serv Res. 2015;15:374. doi: 10.1186/s12913-015-1046-2.
- Smith J and Medalia Carla. U.S. Census Bureau, Current Population Reports, P60-253, Health Insurance Coverage in the United States: 2014. Washington, DC: U.S. Government Printing Office; 2015,
- 100. Sosa ET. Mexican American mothers' perceptions of childhood obesity: a theory-guided systematic literature review. Health Educ Behav. 2012;39:396–404.
- 101. Spector R. Health and illness in Hispanic American communities. In: Spector R, editor. Cultural diversity in health and illness, 4th edn. Appleton and Lange; Chestnut Hill, MA 1996. p. 279–97.

- Sussner KM, Lindsay AC, Greaney ML, Peterson KE. The influence of immigrant status and acculturation on the development of overweight in Latino families: a qualitative study. J Immigr Minor Health. 2008;10(6):497–505.
- Teruya SA, Bazargan-Hejazi S. The immigrant and Hispanic paradoxes: a systematic review of their predictions and effects. Hisp J Behav Sci. 2013;35(4):486–509.
- 104. Thomson MD, Hoffman-Goetz L. Defining and measuring acculturation: a systematic review of public health studies with Hispanic populations in the United States. Soc Sci Med. 2009;69(7):987.
- 105. Tovar A, Must A, Metayer N, Gute D, Pirie A, Hyatt R, et al. Immigrating to the US: what Brazilian, Latin American and Haitian women have to Say about changes to their lifestyle that may be associated with obesity. J Immigr Minor Health. 2013;15(2):357–64.
- 106. Unger JB, Reynolds K, Shakib S, Spruijt-Metz D, Sun P, Johnson CA. Acculturation, physical activity, and fast-food consumption among Asian-American and Hispanic adolescents. J Community Health. 2004;29(6):467– 81. 15p.
- 107. US Bureau of Labor Statistics. Employment status of the Hispanic or Latino population by sex and age. Division of Labor Force Statistics, Washington, DC; 2015. http://www.bls.gov/news.release/empsit.t01.htm. Accessed 9 Nov 2015. US Census Bureau. Total hispanics in the US. 2015. http://www.census.gov/population/hispanic. Accessed 6 Nov 2015.
- US Department of Commerce. Educational attainment in the US 2014. US Census Bureau. 2014. http://www.census.gov/hhes/socdemo/education/data/cps/2014/tables.html. Accessed 9 Nov 2015.
- US Office of Management and Budget. The 1997 revisions to the standards for the classification of federal data on race and ethnicity. Washington, DC; 1997. https://www.whitehouse.gov/omb/fedreg_1997standards. Accessed 9 Nov 2015.
- 110. Umpierrez G, Gonzalez A, Umpierrez D, Pimentel D. Diabetes mellitus in the Hispanic/Latino population: an increasing health care challenge in the United States (review). Am J Med Sci. 2007;334(4):274–82.
- Valencia WM, Oropesa-Gonzalez L, Hogue C, Florez HJ. Diabetes in older Hispanic/Latino Americans: understanding who is at greatest risk. Generations. 2014;38(4):33–40.
- 112. Van Hook J, Quiros S, Frisco ML. The Food Similarity Index: a new measure of dietary acculturation based on dietary recall data. J Immigr Minor Health. 2014;17(2):441–9.
- 113. Van Wieren A, Roberts M, Arellano N, Feller E, Diaz J. Acculturation and cardiovascular behaviors among Latinos in California by country/region of origin. J Immigr Minor Health. 2011;13(6):975–81.
- Vasquez E, Shaw BA, Gensburg L, Okorodudu D, Corsino L. Racial and ethnic differences in physical activity and bone density: National Health and Nutrition Examination Survey, 2007–2008. Prev Chronic Dis. 2013;10:E216.
- 115. Visocky S. We are what we eat: a cultural examination of immigrant health and nutrition in Middle Tennessee. Scientia et Humanitas. 2011;1:93–112.
- Wang M, Lemon S, Olendzki B, Rosal M. Beverage-consumption patterns and associations with metabolic risk factors among low-income Latinos with uncontrolled type 2 diabetes. J Acad Nutr Diet. 2013;113(12):1695–703.
- 117. Xue X, Gao Q, Qiao J, Zhang J, Xu C, Liu J. Red and processed meat consumption and the risk of lung cancer: a dose–response meta-analysis of 33 published studies. Int J Clin Exp Med. 2014;7(6):1542–53.
- 118. Zemore SE. Acculturation and alcohol among Latino adults in the United States: a comprehensive review. Alcohol Clin Exp Res. 2007;31(12):1968–90.
- 119. Martinez J, Powell J, Agne A, Scarinci I, Cherrington A. A focus group study of Mexican immigrant men's perceptions of weight and lifestyle. Public Health Nurs. 2012;29(6):490–8.
- Kieffer EC, Welmerink DB, Sinco BR, Welch KB, Rees Clayton EM, Schumann CY, Uhley VE. Dietary outcomes in a Spanish-language randomized controlled diabetes prevention trial with pregnant Latinas. Am J Public Health. 2014;104(3):526–33.
- 121. Rocha- Goldberg MDP, Corsino L, Batch B, Voils CI, Thorpe CT, Bosworth HB, Svetkey LP. Hypertension Improvement Project (HIP) Latino: results of a pilot study of lifestyle intervention for lowering blood pressure in Latino adults. Ethn Health. 2010;15(3):269–82.
- 122. Rothschild SK, Martin MA, Swider SM, Tumialán Lynas CM, Janssen I, Avery EF, Powell LH. Mexican American trial of community health workers: a randomized controlled trial of a community health worker intervention for Mexican Americans with type 2 diabetes mellitus. Am J Public Health. 2014;104(8):1540–8.
- 123. Taylor T, Serrano E, Anderson J, Kendall P. Knowledge, skills, and behavior improvements on peer educators and low-income Hispanic participants after a stage of change-based bilingual nutrition education program. J Community Health. 2000;25(3):241–62.
- 124. Sorkin DH, Mavandadi S, Rook KS, Biegler KA, Kilgore D, Dow E, Ngo-Metzger Q. Dyadic collaboration in shared health behavior change: the effects of a randomized trial to test a lifestyle intervention for high-risk Latinas. Health Psychol. 2014;33(6):566–75.
- 125. Ockene IS, Tellez TL, Rosal MC, Reed GW, Mordes J, Merriam PA, Olendzki BC, Handelman G, Nicolosi R, Ma Y. Outcomes of a Latino community-based intervention for the prevention of diabetes: the Lawrence Latino Diabetes Prevention Project. Am J Public Health. 2012;102(2):336–42.
- 126. Hawkins M, Hosker M, Marcus BH, Rosal MC, Braun B, Stanek 3rd EJ, Markenson G, Chasan-Taber L. A pregnancy lifestyle intervention to prevent gestational diabetes risk factors in overweight Hispanic women: a feasibility randomized controlled trial. Diabet Med. 2015;32(1):108–15.

- Hu J, Wallace DC, McCoy TP, Amirehsani KA. A family-based diabetes intervention for Hispanic adults and their family members. Diabetes Educ. 2014;40:48–59. 0145721713512682.
- 128. Kilanowski JF, Lin L. Effects of a Healthy Eating Intervention on Latina migrant farmworker mothers. Fam Community Health. 2013;36(4):350.
- 129. Vincent D, McEwen MM, Hepworth JT, Stump CS. The effects of a community-based, culturally tailored diabetes prevention intervention for high-risk adults of Mexican descent. Diabetes Educ. 2014;40(2):202–13.
- 130. Pérez-Escamilla R, Damio G, Chhabra J, Fernandez ML, Segura-Pérez S, Vega-López S, Kollannor-Samuel G, Calle M, Shebl FM, D'Agostino D. Impact of a community health workers–led structured program on blood glucose control among Latinos with type 2 diabetes: the DIALBEST trial. Diabetes Care. 2015;38(2):197–205.
- 131. Greenlee H, Gaffney AO, Aycinena AC, Koch P, Contento I, Karmally W, Richardson JM, Lim E, Tsai WY, Crew K, Maurer M, Kalinsky K, Hershman DL. Cocinar Para Su Salud!: Randomized Controlled Trial of a Culturally Based Dietary Intervention among Hispanic Breast Cancer Survivors. J Acad Nutr Diet. 2015;115(5):S42–56.
- Cuy Castellanos DK. Dietary acculturation in Latinos/Hispanics in the United States [seriel online]. Am J Lifestyle Med. 2014. Accessed www.djl.sagepub.com.

Chapter 19 Optimal Nutrition for the Older Adults

Alice H. Lichtenstein

Key Points

- Dietary guidance specially targeted to older adults is intended to promote the maintenance of optimal health and forestall the onset of chronic diseases.
- With advancing years, energy needs decline and nutrient needs either remain the same or increase, necessitating the need to choose nutrient-dense foods frosm all food categories.
- Changes in social situations with advancing years may result in the need to reassess eating patterns and when appropriate make modifications to ensure nutrient adequacy.

It is never too early in the life cycle to adapt eating patterns consistent with optimal health outcomes.

Keywords Nutrition • Older adults • Diet quality • Aging • Sight • Taste • Smell • Mobility • MyPlate • Chronic disease

Introduction

In 2015, approximately 14.9% of the U.S. population, or 47.8 million people, was over 65 years of age. This percent is expected to increase to approximately 21.6%, or 82.3 million, in the next 25 years. In 2015, approximately 2.0% of the U.S. population, or 6.3 million people, was over 85 years of age, sometimes referred to as the oldest-old. This percent is expected to almost double, to 3.8%, in the next 25 years. Similar trends are seen in other populations globally. Evidence suggests that within a population, older adults who score in the higher categories for diet quality and measures of physical activity have the lowest mortality rates, particularly, for cardiovascular disease, cancer, and type 2 diabetes mellitus [1–5]. As an increasing proportion of the world population enters the older age categories, more emphasis needs to be placed on optimal dietary guidance to enable these older adults to stay healthy and active. This emphasis should be provided within the context of the biological and psychological changes known to occur with advancing years.

A.H. Lichtenstein, D.Sc.

Cardiovascular Nutrition Laboratory, Jean Mayer USDA Human Nutrition Research Center on Aging, Tufts University, 711 Washington Street, Boston, MA 02465, USA e-mail: alice.lichtenstein@tufts.edu

Current Recommendations for Older Adults

The Recommended Dietary Allowances (RDA), established by the Food and Nutrition Board of the Institute of Medicine, has not been revamped since the late 1990s and early 2000s [6–12], with the exception of calcium and vitamin D [13]. Of note, prior to that time, no distinction was made for the nutrient requirements among adults above the age of 50 years. That category was expanded to include specific guidance for adults age 51–70 years and greater than 70 years [14].

RDAs and Adequate Intakes (AI) for most nutrients, including vitamin A, vitamin C, vitamin E, vitamin K, thiamin, riboflavin, niacin, folate, vitamin B_{12} , pantothenic acid, biotin, choline, chromium, copper, fluoride, iron, magnesium, manganese, molybdenum, phosphorus, selenium, and zinc, do not differ between adults above and below the age of 70 years. The nutrient recommendation for three nutrients, vitamin D, calcium, and vitamin B_6 , is higher for adults greater than 70 years (Table 19.1) [14]. The nutrient recommendations for two nutrients, chromium and sodium, is lower for the older age group. Emerging evidence suggests future revisions in the DRIs for individuals over the age of 70 years may be necessary [15, 16].

Although the RDAs or AIs for most nutrients do not increase for adults above the age of 70 years, it can become increasingly difficult to achieve the recommended intakes. In general, total energy requirements decreases with advancing years to compensate for the diminished basal metabolic rate associated with a higher proportion of fat mass relative to lean muscle mass and lower levels of physical activity [17–19].

Nutrient needs must be met within the context of lower energy intakes. This can be accomplished by judiciously choosing foods with a relatively high nutrient density (amount of nutrient/calorie). A version of the USDA MyPlate, MyPlate for Older Adults, has been developed specifically to provide guidance to achieve this goal (Fig. 19.1). Modifications made to the original USDA MyPlate specifically for older adults include the addition of food icons in the different sectors of the plate to provide illustrative examples of nutrient-dense choices such as deeply colored vegetables, fruits, and whole grains; shift of the dairy sector into the protein sector; fusion of the vegetable and fruit sectors; creation of a fluid sector on the top right of the plate to emphasize the importance of adequate hydration with advancing years; construction of a sector in the center of the plate containing "healthy fats" (liquid vegetable oil and soft margarine) to emphasize the importance across the diet of using these fats in place of animal fats in food preparation; and depiction of a broad range of different forms of foods particularly useful to older adults such as bags of frozen fruits, pre-cut and pre-washed vegetables, and canned low sodium foods.

Special Dietary Considerations for Older Adults

Approaches to maintaining optimal nutritional status in older adults should be considered in terms of both physiological and psychological factors. Consideration of both is critical to ensure optimal food intake and health outcomes.

Physiological Changes

The way the body handles nutrients can change with advancing age. These changes are generally attributed to alternations in the functioning of organ systems, which impact the utilization of some nutrients. Those systems most likely to be altered with advancing years include the stomach and small intestine, liver, heart, kidneys, skin, immune and oral cavity (Table 19.2).

	Females			Males			
	(years)						
Nutrient	31–50	51-70	>70	31-50	51-70	>70	
Vitamin A (µg/d) ^a	700	700	700	900	900	900	
Vitamin C (mg/d)	75	75	75	90	90	90	
Vitamin D (µg/d)	15	15	20	15	15	20	
Vitamin E (mg/d)	15	15	15	15	15	15	
Vitamin K (µg/d) ^b	90	90	90	120	120	120	
Thiamin (mg/d)	1.1	1.1	1.1	1.2	1.2	1.2	
Riboflavin (mg/d)	1.1	1.1	1.1	1.3	1.3	1.3	
Niacin (mg/d)	14	14	14	16	16	16	
Vitamin B_6 (mg/d)	1.3	1.5	1.5	1.3	1.7	1.7	
Folate (µg/d)	400	400	400	400	400	400	
Vitamin B_{12} (µg/d)	2.4	2.4	2.4	2.4	2.4	2.4	
Pantothenic Acid (mg/d)	5	5	5	5	5	5	
Biotin (µg/d)	30	30	30	30	30	30	
Choline (mg/d)	425	425	425	550	550	550	
Calcium (mg/d)	1000	1200	1200	1000	1000	1200	
Chromium (µg/d)	25	20	20	35	30	30	
Copper (µg/d)	900	900	900	900	900	900	
Fluoride (mg/d)	3	3	3	4	4	4	
Iodine (µg/d)	150	150	150	150	150	150	
Iron (mg/d)	18	8	8	8	8	8	
Magnesium (mg/d)	320	320	320	420	420	420	
Manganese (mg/d)	1.8	1.8	1.8	2.3	2.3	2.3	
Molybdenum (µg/d)	45	45	45	45	45	45	
Phosphorus (mg/d)	700	700	700	700	700	700	
Selenium (µg/d)	55	55	55	55	55	55	
Zinc (mg/d)	8	8	8	11	11	11	
Potassium (g/d)	4.7	4.7	4.7	4.7	4.7	4.7	
Sodium (g/d)	1.5	1.3	1.2	1.5	1.3	1.2	
Chloride (g/d)	2.3	2.0	1.8	2.3	2.0	1.8	

Table 19.1 Recommended dietary allowances for older adults age 31–50 years, 51–70 years and greater than 70 years

^aStandard font - Recommended dietary allowance values

^bItalic font - Adequate intake values

Of concern in older adults is that many individuals experience a decline in gastric hydrochloric acid secretion [20]. The resulting hypochlorydria causes a decline in the bioavailability of vitamin B_{12} [21]. Due to a decline in the skin's capacity to synthesize vitamin D from 7-dehydrocholesterol and less exposure to sunlight, older adults may be at a compromised status for vitamin D, and consequently, for calcium nurture. Changes in body composition (decreased lean muscle mass and increased fat mass) result in decreased basal metabolic rates, hence energy needs, and capacity for physical activity. Increased use of prescription and nonprescription medications, chronic drug therapy, and decreased capacity of the liver to metabolize drugs can compromise nutrient unitization. Health care providers need to be vigilant about identifying any changes that are of a sufficient magnitude to compromise nutrient status.

Taste, Smell

Retaining the desire to eat a variety of foods is fundamental to ensuring optimal nutritional status for older adults (Table 19.3). This is of particular concern because diminished taste and smell acuity associated with aging can lead to poor appetite. Changes that may occur include a decrease in taste

MyPlate for Older Adults



System	Potential changes			
Digestive system	↓ Hydrochloric acid secretion			
	↓ Digestive juice secretion (pancreas and small intestine)			
	↓ Absorptive capacity (malabsorption)			
	↓ Muscles tone large intestine (↓ gastrointestinal motility)			
	↓ Chronic blood loss due to ulcers and hemorrhoids			
Liver	↓ Hepatic and biliary function			
	↓ Rate detoxification			
Heart	↓ Cardiac output			
	↓ Strength and flexibility of blood vessels			
Kidneys	↓ Blood flow			
	↓ Glomerular filtration			
Skin	↓ Synthesis vitamin D			
Body composition	\downarrow Lean muscle mass and \uparrow fat mass			
	↓ Physical activity			
Immune system	↓ T cell-mediated function			
	↑ Susceptibility to infection and malignancy			
Oral cavity	↑ Peritoneal disease			
	↑ Ill-fitting dentures			
	↓ Salivary gland secretions			
	↑ Altered bite pattern due to tooth loss			
Phamacokinetics	↑ Prescription and nonprescription drug use			
	↑ Chronic drug therapy			
	↓ Capacity to metabolize drugs			

sensitivity, primarily to salt and sweet. This, in turn, may result in greater sensitivity to acid and bitter [22]. Another change that may occur with advancing years is diminished sense of smell. Older adults with poor odor perception have lower nutrient intakes than those with more acute odor perception [23–25].

Vision, Dexterity, and Mobility

Diminished vision, dexterity, and mobility can make food accusation and preparation challenging (Table 19.3). Difficulty opening jars, cans, or packaged foods due to arthritis or diminished strength can lead to decreased variety and the ability to consume preferred foods. Small accommodations to an individual's environment such as ergonomically designed kitchen aides (e.g., can openers and scissors), kitchen reorganization (e.g., eliminating clutter and shifting frequently used items to most accessible places), and shifts to the use of partially prepared foods can minimize a decline in diet quality. For example, re-sealable bags of frozen vegetables and fruits are particularly good choices because they allow for easy apportioning of single or double servings, minimize pre-preparation which can be difficult or even painful, eliminate waste due to spoilage, reduce the need for frequent trips to the market, and provide variety during inclement weather. Likewise, purchasing boneless chicken breasts can decrease preparation and cooking times, and is adaptable to preparation of individual small portions. Older adults may not automatically take advantage of newer forms of common food items (e.g., pre-washed and cut salad, shredded cheese) and require some regular guidance in this area.

Social Factors

In addition to dealing with declines in physical capacity associated with the aging process, there are also changes in the social environment that can have an impact on nutritional status (Table 19.4). With advancing years, the loss of a spouse or other family members with whom an individual shared and

Factor	Change				
Senses	↓ Acuity vision and hearing				
	↓ Taste (loss taste buds, mainly salt and sweet)				
	↓ Smell				
Mobility	↓ Physical activity				
	↓ Respiratory capacity				
	↓ Lean muscle mass (strength, physical disability)				
	↑ Physical isolation				
Dexterity	↑ Sarcopenia				
	↑ Arthritic involvement in finger and hand joints				
	↑ Tremor				
	↓ Manual dexterity				
	↓ Gait				
	↓ Balance				
Energy needs	↓ Energy requirements				
	↑ Geriatric cachexia				
	↓ Volume capacity				

 Table 19.3
 Activity of daily life factors potentially contributing to compromised food intake in older adults

Factor	Change			
Companionship	↑ Loss of spouse			
	↑ Social isolation			
	↑ Loss of contemporaries			
	↓ Social interaction secondary to ↓ mobility			
	\downarrow Social interaction secondary to \downarrow change in domicile			
Mental state	↑ Depression			
	↑ Mental deterioration (dementia)			
	↑ Alcohol abuse			
Economic	↑ Fixed income			
	↓ Choice, variety, and availability of foods			
Nutrition knowledge	↑ Susceptibility to food fads			
	↑ Susceptibility to dietary supplement claims			
Housing	↑ Change in status (loss of home)			
	↑ Change in availability of preferred foods			

 Table 19.4
 Potential psycho-social factors contributing to compromised nutrient status in older adults

prepared meals is common. This can lead to social isolation, especially during mealtime, and diminished desire to prepare balanced and varied meals. Due to deterioration in mental or economic status, older adults are frequently faced with having to adapt to a new living environment. This can result in dramatic changes in meal times, food preparation, and foods available. The onset of chronic disease can further limit food choices and make older adults susceptible to the lures of food fads or dietary supplements that promise a fountain of youth. At worst, these claims risk draining scarce resources available for food purchases and overconsumption of individuals nutrients which can interfere with prescription drug actions or the utilization of other essential nutrients. Depression can accompany the aging process, particularly, in individuals without adequate support to make the necessary adaptations that come with advancing years. Older adults may be at increased risk of alcohol abuse. All of these factors may contribute to poor food consumption patterns [26].

Nutrition Knowledge/Susceptibility to Food Fads and Nutrient Supplement Claims

Nutrient supplement use is more common in older than younger adults [27–31]. The primary reasons cited by older adults for taking nutrient supplements are to improve health and delay the onset of chronic disease [32–34]. This issue is of particular concern because, in general, older adults who choose to use nutrient supplements are least likely to have biomarkers of nutrient inadequacy or diets rated as a poor [31, 35]. In light of the widespread availability of fortified foods, this group may be particularly vulnerable to excess nutrient intakes and drug-nutrient interactions [28, 30, 36–38]. The latter issue is of particular concern given the limited amount of information available on the topic [39]. General characteristics of individuals using supplements, in addition to being older [27–31], include being female [29–31], non-Hispanic white [30, 31], college educated or beyond [27, 29–31], and affluent [31]. In addition, nutrient supplement users are more likely to have body mass indices within the normal range [27, 30], engage in regular physical activity [27, 30], have optimal chronic disease biomarkers [31], have low rates of smoking [27], achieve nutrient requirement recommendations, and hold strong attitudes about the importance of a good diet [33, 35, 38]. Recent work on the potential adverse effects of excess vitamin A intake and risk of bone fracture in older women highlight the importance of this issue [40].

Chronic Diseases of Particular Concern in Older Adults Related to Lifestyle Behaviors

Nutrient-related chronic diseases, particularly prevalent in middle and later years, include disorders of dentition and associated senses, cardiovascular disease, osteoporosis, Type 2 diabetes, hypertension, immune, and cancer. In some cases, the goals of nutrient recommendations for older adults are aimed at delaying the onset of chronic disease while in others it is aimed at treatment or accommodating the disorder.

Dentition and Associated Senses

Salivary secretions decrease with increasing age. Changes in bite pattern from partial or complete tooth extraction/loss are common. Poorly fitted dentures can make eating painful and distasteful. The prevalence of root canals is higher in older than younger adults [41, 42]. Increased incidence of tooth disease in older adults has been related, in part, to high levels of sugar consumption [43]. Any one or combination of these factors can restrict the type and variety of foods consumed. For example, chewing and swallowing fibrous foods may be difficult due to poor dentition, resulting in a shift towards highly processed foods or juices that are low in fiber [44]. It is critical when evaluating dietary intakes of older adults to consider possible concerns regarding food textures and preparation methods and to assess dentition.

Cardiovascular Disease

The rate of cardiovascular disease increases with age, especially after menopause in females [45]. Higher saturated fat coupled with lower polyunsaturated fat intakes has consistently been associated with higher rates of cardiovascular disease [46, 47]. The American Heart Association (AHA) [48], the AHA/American College of Cardiology [49], and the 2015 Dietary Guidelines Advisory Committee [50] recommend dietary patterns that are higher in vegetables, fruits, whole grains, low-or nonfat dairy, seafood, legumes, and nuts; moderate in alcohol; lower in red and processed meat; and low in sugar sweetened foods and drinks and refined grains. Individuals should be encouraged to adapt this general dietary pattern to personal and cultural preferences to enhance enjoyment of their food. No specific recommendations for dietary change are made for adults as they age. The response to these recommendations in terms of plasma lipids appears consistent for both genders and age groups [51, 52].

Osteoporosis

Age-related or type II osteoporosis (bone loss) is positively associated with the aging process. It has been estimated that osteoporotic fractures affect 50% of females and 30% of males over the age of 50 years [53]. Age-associated bone loss is attributed to diminished estrogen production, decreased calcium absorption from the gastrointestinal tract, decreased calcium resorption by the kidney, decreased rates of physical activity, compromised vitamin D status, and decreased calcitriol production second-ary to hyperparathyroidism [53, 54]. In older adults, calcium balance is favorably affected by attaining

adequate vitamin D nutriture and negatively affected by high sodium, protein, alcohol, and caffeine intakes [54]. Supplemental calcium and vitamin D in postmenopausal women living in northern latitudes (42°N) may minimize bone loss [55]. Because serum osteocalcin, calcidiol, and vitamin D fluctuate seasonally due to sun exposure, vitamin D intake is particularly important during the periods of winter and spring in this group. These data strongly support routine screening of older adults for vitamin D status.

Glucose Intolerance/Type 2 Diabetes

The incidences of glucose intolerance and Type 2 diabetes mellitus increase with age [56, 57]. The increased incidence has been strongly associated with weight gain in later years. Lifestyle interventions have been shown to be efficacious in preventing or delaying the onset of Type 2 diabetes mellitus in some but not all studies [58–61]. These include regular daily physical activity, weight loss, and dietary modification consistent with that advocated to the prevention and treatment of cardiovascular disease.

Hypertension

The incidence of hypertension, particularly increases in systolic blood pressure, occur with age [45]. This increase is associated with changes in the vasculature and kidneys, and is exacerbated by weight gain. A number of clinical trials have demonstrated clear benefits of dietary modification to treat hypertension in older adults. The Dietary Approaches to Stop Hypertension (DASH) type dietary pattern, rich in vegetables, fruits, and fat-free and low-fat dairy products, decreases blood pressure in a wide range of individuals [62]. Further coupling this dietary pattern with sodium restriction can lead to an additional decrease in blood pressure [63].

Immune Function

The most commonly associated age-related change in the immune response is cell-mediated function [64, 65]. Vitamin E supplementation may be beneficial in decreasing the incidence of respiratory infections in older adults [66].

Cancer

The incidence of cancer shows tremendous variability on the basis of worldwide distribution, type, and site in the body. The incidence of all types of cancer increases with age. Support for a diet/cancer incidence link comes from data suggesting associations between markedly divergent food consumption patterns and incidence rates of cancer among populations worldwide [67]. Some data has suggested a positive association with cancer incidence and alcohol intake (laryngeal) and total fat intake

(breast, colon, prostate); and negative association with cancer incidence and calcium and vitamin D intake (stomach, colon, breast), fiber intake (breast), antioxidant vitamin and/or orange and dark green vegetable intake (rich in vitamin A and beta-carotene, vitamin C, vitamin E), and trace elements (wide range of sites) [67–71]. Results from randomized controlled trials are limited [72]. At this time, the general dietary guidance to reduce cancer risk is consistent with the dietary guidance to prevent the onset of chronic diseases of concern in the twenty-first century.

Dietary Guidance for Older Adults

Current Intake Patterns

Older adults (>70 years) have a Healthy Eating Index of approximately 65, higher than that of their younger counterparts (Fig. 19.2). Nevertheless, there is room for improvement. Although older adults consume approximately 90% of the recommended servings for total fruit, whole fruit, total protein foods and seafood/plant proteins, and 80% of the recommended servings for total vegetables, they consume under 60% of the recommended servings for greens and beans and dairy and under 50% of whole grains.

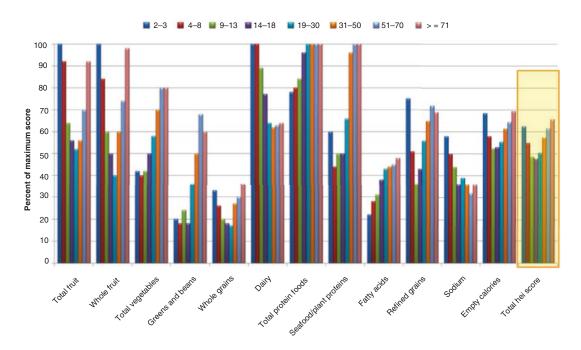


Fig. 19.2 Average HEI-2010 scores for Americans by age group (Source: What we eat in America NHANES 2007–2010)

Conclusions

The aim of dietary guidance specifically targeted for older adults is to maintain optimal health and forestall the onset of chronic disorders. The actual dietary recommendations, for the most part, are consistent throughout the adult lifecycle. Diet quality can have an important effect on the ability to perform activities of daily life and survival rates. Due to lower levels of physical activity, decreased metabolic rates secondary to increased proportions of fat to lean muscle mass, energy requirements decline with advancing years yet nutrient requirements remain unchanged, or in some cases increase. This situation requires a greater emphasis on choosing nutrient-dense foods within each food category. With advancing years special attention needs to be given to adapting living environments to retain the ability to acquire and prepare food. Changes in social situations that could impact on food intake should be monitored on a regular basis. Evidence suggests that diet and lifestyle interventions can forestall the onset of cardiovascular disease, osteoporosis, diabetes, hypertension, immune function, and possibly cancer. There are no data to suggest a person is too old to benefit from improvements in diet quality. The definitions for old age and expectations for the period of time individuals can remain active, productive, and live independently are expanding. Efforts towards improving diet quality and levels of physical activity as individuals get older should keep up with this trend.

References

- Anderson DR, Grossmeier J, Seaverson EL, Snyder DJ. The role of financial incentives in driving employee engagement in health management. ACSM Health Fit J. 2008;12:18–22.
- Schwingshackl L, Hoffmann G. Diet quality as assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension score, and health outcomes: a systematic review and metaanalysis of cohort studies. J Acad Nutr Diet. 2015;115:780–800.
- Reedy J, Krebs-Smith SM, Miller PE, Liese AD, Kahle LL, Park Y, et al. Higher diet quality is associated with decreased risk of all-cause, cardiovascular disease, and cancer mortality among older adults. J Nutr. 2014;144:881–9.
- 4. Blain H, Carriere I, Sourial N, Berard C, Favie F, Colvez A, et al. Balance and walking speed predict subsequent 8-year mortality independently of current and intermediate events in well-functioning women aged 75 years and older. J Nutr Health Aging. 2010;14:595–600.
- 5. Studenski S, Perera S, Patel K, Rosano C, Faulkner K, Inzitari M, et al. Gait speed and survival in older adults. JAMA. 2010;305(1):50–8.
- IOM. Dietary reference intakes. Calcium, phosphorus, magnesium, vitamin D and fluoride. Washington, DC: National Academy of Sciences; 1997.
- 7. IOM. Dietary reference intakes. Thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin and choline. Washington, DC: National Academy of Sciences; 1998.
- 8. IOM. Dietary reference intakes. Vitamin C, vitamin E, selenium and carotenoids. Washington, DC: National Academy of Sciences; 2000.
- 9. IOM. Dietary reference intakes. Vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium and zinc. Washington, DC: National Academy of Sciences; 2001.
- 10. IOM. Dietary supplements: a framework for evaluating safety. Committee on the framework for evaluating the safety of dietary supplements. Washington, DC: National Academy of Sciences; 2004.
- 11. IOM. Dietary reference intakes, water, potassium, sodium, chloride and sulfate. Washington, DC: National Academy of Sciences; 2005.
- 12. IOM. Dietary reference intakes. Energy, carbohdyrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. Washington, DC: National Academy of Sciences; 2005. p. 482.
- IOM. Dietary Reference Intakes for Calcium and Vitamin D. 2010. http://www.iom.edu/Reports/2010/ Dietary-Reference-Intakes-for-Calcium-and-Vitamin-Daspx.
- 14. IOM. Dietary reference intakes. The essential guide to nutrient requirements. Washington, DC: National Academy of Sciences; 2006.
- 15. Wolfe RR, Miller SL, Miller KB. Optimal protein intake in the elderly. Clin Nutr. 2008;27(5):675-84.
- 16. Kim IY, Schutzler S, Schrader A, Spencer H, Kortebein P, Deutz NE, et al. Quantity of dietary protein intake, but not pattern of intake, affects net protein balance primarily through differences in protein synthesis in older adults. Am J Physiol Endocrinol Metab. 2015;308(1):E21–8.

- 17. Williamson DF. Descriptive epidemiology of body weight and weight change in U.S. adults. Ann Intern Med. 1993;119(7 Pt 2):646–9.
- 18. Hoffman N. Diet in the elderly. Needs and risks. Med Clin North Am. 1993;77(4):745-56.
- Verreijen AM, Verlaan S, Engberink MF, Swinkels S, de Vogel-van den Bosch J, Weijs PJ. A high whey protein-, leucine-, and vitamin D-enriched supplement preserves muscle mass during intentional weight loss in obese older adults: a double-blind randomized controlled trial. Am J Clin Nutr. 2015;101(2):279–86.
- 20. Byrd D, Russell RM. Malabsorption in an elderly patient. Gastroenterologist. 1993;1:287-90.
- 21. Chu S, Schubert ML. Gastric secretion. Curr Opin Gastroenterol. 2012;28(6):587-93.
- 22. Lipson LG, Bray GA. In: Chen LH, editor. Nutritional aspectes of aging, vol. I. Boca Raton: CRC Press, Inc; 1986.
- Griep MI, Collys K, Mets TF, Slop D, Laska M, Massart DL. Sensory detection of food odour in relation to dental status, gender and age. Gerodontology. 1996;13(1):56–62.
- Griep MI, Verleye G, Franck AH, Collys K, Mets TF, Massart DL. Variation in nutrient intake with dental status, age and odour perception. Eur J Clin Nutr. 1996;50(12):816–25.
- Griep MI, Mets TF, Collys K, Ponjaert-Kristoffersen I, Massart DL. Risk of malnutrition in retirement homes elderly persons measured by the "mini-nutritional assessment". J Gerontol A Biol Sci Med Sci. 2000;55(2):M57–63.
- James WP, Nelson M, Ralph A, Leather S. Socioeconomic determinants of health. The contribution of nutrition to inequalities in health. BMJ. 1997;314(7093):1545–9.
- Foote JA, Murphy SP, Wilkens LR, Hankin JH, Henderson BE, Kolonel LN. Factors associated with dietary supplement use among healthy adults of five ethnicities: the Multiethnic Cohort Study. Am J Epidemiol. 2003;157(10):888–97.
- Yoon SL, Schaffer SD. Herbal, prescribed, and over-the-counter drug use in older women: prevalence of drug interactions. Geriatr Nurs. 2006;27(2):118–29.
- Gardiner P, Graham R, Legedza ATR, Ahn AC, Eisenberg DM, Phillips RS. Factors associated with herbal therapy use by adults in the United States. Altern Ther Health Med. 2007;13(2):22–9.
- 30. Rock CL. Multivitamin-multimineral supplements: who uses them? Am J Clin Nutr. 2007;85(1):277S-9.
- 31. Block G, Jensen CD, Norkus EP, Dalvi TB, Wong LG, McManus JF, et al. Usage patterns, health, and nutritional status of long-term multiple dietary supplement users: a cross-sectional study. Nutr J. 2007;6:30.
- 32. Buhr G, Bales CW. Nutritional supplements for older adults: review and recommendations-part I. J Nutr Elder. 2009;28(1):5–29.
- Buhr G, Bales CW. Nutritional supplements for older adults: review and recommendations part II. J Nutr Elder. 2010;29(1):42–71.
- Bailey RL, Gahche JJ, Miller PE, Thomas PR, Dwyer JT. Why US adults use dietary supplements. JAMA Intern Med. 2013;173:355–61.
- Sebastian RS, Cleveland LE, Goldman JD, Moshfegh AJ. Older adults who use vitamin/mineral supplements differ from nonusers in nutrient intake adequacy and dietary attitudes. J Am Diet Assoc. 2007;107(8):1322–32.
- Radimer K, Bindewald B, Hughes J, Ervin B, Swanson C, Picciano MF. Dietary supplement use by US adults: data from the National Health and Nutrition Examination Survey, 1999–2000. Am J Epidemiol. 2004;160(4):339–49.
- Kishiyama SS, Leahy MJ, Zitzelberger TA, Guariglia R, Zajdel DP, Calvert JF, et al. Patterns of dietary supplement usage in demographically diverse older people. Altern Ther Health Med. 2006;11:48–53.
- Murphy SP, White KK, Park SY, Sharma S. Multivitamin-multimineral supplements' effect on total nutrient intake. Am J Clin Nutr. 2007;85(1):280S–4.
- Yetley EA. Multivitamin and multimineral dietary supplements: definitions, characterization, bioavailability, and drug interactions. Am J Clin Nutr. 2007;85(1):2698–76.
- 40. Wu AM, Huang CQ, Lin ZK, Tian NF, Ni WF, Wang XY, et al. The relationship between vitamin A and risk of fracture: meta-analysis of prospective studies. J Bone Miner Res. 2014;29:2032–9.
- Papas AS, Joshi A, Giunta JL, Palmer CA. Relationships among education, dentate status, and diet in adults. Spec Care Dentist. 1998;18(1):26–32.
- Papas AS, Palmer CA, Rounds MC, Russell RM. The effects of denture status on nutrition. Spec Care Dentist. 1998;18(1):17–25.
- Papas AS, Joshi A, Palmer CA, Giunta JL, Dwyer JT. Relationship of diet to root caries. Am J Clin Nutr. 1995;61(2):423S–9.
- NIDDK. NIH Publication No. 07–2754 July 2007 National Digestive Diseases Information Clearinghouse. http:// digestive.niddk.nih.gov/ddiseases/pubs/constipation/2007.
- 45. AHA website. www.americanheart.org/presenter.jhtml?identifier=1200011.
- 46. Jakobsen MU, O'Reilly EJ, Heitmann BL, Pereira MA, Balter K, Fraser GE, et al. Major types of dietary fat and risk of coronary heart disease: a pooled analysis of 11 cohort studies. Am J Clin Nutr. 2009;89(5):1425–32.
- 47. Li Y, Hruby A, Bernstein AM, Ley SH, Wang DD, Chiuve SE, et al. Saturated fats compared with unsaturated fats and sources of carbohydrates in relation to risk of coronary heart disease: a prospective cohort study. J Am Coll Cardiol. 2015;66(14):1538–48.
- Lichtenstein AH, Appel LJ, Brands M, Carnethon M, Daniels S, Franch HA, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. Circulation. 2006;114:82–96.

- 49. Expert Panel on Detection Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Executive summary of the third report of the National Cholesterol Education Program (NCEP). JAMA. 2001;285(19):2486–97.
- Dietary Guidelines for Americans. 2015. http://health.gov/dietaryguidelines/2015-scientific-report/pdfs/scientific-report-of-the-2015-dietary-guidelines-advisory-committee.pdf.
- Lapointe A, Balk EM, Lichtenstein AH. Gender differences in plasma lipid response to dietary fat. Nutr Rev. 2006;64(5 Pt 1):234–49.
- Reidlinger DP, Darzi J, Hall WL, Seed PT, Chowienczyk PJ, Sanders TA. How effective are current dietary guidelines for cardiovascular disease prevention in healthy middle-aged and older men and women? A randomized controlled trial. Am J Clin Nutr. 2015;101(5):922–30.
- 53. Prince RL. Diet and the prevention of osteoporotic fractures. N Engl J Med. 1997;337(10):701-2.
- 54. Faine MP. Dietary factors related to preservation of oral and skeletal bone mass in women. J Prosthet Dent. 1995;73(1):65–72.
- 55. Chung M, Balk E, Bendel M, Ip S, Lee J, Lichtenstein AH, et al. Vitamin D and calcium: systematic review of health outcomes. evidence report/technology assessment No.183. (prepared by tufts evidence-based practice centre under contract no.290-2007-10055-I) AHRQ Publication No. 09-E015, Rockville MD: Agency for Healthcare Research and Quality; 2009.
- Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, et al. Explaining the decrease in U.S. deaths from coronary disease, 1980–2000. N Engl J Med. 2007;356(23):2388–98.
- Ford ES, Li C, Zhao G, Pearson WS, Mokdad AH. Prevalence of the metabolic syndrome among U.S. adolescents using the definition from the International Diabetes Federation. Diabetes Care. 2008;31(3):587–9.
- Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med. 2001;344(18):1343–50.
- Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med. 2002;346(6):393–403.
- 60. Sakane N, Sato J, Tsushita K, Tsujii S, Kotani K, Tsuzaki K, et al. Prevention of type 2 diabetes in a primary healthcare setting: three-year results of lifestyle intervention in Japanese subjects with impaired glucose tolerance. BMC Public Health. 2011;11:40–9.
- Wing RR, Bolin P, Brancati FL, Bray GA, Clark JM, Coday M, et al. Cardiovascular effects of intensive lifestyle intervention in type 2 diabetes. N Engl J Med. 2013;369(2):145–54.
- Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, et al. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997;336(16):1117–24.
- 63. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. DASH-Sodium Collaborative Research Group. N Engl J Med. 2001;344(1):3–10.
- 64. Meydani SN, Wu D. Nutrition and age-associated inflammation: implications for disease prevention. JPEN J Parenter Enteral Nutr. 2008;32(6):626–9.
- 65. Meydani SN, Wu D. Age-associated inflammatory changes: role of nutritional intervention. Nutr Rev. 2007;65(12 Pt 2):S213–6.
- 66. Meydani SN, Leka LS, Fine BC, Dallal GE, Keusch GT, Singh MF, et al. Vitamin E and respiratory tract infections in elderly nursing home residents: a randomized controlled trial. JAMA. 2004;292(7):828–36.
- Serra-Majem L, La Vecchia C, Ribas-Barba L, Prieto-Ramos F, Lucchini F, Ramon JM, et al. Changes in diet and mortality from selected cancers in southern Mediterranean countries, 1960–1989. Eur J Clin Nutr. 1993;47 Suppl 1:S25–34.
- Johnson K, Kligman EW. Preventive nutrition: disease-specific dietary interventions for older adults. Geriatrics. 1992;47(11):39–40.
- 69. Bostick RM, Potter JD, McKenzie DR, Sellers TA, Kushi LH, Steinmetz KA, et al. Reduced risk of colon cancer with high intake of vitamin E: the Iowa Women's Health Study. Cancer Res. 1993;53(18):4230–7.
- Bostick RM, Potter JD, Sellers TA, McKenzie DR, Kushi LH, Folsom AR. Relation of calcium, vitamin D, and dairy food intake to incidence of colon cancer among older women. The Iowa Women's Health Study. Am J Epidemiol. 1993;137(12):1302–17.
- Jevtic M, Velicki R, Popovic M, Cemerlic-Adjic N, Babovic SS, Velicki L. Dietary influence on breast cancer. J BUON. 2010;15(3):455–61.
- 72. Kushi LH, Byers T, Doyle C, Bandera EV, McCullough M, Gansler T, et al. American Cancer Society guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. CA Cancer J Clin. 2006;56(5):254–81.

Part VI Controversies in Nutrition and Lifestyle Medicine

Chapter 20 Added Sugars and Health: What Do We Really Know?

James M. Rippe and Theodore J. Angelopoulos

Key Points

- The health effects of added sugars remain controversial with mixed evidence.
- Most of the putative adverse effects of added sugars relate to their fructose component which is well known to be metabolized differently than glucose.
- Major fructose-containing sugars in the human diet are sucrose and high fructose corn syrup (HFCS) as well as concentrated fruit juices all of which are comprised of approximately 50% fructose and 50% glucose.
- Current research provides mixed evidence related to adverse effects of added sugars; however, research where added sugars are substituted isocalorically for other carbohydrates does not suggest any unique adverse metabolic effects of these sweeteners.

 $\label{eq:construction} Keywords \ \ \ Fructose \bullet Glucose \bullet High fructose \ \ \ \ corn \ syrup (HFCS) \bullet Sucrose \bullet Diabetes \bullet Cardiovascular \ disease$

Introduction/Background

The potential health consequences of consuming added sugars have resulted in considerable controversy and debate, particularly over the past decade [1-14]. Numerous research studies both in humans and animals have been published related to the effects of added sugars in the diet on various health issues [15-17]. Human studies have included epidemiologic and cohort studies as well as randomized controlled trials and meta-analysis. It should be noted that many research studies have compared pure fructose to pure glucose, assessing metabolic and health effects despite the fact that these two

J.M. Rippe, MD (🖂)

Rippe Lifestyle Institute, 21 North Quinsigamond Avenue, Shrewsbury, MA 01545, USA e-mail: bgrady@rippelifestyle.com

T.J. Angelopoulos, MPH, PhD Department of Physical Therapy, School of Health Sciences, Emory & Henry College, Emory, VA, USA e-mail: tangelop123@comcast.net

monosaccharides are rarely consumed in isolation in the human diet [15–17]. The commonly consumed fructose-containing sugars, namely, fructose itself, high fructose corn syrup (HFCS) (also known as isoglucose in Europe), and sucrose, have been the subject of multiple misconceptions and are some of the most poorly understood nutrients in all of nutrition.

The potential health effects of added sugars raise not only important scientific questions but are considerable interest to the media, the public, and regulatory bodies. Added sugars have been blamed for contributing to the worldwide obesity epidemic [4–6] and contributing to or even causing diabetes [18, 19], increasing the likelihood of coronary artery disease [19], and contributing to the rise of the metabolic syndrome [20], non-alcoholic fatty liver disease (NAFLD) [7], hypertension [20], and various lipid disorders [21]. All of these findings and assertions have been challenged by other investigators relying on high-level evidence to the contrary from randomized controlled trials (RCTs), prospective cohort studies, and meta-analyses.

It has even been asserted that added sugars may be "addictive" and lead to the overconsumption of calories [7, 8, 22, 23]. This concept has been strongly disputed by researchers who have challenged the assertion that sugars are addictive and challenged the fundamental concept of whether "sugars addiction" or even more broadly, "food addiction" even exists except under narrowly restricted experimental conditions and in small population groups [24–26].

There is considerable debate among scientific organizations about what the proper recommended upper limit of sugars should be. The American Heart Association (AHA) [27], World Health Organization (WHO) [28], and the Scientific Advisory Committee on Nutrition (SACN) in England [29] have all recommended significant restrictions in the amount of calories consumed from added sugars and recommended an upper limit of no more than 10% of calories from all added sugars combined. This is the same recommendation made by the Dietary Guidelines for Americans 2015 [30] and is the basis for the Food and Drug Administration (FDA) proposed recommendation of no more than 10% of calories from added sugars and a proposed rule calling out added sugars as a separate line item on the nutrition facts panel and establishing a 10% recommended upper limit [31]. Other organizations including the Institute of Medicine (IOM) Carbohydrate Report [32] and the Dietary Guidelines for Americans 2010 (DGA 2010) [33] as well as the European Food Safety Authority (EFSA) [34] have not found any harm in added sugars below the 25% of calories consumption level.

The concern about sugars sweetened beverages (SSBs) potentially contributing to multiple adverse health consequences has even caused some schools to eliminate chocolate milk from their school lunch menu thereby creating a host of unintended consequences including significant reduction in overall milk consumption and attendant reductions in three of the four "shortfall" nutrients cited in the DGA 2010 and 2015 reports [35]. Several state legislators have explored potentially banning added sugars from school lunch menus, and the former Mayor of New York advocated prohibiting certain size SSBs from restaurants regulated by the Department of Health in New York City based on his belief that this could make a meaningful contribution to obesity prevention. This concept was disputed by many scientists and ultimately struck down by the courts.

Several prominent scientists have also suggested taxation or other regulatory measures to limit consumption of added sugars [36, 37], although very little evidence exists suggesting a link between taxation and consumption.

Over the past 4 years, the amount of fructose-containing sugars consumed in the USA and worldwide has increased along with other major sources of calories in the diet including fat, flours, and cereal products [13]. While several epidemiologic studies have linked consumption of SSBs with risk of heart disease [38, 39], obesity [4, 6, 9], hypertension [40, 41], and a decrease in dietary quality, others have disputed these findings, leading to an intense scientific debate [3, 42].

With all this as background, the current chapter will attempt to provide basic facts concerning composition, production, and consumption of added sugars as well as the health effects of these sweeteners. While there are numerous types of added sugars, this chapter will focus on fructose-containing sugars including sucrose, HFCS, and fructose itself since the first two are predominant

sources of added sugars in the human diet and have been the center of controversy related to potential adverse health consequences and it is the fructose moiety of these sugars which has been blamed for these adverse consequences. It is important to note, however, that additional fructose-containing sweeteners in the diet may include honey, fruit juice concentrate, and agave nectar.

Composition of Fructose, HFCS, Sucrose, and Other Nutritive (Caloric) Sweeteners

The compositions of the most common nutritive sweeteners are listed in Table 20.1. The major sweeteners used in commerce include HFCS-42 and HFCS-55 as well as sucrose. Occasionally HFCS is confused with common corn syrup or pure fructose, but as illustrated in Table 20.1, the proper comparison is between HFCS-42 or HFCS-55 and sucrose since they are very similar in composition. All three of these sweeteners contain roughly 50% of glucose and fructose. The same 1:1 ratio of glucose and fructose is found in over 50 fruits and fruit juices as well as vegetables and nuts.

The major differences between sucrose and HFCS pre-digestion is that the glucose and fructose in sucrose is bound together in a covalent bond whereas fructose and glucose reside as monosaccharides in HFCS. However, it is important to recognize that in many instances the bond between fructose and glucose has already been hydrolyzed (broken) prior to ingestion. This process is called inversion and can be catalyzed by either warm temperature or low pH (many carbonated soft drinks, for example, have a pH of 3.5 and are often stored in warehouses at ambient temperature sometimes for weeks). Thus, in many instances all of the sucrose in sucrose-sweetened soft drinks may already have hydrolyzed to invert sugar where fructose and glucose are not bound together. In addition, any remaining bonds between fructose and glucose in sucrose are rapidly hydrolyzed by the enzyme sucrase within the human intestine. Once either sucrose or HFCS is metabolized, there are essentially no distinctions between the products that are actually experienced by the human body since both are absorbed into the bloodstream as pure fructose and pure glucose.

Consumption of Sugars

There are many misconceptions about the consumption of added sugars. These have contributed to a misguided focus on added sugars as the driving force for obesity and other public health concerns. Figure 20.1 shows trends in consumption of sugars, HFCS, added sugars, and fructose. This figure is

Component	HFCS-42%	HFCS-55%	Corn syrup %	Fructose %	Sucrose %	Invert sugar ^a %	Honey %
Fructose	42	55	0	100	50	45	49
Glucose	53	42	100	0	50	45	43
Others	5 ^b	3 ^b	0	0	0	10 ^c	5 ^d
Moisture	29	23	20	5	5	25	18

 Table 20.1
 Sugars composition of common nutritive (caloric) sweeteners, as consumed

Adapted from references White [17], Buck [118], Chen and Chou [119], Clarke [120]

^aTotal invert sugar - nearly completely hydrolyzed (inverted) sucrose using acid or enzyme (invertase)

HFCS high fructose corn syrup

^bReadily hydrolyzable oligomers of glucose

^cUnhydrolyzed sucrose

^dSucrose and minor amounts of other carbohydrates

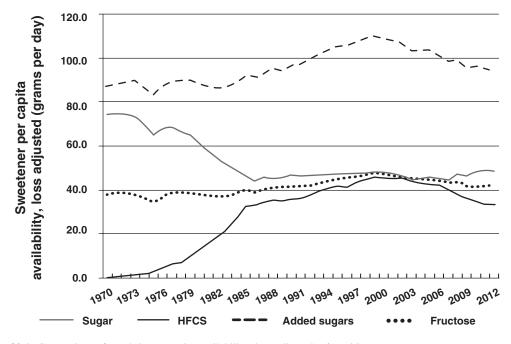


Fig. 20.1 Comparison of trends in per capita availability (loss adjusted) of nutritive sweeteners

derived from USDA-ERS per capita availability data, adjusted for loss [43]. It provides a reasonable estimate for the actual amount of sugars per capita ingested for the years 1970–2012.

Several issues are important to emphasize about the data presented in this Figure. First, sucrose and HFCS consumption curves are nearly mirror images. In other words, when HFCS consumption started assuming more importance in the beverage industry, there was a corresponding fall in sucrose consumption. Secondly, HFCS consumption grew rapidly after 1985 and peaked in 1999 and has been in continual decline for the past 15 years. Consumption in 2012 was comparable to that in 1987. Third, individuals in the USA consume about 1.5 times as much sucrose as HFCS. Fourth, the consumption of fructose, which has claimed to be an important contributor to many significant diseases, has been relatively constant for the past 40 years. Fifth, per capita sugars consumption in the USA has significantly declined since 1999. In contrast, obesity rates rose throughout the decade from 2000 to 2010 [44]. Thus, a reported correlation between sugars consumption and obesity should be interpreted with caution.

The emphasis on added sugars suggesting that consumption of these nutritive sweeteners has increased disproportionally in the US diet is almost certainly incorrect. Between the years 1970 and 2010, it has been estimated that there was an increase of 449 kcals/day in daily energy intake per capita in the USA. Of that energy increase, calories from all added sugars accounted for just 34 cal – about 7% of the increase [43]. Over 90% of the increase came from increased consumption of added fats and flour/cereal products. Thus, it appears more reasonable to conclude that Americans are not overweight and obese because of added sugars consumption, Americans are overweight because they eat too much of everything.

Metabolism of Fructose, HFCS, and Sucrose [1, 12, 14, 45–49]

Several issues are important to emphasize when considering the metabolism and metabolic effects of fructose-containing sugars. First, humans rarely consume fructose alone. It is found together with roughly equivalent amounts of glucose in nearly every dietary source, including added sugars (e.g., sucrose,

HFCS, honey, fruit juice concentrates) and natural sources (e.g., fruits, vegetables, nuts). A minor exception exists in crystalline fructose, which is, as the name suggests, 100% fructose but this is a specialty sweetener used in very limited quantities making it insignificant compared to other sources.

It has been argued by some that the metabolic consequences of sucrose and HFCS are different. However, from a post-digestion standpoint, this argument is not based on good scientific ground. As illustrated in (Fig. 20.2), there is no difference in terms of what is delivered to the portal blood when either HFCS, honey, fruit juice concentrates, or sucrose are metabolized. In each of these instances, free fructose and glucose are delivered to the portal circulation. It is also important to view the consequences of sugars consumption in the context of the whole diet, not as isolated nutrients. Sugars are only one nutrition group contributing to the overall net body metabolism, particularly in a diet rich in fat, complex carbohydrates, and proteins.

Absorbed fructose and glucose pass to the liver via portal blood. It is estimated the liver metabolizes up to 90% of absorbed fructose, but only 20% of absorbed glucose. This is a major distinction. However, there is much more glucose in the diet (on the order of $3-5\times$ more glucose than fructose). Thus, the liver, in all likelihood, processes comparable levels of each. Moreover, even though the pathways for fructose and glucose are distinct, they are interactive, as illustrated in (Fig. 20.3). It has been estimated that 50% of ingested fructose is converted directly into glucose while another 15% is converted and stored as glycogen. Approximately 25% of ingested fructose is converted to lactate and a few percent to carbon dioxide. As illustrated in (Fig. 20.4), 1-5% of fructose may be converted into free fatty acids through the process of de novo lipogenesis. Investigators who have argued that fructose, through this final pathway, can create fatty liver, ignore the relatively minor quantity of fat generated though the metabolism of fructose. It has been estimated that approximately 1 g of fat in human

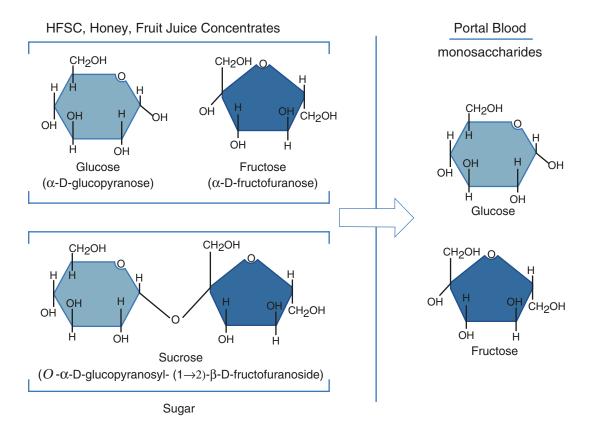
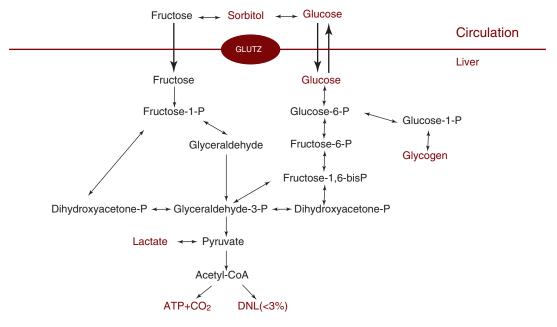


Fig. 20.2 Structures of fructose sweeteners before and after digestion



Fructose + Glucose = Merged metabolism

Merged metabolic pathyways of fructose and glucose

Fig. 20.3 Merged metabolic pathways of fructose and glucose

beings is produced via this mechanism on a daily basis compared to the 100–125 g of fat that human beings typically ingest on a daily basis.

Sugars and Energy-Regulating Hormones

Confusion about metabolism related to fructose, sucrose, and high fructose corn syrup was compounded by early studies in animals and humans, which often utilized a model comparing pure fructose to pure glucose, and often at very high dosages, to compare their effects on energy-regulating hormones such as insulin, leptin, and ghrelin [15, 16]. The scientific rationale for this approach came from the well-known differences in hepatic metabolism between these two monosaccharides which were outlined in the previous section of this chapter. It should be noted, however, that this type of experimentation creates a very artificial environment since neither of these monosaccharides is consumed to any appreciable degree in isolation in the human diet.

Teff et al. compared fructose-sweetened to glucose-sweetened beverages when both were consumed at 25% of calories [15]. These investigators demonstrated increased 24 h circulating insulin, glucose, and leptin and decreased post-prandial suppression of plasma ghrelin when comparing these two monosaccharides. Other investigators have also utilized a similar model comparing fructose to glucose and demonstrated that post-prandial triglycerides increased following fructose consumption particularly in overweight or obese subjects [16].

Given that insulin, leptin, and ghrelin interact with each other and play important roles in energy consumption, these data have been extrapolated to speculate that prolonged consumption of energy from fructose could contribute to increased caloric intake and ultimately weight gain and obesity. However, these experiments were repeated in our research laboratory and others utilizing similar levels of the more

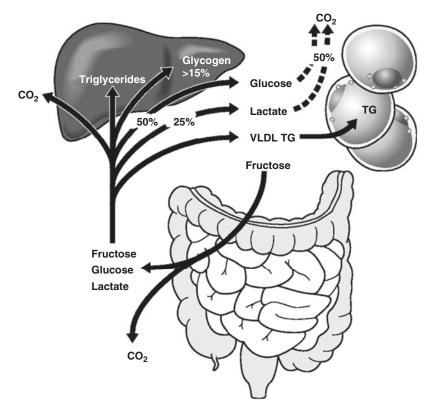


Fig. 20.4 Metabolic conversion of fructose in various organ systems. Note that a high percentage of ingested fructose is converted to glucose, glycogen and lactate (From Tappy and Le [47] with permission)

commonly consumed sugars, namely, HFCS and sucrose (each of which consists of approximately 50% fructose and 50% glucose). These "real world" studies showed no difference with respect to energy-regulating hormones, appetite, or *ad libitum* energy consumption [50] either the day of consuming, either of the two commonly consumed sugars, or the day after. These findings have been duplicated by another research group. In addition, a prospective, blinded study also in our research trial compared 8% of calories (25% percent population consumption level of fructose) to 18% of calories (the 50th percentile population consumption level of fructose) and demonstrated no differences between HFCS and sucrose with regard to energy-regulating hormones either in an acute setting or following 10 weeks of consumption at these levels [51].

Thus, the initial findings which compared the monosaccharides fructose and glucose and found differences with regard to energy-regulating hormones must be treated with great caution since studies that reflect the more real world situation where fructose and glucose are consumed together have not shown any differences between the major sources of added sugars in the human diet and have not yielded adverse metabolic consequences.

Sugars and Obesity

One of the most controversial areas related to sugars consumption and their potential health effects relates to their putative relationship to weight gain and obesity. In retrospect, modern concern about this issue can be traced back to a commentary in the *American Journal of Clinical Nutrition* by Bray,

Neilson, and Popkin in 2004 [6]. In this article, the authors argued there was a temporal association between the rapid increase in obesity prevalence in the USA and the use of HFCS in the food supply. They argued that the metabolism of fructose compared to glucose differed in such a way that energy consumption could be increased following fructose consumption, and thereby, increase the risk of obesity. Subsequent research showed that there was nothing unique about HFCS and that the metabolic effects were no different from sucrose.

The arguments of Bray, Nielson, and Popkin were not new since they were similar to the issues raised in 1972 by John Yudkin in his book *Pure*, *White and Deadly: How Sugar Is Killing Us and What We Can Do to Stop It* (updated in 1986 and 2012) [52]. In his book, Yudkin suggested that the nutritional differences between simple sugars and complex carbohydrates were such that sugars had adverse effects when consumed at levels typical in the Western diet. Scientific papers at that time from Sheldon Reiser (USDA, Beltsville, MD) and Gerald Reaven (Stanford University) also focused on the fructose component of sucrose and HFCS as a potential cause of obesity. Many of these arguments were addressed in the 1993 Fructose Nutrition monograph edited by Alan Forbes and Barbara Bowman (published in the *American Journal of Clinical Nutrition*), which concluded that "on the basis of currently available information there is little basis for recommending increase/decrease use of fructose in the general food supply or in products for special dietary use [53]."

Following the Bray/Nielson/Popkin commentary, a number of research organizations have conducted studies which have failed to support a unique link between HFCS and obesity [50, 54]. These studies have now shown that HFCS and sucrose are virtually identical with regard to calories, sweetness, and absorption and that when these sweeteners as well as fructose itself when substituted isocalorically for other carbohydrates were not uniquely related to weight gain or obesity [55–57].

Once the equivalence of HFCS and sucrose was established, focus turned to whether or not fructose-containing sugars in general might be uniquely linked to obesity [42, 58, 59]. Three systematic reviews and meta-analyses of randomized controlled trials explored specifically SSBs and body weight. All of these meta-analyses demonstrated that replacing sugar with other energy-equivalent macronutrients had no effect on body weight. Some evidence from these studies suggested that increasing overall energy consumption by adding sugars to a normal diet could lead to modest weight increase. However, this increase does not appear due to any unique property of sugars but an increase in energy consumed in hypercaloric trials.

Recent summary articles have also drawn the same conclusion – that there is a lack of evidence uniquely linking sugars to obesity. A systematic review by Te Morenga et al. [58] commissioned by the World Health Organization, reported that in *ad libitum* trials added sugars were associated with an increase in weight on average of 0.75 kg. However, when fructose-containing sugars were substituted isocalorically for other carbohydrates, no weight gain occurred. The authors concluded that the likely cause of weight gain in *ad libitum* was not due to sugar *per se*, but increased caloric consumption. Nonetheless, the WHO concluded that a linkage between added sugars and increased weight was "moderate" and recommended an upper limit of no more than 10% of calories from added sugars in the diet. The SCAN report in England, however, found no unique link of added sugars to obesity [29].

Thus, the evidence suggesting an association between added sugars consumption and weight gain and obesity should be viewed with considerable caution. Furthermore, in a disease as complicated as obesity it is highly unlikely that one single nutrient would be a unique cause. This view is also consistent with the recent scientific statement from the American Society of Nutrition emphasizing that energy regulation and weight are complicated and cautioned against isolating a single component of the diet as a primary cause of weight gain or obesity [60].

Sugars and Risk Factors for Cardiovascular Disease

Some epidemiologic studies have linked SSB consumption to increased risk of CHD which remains the leading cause of mortality worldwide [38, 39, 61]. In the USA, CHD and stroke account for 37% of all mortality on an annual basis. The number of risk factors for cardiovascular disease relate to nutritional practices. Fructose-containing sugars have recently been singled out as a potential cause of cardiovascular disease by the American Heart Association. In a Scientific Statement on sugars [27], the AHA advocated that adult males consume no more than 150 kcals/day from added sugars and females consume no more than 100 kcals/day from added sugars. The AHA acknowledged that these recommendations were not based on randomized controlled trials, but based largely on epidemiologic studies. Over 80% of all adults in the USA consume more added sugars on a daily basis than recommended in these guidelines.

There are no reported RCTs examining the effects of fructose-containing sugars on CHD itself. Thus, most information in this area comes from risk factors for CHD rather than CHD itself.

Prospective cohort studies have focused on an association between SSBs consumption and incidence of CHD. The Male Health Professionals Follow-up Study [38] and also the Nurses' Health Study found a significant association between CHD events and the highest quintile of SSB consumption [39]. However, another large prospective study reported by Eshak et al. found no association between SSBs and myocardial infarction [62]. All of these studies, of course, are subject to the limitations of prospective cohort studies which do not establish cause and effect.

Consumption of added sugars and risk of dyslipidemias has been reported in a number of studies. In particular, elevations in triglycerides in response to fructose-containing sugars have been the subject of numerous systematic reviews and meta-analyses. However, the findings have been mixed and variable [63–73]. There seems to a threshold effect at 20% of calories or more from added sugars where increased triglyceride levels may occur.

The AHA has recommended restricting consumption of fructose-containing sugars as one mechanism for controlling triglycerides [74]. Several recent reviews, however, have found no link between fructose consumption and either fasting triglycerides or post-prandial triglycerides when they are substituted isocalorically for other carbohydrates [63, 64]. Several research studies in our laboratory have yielded mixed results. In one research trial, at average levels of fructose-containing sugars (18% of calories from either HFCS or sucrose), no significant increase in triglycerides was found [68]. In another study comparing 8, 18, and 30% of calories from added sugars, however, a 10% increase in triglycerides was found [73].

Studies looking at the effects of added sugars on total cholesterol and LDL cholesterol have been similarly mixed with some studies supporting an increase in cholesterol and LDL cholesterol while others have not.

With regard to blood pressure, a meta-analysis by Te Morenga et al. found no increases in systolic blood pressure but a slight (1 mmHg) increase in diastolic pressure [75]. A recent meta-analysis of 13 randomized and non-randomized trials performed by Ha et al., however, did not show any increases in blood pressure with isocaloric exchange of fructose-containing sugars for other carbohydrates. A recent RCT in our research laboratory showed no adverse effect on blood pressure in response to fructose-containing sugars consumed up to the 90th percentile population consumption level for fructose [76].

Taken as a whole, these studies suggest that increases in risk factors for CHD based on fructosecontaining sugars are small and mixed. Nonetheless, the Dietary Guidelines Advisory Committee 2015 reported "moderate" evidence for recommending an individual consume no more than 10% of calories from added sugars to lower risk of CHD [30]. This recommendation, however, was based largely on overall dietary modeling rather than specific RCTs related to sugars and risk of cardiovascular disease.

Sugars and Diabetes

Diabetes has increased dramatically worldwide in the past 30 years [77]. It has been estimated that 6.4% of the population of the world currently has diabetes, and this is expected to rise to 7.7% worldwide by the end of 2030 [78]. This increase in diabetes has largely paralleled an increase in obesity and insulin resistance [79]. This has led to the exploration of a variety of dietetic factors related to the etiology of diabetes.

To our knowledge, there are no randomized controlled trials related to added sugars consumption and diabetes. Thus, most of the studies have focused on risk factors for diabetes. Several epidemiologic studies have linked consumption of SSBs to increased risk of diabetes [80, 81]. Two recent ecological studies also linked the rise of fructose availability (either from HFCS or sucrose) to an increase in prevalence of diabetes [18, 19]. These ecological analyses, however, are considered a weak form of evidence. Moreover, there are also ecologic studies which have failed to show a positive correlation between sugar intake and diabetes. In Australia, for example, there has been 10% decrease in caloric consumption from SSBs yet significant increases in obesity and diabetes over the past 10 years [82]. This has been called the "Australian Paradox." Similar "paradoxes" have been seen in the USA and the United Kingdom.

Prospective cohort studies provide mixed evidence concerning sugars consumption and diabetes [80]. In one meta-analyses of cohorts related to SSBs and incidence of diabetes, eight studies were analyzed; four did not find any significant effect of SSBs on incidence of diabetes and five did not adjust findings for energy intake and body weight. One large cohort study reported no association between diabetes risk and SSB consumption once data were adjusted for total energy intake [81]. Other prospective cohort studies have also failed to find significant associations between sugars intake and diabetes [83–85].

Meta-analyses of RCTs also did not support an association between sugars intake and diabetes. In one report by Cozma et al. involving controlled feeding studies, fructose had no impact on fasting insulin and glucose or glycosylated blood proteins (including HbA1C) [86]. Most RCTs in nondiabetic patients which have substituted fructose-containing sugars for other carbohydrates in an isocaloric fashion have also not yielded effects on multiple risk factors for diabetes including insulin, post-prandial glucose, and fasting glucose [87–91].

Taken together, there is little direct evidence for sugars consumption as a unique cause for increased risk for diabetes. However, it should be noted that most diabetes organizations recommend decreased sugars consumption as part of an overall strategy for controlling blood sugar. Moreover, since added sugars are energy dense, it would seem prudent to decrease consumption of added sugars as a strategy for controlling both weight and blood glucose.

Sugars and the Risk of the Metabolic Syndrome

The metabolic syndrome (MetS) represents a constellation of factors including abnormal glucose handling, dyslipidemia, and high blood pressure [92]. The prevalence of MetS has risen considerably in the USA in the past 20 years. Reports utilizing NHANES data have suggested the prevalence of MetS is up to 39% in adults [93]. MetS is a significant risk factor for both diabetes and CHD. The National Cholesterol Education Program guidelines (NCEP) recommend that individuals who have MetS be treated as though they already have CHD [92].

It has been postulated that the consumption of fructose-containing sugars may increase the risk of developing MetS. Johnson et al. have proposed a model where fructose metabolism in liver may lead to ATP depletion and ultimately increases in uric acid through the degradation to AMP [20]. According

this model, these increases in uric acid could lead to endothelial dysfunction and increase the risk of MetS to increased blood pressure, insulin resistance, and inflammation.

Excess accumulation of abdominal fat has been strongly associated with MetS [94]. Several investigators have published data suggesting that consumption of fructose or fructose-containing sugars may increase abdominal fat. In one, study Stanhope et al. reported increases in abdominal fat when comparing fructose at 25% of calories to glucose at 25% of calories [95]. Maersk et al. reported that daily consumption of a liter of sucrose-sweetened cola compared to a liter of diet cola, milk, or water resulted in an increase of abdominal fat and other risk factors for MetS [71]. RCTs conducted in our research laboratory, however, have not yielded increases in abdominal fat. In one study of 116 individuals who consumed fructose-containing sugars at the 25th, 50th, and 90th percentile population consumption, no increase in abdominal fat was found [73]. In another study involving 123 individuals who consumed HFCS and sucrose at 18% of calories compared to glucose and fructose at 9% of calories, slight decreases in HDL and increase in triglycerides occurred although no increase in abdominal fat occurred [96].

As already indicated, multiple other research trials have not yielded an association between added sugars consumption and elevated blood pressure, triglycerides, or post-prandial triglycerides when fructose-containing sugars are substituted isocalorically for other carbohydrates. In addition, a research trial conducted by our laboratory demonstrated no increase in uric acid levels under the curve following a 10 week consumption of up to 30% of calories from added sugars [76]. Thus, if fructose-containing sugars increase risk factors for MetS, the effects must be very small, if present.

Sugars and Liver Fat Accumulation Including Non-alcoholic Fatty Liver Disease (NAFLD)

Fatty infiltration of the liver leading to NAFLD now represents the leading cause of chronic liver failure and the need for liver transplantation around the world [97]. Because of the differences in metabolism between fructose and glucose, some investigators have argued that fructose consumption could increase the risk of NAFLD through accumulation of fat in the liver [98–100] (See Fig. 20.5).

However, it should be noted that various investigators have reported that only 1-5% of ingested fructose is converted to triglycerides through the process of de novo lipogenesis [101]. The amount of fat generated in this process is estimated to be 1% of fat typically consumed in the human diet. Moreover, multiple RTCs in humans have not demonstrated that fructose-containing sugars lead to increased fat in the liver. Studies reported that hypercaloric glucose and fructose similarly increased intrahepatic fat while isocaloric substation of fructose for carbohydrates did not [102]. These studies suggest that increases in liver fat appear to be energy mediated rather than a specific macronutrient-mediated effect. Moreover, a relationship between NAFLD and obesity is well established [97].

A study performed in our research laboratory comparing 8, 18, 30% of calories from added sugars showed no increased accumulation of liver fat over a 10 week period of added sugars consumption at these levels [103].

Sugars and Neurologic Response

Some animal experiments have suggested differences in brain responses comparing fructose to glucose [104, 105]. These experiments, however, must be treated with great caution because animal brains (particularly rodents, which have been used in many of these studies) differ in significant ways from the human brain.

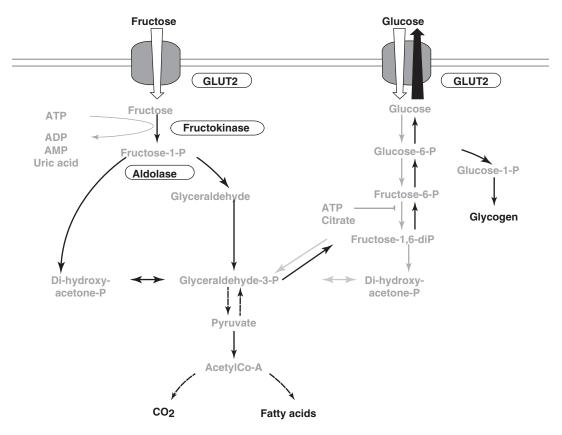


Fig. 20.5 Metabolism of the fructose and glucose in the liver (Source: Tappy and Le [47])

Technologies are now available employing functional magnetic resonance imaging (fMRI) to explore potential differential neurologic responses to various sugars in human beings [106]. Page et al. compared 75 g oral bolus of fructose to 75 g oral bolus of glucose in 20 healthy, young volunteers in a randomized, blinded fashion [22]. These investigators reported differences in hypothalamic blood flow with glucose suppressing blood flow assessed by arterial spin labeling while fructose did not suppress this blood flow. Purnell et al. explored the neurologic response to 25 g of either fructose or glucose delivered as an intravenous bolus [23]. This team of investigators reported no changes in blood flow to the hypothalamus, but differences between fructose and glucose in blood flow to the cerebral cortex.

Already noted in this chapter, both of these experiments compared highly artificial conditions with large doses of pure fructose or pure glucose, neither of which is consumed any appreciable degree in the human diet. Moreover, in the Purnell model, these monosaccharides were supplied through an atypical route (intravenously).

A pilot study conducted in our research laboratory compared average levels (18% of calories from HFCS or sucrose versus 9% of calories from either fructose or glucose alone) in the context of a mixed nutrient diet and found no differences in blood flow to the hypothalamus [107].

The issue of putative effects of fructose-containing sugars on the brain is very important since several investigators have suggested that the sweetness inherent to fructose-containing sugars creates the potential for individuals to become "addicted" to sugar. The research to support this, however, has been largely based on animal models. Several recent reviews have called into question the fundamental concept of either "food addiction" or "sugars addiction" except in very limited situations, such as binge eating disorder [24–26].

Public Health/Public Policy Considerations

Considerations related to the metabolism and health consequences of consuming sugars carry both important public health and public policy implications. For example, as already indicated, there are at least three different recommendations for appropriate upper limits for sugars consumption in the diet.

The World Health Organization (WHO) recommended an upper limit of consumption of 10% of calories [28]. The SACN in England adopted the same guidelines [29] as did the DGA 2015 [30]. However, the IOM carbohydrate report [32], the EFSA report [34], and the DGAC 2010 [33] all found no increase in chronic disease risk at up to 25% of calories. In this confusing environment, regulatory bodies in a variety of countries have struggled with how to make appropriate recommendations for added sugars consumption. Approximately 60 different countries in seven global geographic regions recommend consumers limit intake of added sugars; however, many of these recommendations are qualitative, while others specify quantities based on percent total sugars. The specific guidelines from various regions of the world are beyond the scope of this chapter but have been reviewed extensively elsewhere [108].

One approach intended to decrease SSBs consumption under the guise of global obesity reduction and promotion of weight loss and improved health is taxation [109, 110].

A number of policymakers in various countries favor such an intervention, yet multiple challenges exist including the determination of the amount of taxation needed to change consumption as well as the financial burdens which may ensue from such taxation. Moreover, taxation from added sugars may be politically difficult to achieve. A recent national survey on this issue in the USA involving a web-based survey among 50,000 participants indicated a lack of political trust (58%), and considerable opposition (52.5%), related to a proposed tax of a penny-per-ounce (for SSBs) [111]. Factors influencing the majority opposition to such a tax included that it would hurt the poor (51.2%) and upset the economy (48.9%).

A potential impact on taxation of SSBs NHANES data (1989–2006) suggested that such an approach may yield only moderate reduction of SSBs consumption among children and adolescents [112]. This study indicated a daily 6–8 kcal reduction associated with a soft drink tax, with a concomitant increase, albeit it slight, in consumption of juices and whole milk, which may be more nutritious alternatives. However, the same study did not find evidence for changes in population BMI, obesity, or overweight.

Investigations among adolescents have reported weak economic impact and no significant change in BMI following implementation of vending machine soda taxes (4.51% decrease in BMI) or grocery store soda taxes (~4.25% decrease in BMI) at the state level [113]. Despite these findings, several states and communities in the USA continue to advocate or legislate a tax on SSBs.

The argument that a tax on SSBs consumption may benefit public health may be complicated by both personal and political issues. For example, consumption tax would be considered a form of income tax and a regressive tax, particularly, on lower socioeconomic status individuals and the elderly.

This issue is further complicated by attempting to determine what level taxation would be required to change consumption. A small Internet supermarket study among 306 adult consumers in the Netherlands indicated that a tax on high-energy dense foods (50% tax or price increase) might lead to a reduction in consumption [114]. Furthermore, estimates of changes in BMI and obesity as a result of even 20–40% of taxation (0.5–1.0 cents per ounce) on SSBs suggest the impact on BMI would be marginal [115]. Mathematical models have suggested this level of taxation could reduce energy intake by 34–47 kcal/d among adults, which may translate into significant weight loss in 1–5 years [116]. However, other statistical estimates suggest that these figures may only relate to consumers who purchased a great amount of these products [117]. Thus, desired outcomes from taxation do not appear to be supported by numerous studies in various populations.

Summary/Conclusions

Evidence from multiple sources including RCTs, systematic reviews, and meta-analyses suggest there is nothing unique with regard to sugars consumption and adverse health consequences, provided the sugars are substituted isocalorically for other carbohydrates and consumed within the normal range of human consumption. Questions remain about whether or not when sugars are added to the normal diet in hypercaloric trials adverse health consequences may occur. Future research trials will be necessary to settle this issue.

For now, we believe it is reasonable to conclude that current scientific evidence does not support a unique relationship between sugars consumption and changes in energy-regulating hormones, obesity, diabetes, NAFLD, or risk factors for cardiovascular disease. Neurologic responses to sugars remain an area of active research; however, concepts such as sugars "addiction" do not appear to be currently supported by research trials in human beings or by expert opinion. It should be pointed out, however, that added sugars are energy dense, and it would appear prudent to limit all energy-dense food as one strategy to help control weight gain and obesity and the associated metabolic risk factors for CDH, diabetes, MetS, and NAFLD.

References

- 1. Rippe JM, Angelopoulos TJ. Sucrose, high-fructose corn syrup, and fructose, their metabolism and potential health effects: what do we really know? Adv Nutr. 2013;4(2):236–45.
- Rippe J. The metabolic and endocrine response and health implications of consuming sweetened beverages: findings from recent, randomized, controlled trials. Adv Nutr. 2013;4:677–86.
- Kahn R, Sievenpiper JL. Dietary sugar and body weight: have we reached a crisis in the epidemic of obesity and diabetes?: we have, but the pox on sugar is overwrought and overworked. Diabetes Care. 2014;37(4):957–62.
- Basu S, Yoffe P, Hills N, et al. The relationship of sugar to population-level diabetes prevalence: an econometric analysis of repeated cross-sectional data. PLoS One. 2013;8:e57873.
- Goran MI, Ulijaszek SJ, Ventura EE. High fructose corn syrup and diabetes prevalence: a global perspective. Glob Public Health. 2013;8:55–64.
- 6. Bray GA, Popkin BM. Dietary sugar and body weight: have we reached a crisis in the epidemic of obesity and diabetes?: health be damned! Pour on the sugar. Diabetes Care. 2014;37(4):950–6.
- Klurfeld DM, Foreyt J, Angelopoulos TJ, et al. Lack of evidence for high fructose corn syrup as the cause of the obesity epidemic. Int J Obes (Lond). 2012;27(6):771–3.
- Bray GA, Nielsen SJ, Popkin BM. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. Am J Clin Nutr. 2004;79:537–43.
- 9. Lustig RH. Fructose: metabolic, hedonic, and societal parallels with ethanol. J Am Diet Assoc. 2010;110(9): 1307-21.
- 10. Lustig RH, Schmidt LA, Brindis CD. Public health: the toxic truth about sugar. Nature. 2012;482(7383):27-9.
- 11. Bray G. Fructose: pure, white, and deadly? fructose, by any other name, is a health hazard. J Diabetes Sci Technol. 2010;4(4):1003–7.
- 12. Rippe JM. The health implications of sucrose, high-fructose corn syrup, and fructose: what do we really know? J Diabetes Sci Technol. 2010;4(4):1008–11.
- 13. Sievenpiper JL, de Souza RJ, Kendall CW, et al. Is fructose a story of mice but not men? J Am Diet Assoc. 2011;111(2):219–20; author reply 220–212.
- van Buul V, Tappy L, Brouns F. Misconceptions about fructose-containing sugars and their role in the obesity epidemic. Nutr Res Rev. 2014;27:119–30.
- Page KA, Luo S, Romero A, et al. Fructose compared to glucose ingestion preferentially activates brain reward regions in response to high-calorie food cues in young, obese Hispanic females. Endocrinol Rev. 2012;33: OR23–5.
- Purnell JQ, Klopfenstein BA, Stevens AA, et al. Brain functional magnetic resonance imaging response to glucose and fructose infusions in humans. Diabetes Obes Metab. 2011;13(3):229–34.
- 17. White J. Straight talk about high-fructose corn syrup. What it is and what it ain't. Am J Clin Nutr. 2008;88: 1716S–21.

- White JS. Challenging the fructose hypothesis: new perspectives on fructose consumption and metabolism. Adv Nutr. 2013;4(2):246–56.
- Teff KL, Grudziak J, Townsend RR, et al. Endocrine and metabolic effects of consuming fructose- and glucosesweetened beverages with meals in obese men and women: influence of insulin resistance on plasma triglyceride responses. J Clin Endocrinol Metab. 2009;94:1562–9.
- 20. Stanhope K, Griffen S, Keim N, et al. Consumption of fructose-, but not glucose sweetened beverages produces an atherogenic lipid profile in overweight/obese men and women. Diabetes. 2007;56 Suppl 1:A16.
- Havel P. Dietary fructose: implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. Nutr Rev. 2005;63:133–57.
- Johnson R, Segal M, Sautin Y, et al. Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. Am J Clin Nutr. 2007;86: 899–906.
- Dhingra R, Sullivan L, Jacques PF, et al. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. Circulation. 2007;116:480–8.
- 24. Benton D. The plausibility of sugar addiction and its role in obesity and eating disorders. Clin Nutr. 2010;29(3):288–303.
- Ziauddeen H, Farooqi I, Fletcher P. Obesity and the brain: how convincing is the addiction model? Nature Rev. 2012;13:279.
- Corwin LW, Hayes JE. Are the sugars addictive? Perspectives for practitioners. In: Rippe JM, editor. Fructose, high fructose corn syrup, sucrose and health. New York: Springer Publications; 2014. p. 199–215.
- 27. Johnson R, Appel L, Brands M, et al. American Heart Association Nutrition Committee of the Council on nutrition, physical activity, and metabolism and the council on epidemiology and prevention. Dietary sugars intake and cardiovascular health: a Scientific Statement from the American Heart Association. Circulation. 2009;120:1011–20.
- World Health Organization (WHO). Draft guideline: sugars intake for adults and children. Online public consultation open: 5–31 Mar 2014. http://www.who.int/mediacentre/news/notes/2014/consultation-sugar-guideline/en/.
- Scientific Advisory Committee on Nutrition. Draft carbohydrates and health report. 26 June–1 Sept 2014. (http:// www.sacn.gov.uk/).
- U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015–2020 Dietary Guidelines for Americans. 8th ed. Available at http://health.gov/dietaryguidelines/2015/guidelines/. 2015. Epub Dec 2015.
- 31. Food labeling: revision of the nutrition and supplement facts labels; supplemental proposed rule to solicit comment on limited additional provisions. A proposed rule by the food and drug administration on 27 July 2015. https://www.federalregister.gov/articles/2015/07/27/2015-17928/food-labeling-revision-of-the-nutrition-and-supplement-facts-labels-supplemental-proposed-rule-to.
- 32. Institute of Medicine. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. Institute of Medicine of the National Academies; Washington, DC 2005.
- Center for Nutrition Policy and Promotion. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans. Washington, DC: US Department of Agriculture; 2010. http://www.nutriwatch. org/05Guidelines/dga_advisory_2010.pdf.
- 34. EFSA panel on food additives and nutrient sources added to food. Scientific opinion on the safety of steviol glycosides for the proposed uses as a food additive. EFSA J. 2010;8:1537. doi:10.2903/j.efsa.2010.1537.
- Clark K, Rippe J. Flavored milk, dietary quality and childhood nutrition. In: Rippe JM, editor. Fructose, high fructose corn syrup, sucrose and health. New York: Springer Publications; 2014. p. 229–46.
- 36. Brownell KD, Gold M. Food and addiction: a comprehensive handbook. Oxford: Oxford University Press; 2012.
- 37. Obesity: we need to move beyond sugar. Lancet. 2016;387(10015):199.
- de Koning L, Malik VS, Kellogg MD, et al. Sweetened beverage consumption, incident coronary heart disease and biomarkers of risk in men. Circulation. 2012;125:1735–41.
- Fung T, Malik V, Rexrode K, et al. Sweetened beverage consumption and risk of coronary heart disease in women. Am J Clin Nutr. 2009;89(4):1037–42. -1042.
- 40. Raben A, Vasilaras T, Møller A, et al. Sucrose compared with artificial sweeteners: different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. Am J Clin Nutr. 2002;76:721–9.
- Brown CM, Dulloo AG, Yepuri G, et al. Fructose ingestion acutely elevates blood pressure in healthy young humans. Am J Physiol Regul Integr Comp Physiol. 2008;294:R730–7.
- 42. Kaiser KA, Shikany JM, Keating KD, et al. Will reducing sugar-sweetened beverage consumption reduce obesity? Evidence supporting conjecture is strong, but evidence when testing effect is weak. Obes Rev. 2013;14:620–33.
- 43. USDA-ERS. Food availability (per capita) data system: loss-adjusted food availability [Internet]. Sugar and sweeteners (added), updated 9 Sept 2014 [cited 10 Nov 2014]. Available from: http://www.ers.usda.gov/data-products/ food-availability-(per-capita)-data-system.aspx -.UvjuyUJdVOE.

- 44. Welsh JA, Sharma AJ, Grellinger L, et al. Consumption of added sugars is decreasing in the United States. Am J Clin Nutr. 2011;94(3):726–34.
- 45. Latulippe ME, Skoog SM. Fructose malabsorption and intolerance: effects of fructose with and without simultaneous glucose ingestion. Crit Rev Food Sci Nutr. 2011;51(7):583–92.
- 46. Marriott BP, Fink CJ, Krakower T. Worldwide consumption of sweeteners and recent trends. In: Rippe JM, editor. Fructose, high fructose corn syrup, sucrose and health. New York: Springer Science+Business Media; 2014.
- Tappy L, Le KA. Metabolic effects of fructose and the worldwide increase in obesity. Physiol Rev. 2010;90(1):23–46.
- Tappy L, Elgi L, Tran C. Metabolism of nutritive sweeteners in humans. In: Rippe J, editor. Fructose, high fructose corn syrup, sucrose and health. New York: Springer; 2014. p. 35–50.
- 49. Sun SZ, Empie MW. Fructose metabolism in humans what isotopic tracer studies tell us. Nutr Metab (Lond). 2012;9(1):89.
- Melanson K, Zukley L, Lowndes J, et al. Effects of high-fructose corn syrup and sucrose consumption on circulating glucose, insulin, leptin, and ghrelin and on appetite in normal-weight women. Nutrition. 2007;23:103–12.
- 51. Yu Z, Lowndes J, Rippe J. High-fructose corn syrup and sucrose have equivalent effects on energy-regulating hormones at normal human consumption levels. Nutr Res. 2013;33:1043–52.
- 52. Yudkin J. Pure, white, and deadly. HarperCollins Distribution Services; New York. 1972.
- Glinsmann WH, Bowman BA. The public health significance of dietary fructose. Am J Clin Nutr. 1993;58:820S–3.
- Soenen S, Westerterp-Plantenga MS. No differences in satiety or energy intake after high fructose corn syrup, sucrose, or milk preloads. Am J Clin Nutr. 2007;86:1586–94.
- Dolan LC, Potter SM, Burdock GA. Evidence-based review on the effect of normal dietary consumption of fructose on development of hyperlipidemia and obesity in healthy, normal weight individuals. Crit Rev Food Sci Nutr. 2010;50(1):53–84.
- Dolan LC, Potter SM, Burdock GA. Evidence-based review on the effect of normal dietary consumption of fructose on blood lipids and body weight of overweight and obese individuals. Crit Rev Food Sci Nutr. 2010;50(10):889–918.
- Sievenpiper JL, de Souza RJ, Mirrahimi A, et al. Effect of fructose on body weight in controlled feeding trials: a systematic review and meta-analysis. Ann Intern Med. 2012;156:291–304.
- Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. BMJ. 2013;346:e7492.
- Malik VS, Pan A, Willett WC, et al. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. Am J Clin Nutr. 2013;98:1084–102.
- 60. Hall KD, Heymsfield SB, Kemnitz JW, et al. Energy balance and its components: implications for body weight regulation. Am J Clin Nutr. 2012;95:989–94.
- 61. Gross LS, Li L, Ford ES, et al. Increased consumption of refined carbohydrates and the epidemic of type 2 diabetes in the united states: an ecologic assessment. Am J Clin Nutr. 2004;79:774–9.
- 62. Eshak ES, Iso H, Kokubo Y, et al. Soft drink intake in relation to incident ischemic heart disease, stroke, and stroke subtypes in Japanese men and women: the Japan public health centre-based study cohort. Am J Clin Nutr. 2012;96:1390–7.
- Chiavaroli L, Mirrahimi A, De Souza RJ, et al. Does fructose consumption elicit a dose–response effect on fasting triglycerides? A systematic review and meta-regression of controlled feeding trials. Can J Diabetes. 2012;36(5):S37.
- 64. Wang DD, Sievenpiper JL, de Souza RJ, et al. Effect of fructose on postprandial triglycerides: a systematic review and meta-analysis of controlled feeding trials. Atherosclerosis. 2014;232(1):125–33.
- 65. Zhang Y, An T, Zhang R, et al. Very high fructose intake increases serum LDL-cholesterol and total cholesterol: a meta-analysis of controlled feeding trials. J Nutr. 2013;143(9):1391–8.
- 66. Livesey G, Taylor R. Fructose consumption and consequences for glycation, plasma triacylglycerol, and body weight: meta-analyses and meta-regression models of intervention studies. Am J Clin Nutr. 2008;88(5): 1419–37.
- 67. Sievenpiper JL, Carleton AJ, Chatha S, et al. Heterogeneous effects of fructose on blood lipids in individuals with type 2 diabetes: systematic review and meta-analysis of experimental trials in humans. Diabetes Care. 2009;32(10):1930–7. 89.
- Angelopoulos T, Lowndes J, Sinnett S, Rippe J. Fructose Containing Sugars at Normal Levels of Consumption do not Effect Adversely Components of the Metabolic Syndrome and Risk Factors for Cardiovascular Disease. Nutrients. 2016;8(4):179. doi:10.3390/nu8040179.
- 69. Lowndes J, Sinnett S, Pardo S, et al. The effects of normally consumed amounts of sucrose or high fructose corn syrup on lipid profiles, body composition, and related parameters in overweight/obese subjects. Nutr. 2014;6(3):1128–44.
- Lê K-A, Faeh D, Stettler R, et al. A 4-wk high-fructose diet alters lipid metabolism without affecting insulin sensitivity or ectopic lipids in healthy humans. Am J Clin Nutr. 2006;84:1374–9.

- Maersk M, Belza A, Stødkilde-Jørgensen H, et al. Sucrose-sweetened beverages increase fat storage in the liver, muscle, and visceral fat depot: a 6-mo randomized intervention study. Am J Clin Nutr. 2012;95:283–9.
- Ha V, Sievenpiper JL, de Souza RJ, et al. Effect of fructose on blood pressure a systematic review and metaanalysis of controlled feeding trials. Hypertension. 2012;59:787–95.
- Lowndes J, Sinnett S, Yu Z, et al. The effects of fructose-containing sugars on weight, body composition and cardiometabolic risk factors when consumed at up to the 90th percentile population consumption level for fructose. Nutr. 2014;6:3153–68.
- Miller M, Stone N, Ballantye C, et al. Triglycerides and cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2011;123:2292–333.
- 75. Te Morenga LA, Howatson AJ, Jones RM, et al. Dietary sugars and cardiometabolic risk: systematic review and meta-analyses of randomized controlled trials of the effects on blood pressure and lipids. Am J Clin Nutr. 2014;100(1):65–79.
- Angelopoulos TJ, Lowndes J, Sinnett S, et al. Fructose containing sugars do not raise blood pressure or uric acid at normal levels of human consumption. J Clin Hypertens (Greenwich). 2015;17(2):87–94. Epub 2014/12/17.
- 77. International Diabetes Federation. IDF diabetes atlas. 2012. http://www.idf.org/diabetesatlas/5e/update2012.
- 78. Hu FB. Globalization of diabetes: the role of diet, lifestyle and genes. Diabetes Care. 2011;34(6):1249–57.
- Rippe J, Angelopoulos T. Preventing and managing obesity: the scope of the problem. In: Rippe J, Angelopoulos T, editors. Obesity: prevention and treatment. Boca Raton: CRC Press; 2012. p. 3–19.
- Malik VS, Popkin BM, Bray GA, et al. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diabetes Care. 2010;33:2477–83.
- Janket SJ, Manson JE, Sesso H, et al. A prospective study of sugar intake and risk of type 2 diabetes in women. Diabetes Care. 2003;26(4):1008–15.
- Barclay AW, Brand-Miller J. The Australian Paradox: a substantial decline in sugars intake over the same timeframe that overweight and obesity have increased. Nutrients. 2011;3(2):491–504. Correction: Nutrients. 2014; 6(2):663–664.
- Hodge AM, English DR, O'Dea K, et al. Glycemic index and dietary fiber and the risk of type 2 diabetes. Diabetes Care. 2004;27(11):2701–6.
- Colditz GA, Manson JE, Stampfer MJ, et al. Diet and risk of clinical diabetes in women. Am J Clin Nutr. 1992;55(5):1018–23.
- Meyer KA, Kushi LH, Jacobs DR, et al. Carbohydrates, dietary fiber, and incident type 2 diabetes in older women. Am J Clin Nutr. 2000;71(4):921–30.
- Cozma AI, Sievenpiper JL, de Souza RJ, et al. Effect of fructose on glycemic control in diabetes: a systematic review and meta-analysis of controlled feeding trials. Diabetes Care. 2012;35(7):1611–20.
- 87. Teff K, Elliott S, Tschop M, et al. Dietary fructose reduces circulating insulin and leptin, attenuates postprandial suppression of ghrelin, and increases triglycerides in women. J Clin Endocrinol Metab. 2004;89:2963–72.
- Aeberli I, Gerber PA, Hochuli M, et al. Low to moderate sugar-sweetened beverage consumption impairs glucose and lipid metabolism and promotes inflammation in healthy young men: a randomized controlled trial. Am J Clin Nutr. 2011;94(2):479–85.
- 89. Aeberli I, Hochuli M, Gerber PA, et al. Moderate amounts of fructose consumption impair insulin sensitivity in healthy young men: a randomized controlled trial. Diabetes Care. 2013;36(1):150–6.
- 90. Stanhope KL, Griffen SC, Bremer AA, et al. Metabolic responses to prolonged consumption of glucose- and fructose-sweetened beverages are not associated with postprandial or 24-h glucose and insulin excursions. Am J Clin Nutr. 2011;94(1):112–9.
- Beck-Nielsen H, Pedersen O, Lindskov HO. Impaired cellular insulin binding and insulin sensitivity induced by high-fructose feeding in normal subjects. Am J Clin Nutr. 1980;33(2):273–8.
- Program NCE. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation and treatment of high blood cholesterol in adults (Adult Treatment Panel III). JAMA. 2001;285:2486–97.
- Ford ES, Giles WH, Mokdad AH. Increasing prevalence of the metabolic syndrome among US adults. Diabetes Care. 2004;27(10):2444–9.
- Seip R. Beyond subcutaneous fat. In: Rippe J, Angelopoulos T, editors. Obesity: prevention and treatment. 1st ed. Boca Raton: CRC Press; 2013. p. 381–408.
- 95. Stanhope K, Schwarz J, Keim N, et al. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. J Clin Invest. 2009;119(5):1322–34.
- 96. Lowndes J, Lu N, Sinnett S, et al. No effect of the type of sugar on changes in traditional risk factors for cardiovascular disease when consumed at the typical levels. Circulation. 2013;128:A13008.
- Welsh JA, Karpen S, Vos MB. Increasing prevalence of nonalcoholic fatty liver disease among United States adolescents, 1988–1994 to 2007–2010. J Pediatrics. 2013;162(3):496–500 e491.
- Ouyang X, Cirillo P, Sautin Y, et al. Fructose consumption as a risk factor for non-alcoholic fatty liver disease. J Hepatol. 2008;48(6):993–9.

- 99. Thuy S, Ladurner R, Volynets V, et al. Nonalcoholic fatty liver disease in humans is associated with increased plasma endotoxin and plasminogen activator inhibitor 1 concentrations and with fructose intake. J Nutr. 2008;138(8):1452–5.
- Parks EJ, Skokan LE, Timlin MT, et al. Dietary sugars stimulate fatty acid synthesis in adults. J Nutr. 2008;138(6):1039–46.
- Hellerstein MK, Schwarz JM, Neese RA. Regulation of hepatic de novo lipogenesis in humans. Annu Rev Nutr. 1996;16:523–57.
- 102. Johnston RD, Stephenson MC, Crossland H, et al. No difference between high-fructose and high-glucose diets on liver triacylglycerol or biochemistry in healthy overweight men. Gastroenterology. 2013;145:1016–25 e1012.
- 103. Bravo S, Lowndes J, Sinnett S, et al. Consumption of sucrose and high-fructose corn syrup does not increase liver fat or ectopic fat deposition in muscles. Appl Physiol Nutr Metab. 2013;38(6):681–8. Epub 2013/06/04.
- 104. Funari VA, Herrera VL, Freeman D, et al. Genes required for fructose metabolism are expressed in Purkinje cells in the cerebellum. Brain Res Mol Brain Res. 2005;142(2):115–22.
- 105. Lindqvist A, Mohapel P, Bouter B, et al. High-fat diet impairs hippocampal neurogenesis in male rats. Eur J Neurol. 2006;13(12):1385–8.
- Matsuda M, Liu Y, Mahankali S, et al. Altered hypothalamic function in response to glucose ingestion in obese humans. Diabetes. 1999;48(9):1801–6.
- 107. Pena-Gomez C, Alonso-Alonso M, Bravo S, et al. (Submitted OSASM, 2013). Hypothalamic fMRI responses to different sugars under normal intake conditions: a pilot study. Presented, Obesity Society Annual Scientific Meeting. 2013;T-729-P.
- Clemens R, Papanikolaou Y. Crystalizing global sugar policy: public health promise or perception. In: Rippe JM, editor. Fructose, high fructose corn syrup, sucrose and health. New York: Springer; 2014. p. 125–35.
- 109. Jou J, Techakehakij W. International application of sugar-sweetened beverage (SSB) taxation in obesity reduction: factors that may influence policy effectiveness in country-specific contexts. Health Policy. 2012;107:83–90.
- 110. Andreyeva T, Chaloupka FJ, Brownell KD. Estimating the potential of taxes on sugar-sweetened beverages to reduce consumption and generate revenue. Prev Med. 2011;52:413–6.
- Barry CL, Niederdeppe J, Gollust SE. Taxes on sugar-sweetened beverages results from a 2011 national public opinion survey. Am J Prev Med. 2013;44:158–63.
- 112. Fletcher JM, Frisvold DE, Tefft N. The effects of soft drink taxes on child and adolescent consumption and weight outcomes. J Public Econ. 2010;94:967–74.
- Powell LM, Chriqui J, Chaloupka FJ. Associations between state-level soda taxes and adolescent body mass index. J Adoles Health. 2009;45:S57–63.
- 114. Waterlander WE, Steenhuis IHM, deBoer MR, et al. Introducing taxes, subsidies or both: the effects of various food pricing strategies in a web-based supermarket randomized trial. Prev Med. 2012;54:323–30.
- 115. Edwards RD. Commentary: soda taxes, obesity, and the shifty behavior of consumers. Prev Med. 2011;52:417–8.
- 116. Lin B-H, Smith TA, Lee J-Y, et al. Measuring weight outcomes for obesity intervention strategies: the case of a sugar-sweetened beverage tax. Econ Human Biol. 2011;9:329–41.
- 117. Finkelstein EA, Zhen C, Bilger M, et al. Implications of a sugar-sweetened beverage (SSB) tax when substitutions to non-beverage items are considered. J Health Econ. 2013;32:219–39.
- Buck AW. High fructose corn syrup. In: Nabors LO, editor. Alternative Sweeteners. 4th ed. Boca Raton: CRC Press; 2012.
- 119. Chen JCP, Chou C-C. Chen-Chou cane sugar handbook: a manual for cane sugar manufacturers and their chemists. 12th ed. New York: J. Wiley; 1993.
- Clarke MA. Cane Sugar. In: Kroschwitz JI, editor. Kirk-Othmer Concise Encyclopedia of Chemical Technology. 4th ed. New York: Wiley; 1999. p. 1915–7.

Chapter 21 Saturated Fat: Friend or Foe?

Benoît Lamarche

Key Points

- There is currently no absolute consensus regarding the association between dietary saturated fat (SFA) intake and cardiovascular health.
- Consumption of dietary SFA raises LDL-C but may have relatively neutral effects on several other cardiometabolic risk factors, especially when consumed as dairy foods.
- Observational cohort studies have generally reported neutral association between dietary SFA and the risk of CHD.
- Data from large randomized intervention studies in which SFA have been replaced by unsaturated fatty acids have yielded inconsistent results.
- More research is warranted to address some of the unanswered questions on this topic and to better inform future dietary guidelines.

Keywords Saturated fat • Ccoronary heart disease • Dietary guidelines • Polyunsaturated fat

Introduction

For a number of years, many health organizations have advocated for a reduction in the intake of saturated fat (SFA) as a means to prevent coronary heart disease (CHD). These recommendations were based on what was believed to be most convincing evidence relating SFA to heart disease. Yet, we have seen over the last 10 years or so a surge of research challenging this concept, which had gone essentially uncontested for almost 60 years. The resulting debate on the association between dietary SFA and heart health is highly polarized within scientific circles, and as a result the population is confused. Is it possible that nutrition researchers have been wrong for so many years regarding the ill effects of SFA? The press and social media is, of course, enjoying all of this, creating even more stir and confusion by publicizing the results of new emerging research on the topic, either way it goes. This chapter intends to shed light on the current status of knowledge regarding this now controversial association between dietary SFA and CHD risk. A brief historical perspective will first be provided to

B. Lamarche, PhD

Institute on Nutrition and Functional Foods, Laval University, 2440, boul. Hochelaga, Québec, QC, Canada, G1V 0A6 e-mail: Benoit.Lamarche@fsaa.ulaval.ca

put the debate into context. A short review of the significant literature on this topic will then be presented. The complexity of investigating the association between any given nutrient, including SFA, and health will be discussed. The chapter concludes with an overview of what should be considered research-wise and from a public health perspective to come to a close on this subject, for the benefit of the population.

SFA and Cardiovascular Health, a Long Story...

We have come a long way since any form of dietary guidelines was crafted. In the USA, the first food guide issued in 1916 included five "food groups," two of which were defined as "fatty foods" and "sugars." The recommendation on fat specifically was that it should represent approximately 30% of daily calories [1]. These recommendations were based much more on perceptions and feeling than on rigorous research.

The food environment reality over the following years has been influenced quite significantly by events such as the two World Wars (WW) and the Great Depression. Organizations such as USDA and also the US Food Administration (USFA), which was created during WW1, proposed new dietary recommendations and implemented restrictions to accommodate the reality of those hard times [2]. The Great Depression was a period during which government and policy makers focused primarily on providing enough food to the population, rather than on healthy nutrition [2]. WW2 again saw a time of food rationing with particular emphasis on meat, which was promoted as a "fighting food" and therefore targeted primarily to the troops on the field [3]. In 1941, the Food and Nutrition Board (FNB), created to prepare recommendations for wartime food policies, released "A Yardstick for Good Nutrition," which included the first minimum dietary requirements [4]. These are considered as the precursor of what we now refer to as recommended daily intakes (RDIs).

The Seven Countries Study in the early 1950s was really the first major research effort to address the complex relationship between food and health. It provided among others the first evidence suggesting that dietary SFA intake is associated with an increased risk of CHD death [5]. The ecological evidence that countries surrounding the Mediterranean Sea with a dietary SFA intake <7% of calories also had a very low rate of CHD death was enough to draw all attention on what is considered the "bad" fat of the diet [6]. The Seven Countries Study had an immense impact on how and which nutrition research questions were being addressed thereafter, by propelling research towards demonstrating and understanding this "ill" effect of SFA. But the inherent limitations of the Seven Countries Study due to the ecological nature of its design have also become more apparent over the subsequent years, with emergence of new research tools and more robust methodologies. Early studies by Keys et al. [5] have also been criticized for not having used all data available to them at the time of analysis, thereby potentially exaggerating the true association between SFA and CHD risk [7]. It is, of course, unthinkable in our modern days of nutrition research that dietary guidelines be based on evidence from studies such as the Seven Countries Study. And thankfully they are not.

Towards the end of the 1970s, the US Senate Select Committee on Health and Human Needs and the USDA engaged on a rather hostile debate regarding the impact of dietary fat and SFA on health. The position of the US Senate Committee was that there was a relatively unanimous perception within the research and health professional communities that intake of fat and SFA were probably the most important nutritional risk factors for chronic diseases including CHD [8, 9]. The USDA argued that there was no absolute scientific proof of the danger and risk posed by dietary fat and SFA [2]. In their report, the US Senate committee emphasized that dietary guidance provided by the USDA to date had not proved to be efficient in influencing the growing rates of chronic diseases associated with poor nutrition. This led the USDA to release their first dietary guidelines in 1980, which are revised every 5 years since then. The US Senate committee and USDA obviously did not agree on the wording of

their dietary recommendations. The USDA until 1990 used wording such as "avoid too much fat and SFA" [2] whereas recommendations from the US Senate Committee were more directive with specified targets for total fat (from 40% to about 30% of daily calories) and SFA (from 16% to about 10% of daily calories) [9]. The senatorial report ended up having a lot of influence in years to come, and in 1990, the USDA identified for the first time the target of <30% of calories as total fat and <10% of energy as SFA. It must be stressed that such numbers were not based on empirical data, at least there is no trace nor reference of robust and rigorous research to support these numbers specifically. The 2010 DGAC report was criticized for having included an incomplete body of relevant science, particularly regarding SFA [10]. Hence, the committee behind the 2015 DGAC revisions has determined that a reexamination of the evidence relating dietary SFA to health was necessary to determine whether revisions to the guidance were warranted. These most recent revisions in the dietary guidelines for Americans will be discussed later in this chapter. But before that, what evidence is causing this stir within nutrition circles to revisit the impact of dietary SFA on cardiovascular health?

Dietary SFA and CHD Risk

The association between SFA and the risk of CHD can be assessed using a variety of experimental approaches. These include purely mechanistic research based on cellular and animal models, clinical intervention research based on surrogate markers of CHD such as LDL-C concentrations and blood pressure, and epidemiological research, from which associations between a nutrient or food and a health outcome can be derived. While all such approaches are important, they often lead to incongruent results, thereby contributing to the general confusion on any particular topic. This chapter will focus on intervention as well as epidemiological research, as this is the evidence that has the most weight when dietary guidelines are being crafted.

Evidence from Intervention Trials

Dietary SFA Versus Cardiometabolic Risk

There is very little debate regarding the LDL-C raising effects of SFA. A very influential meta-analysis of RCTs published prior to 2003 has shown that isocaloric substitution of carbohydrates for SFA increases plasma LDL-C significantly and linearly [11]. Dietary SFA also raise LDL-C compared with monounsaturated and polyunsaturated fats [11]. This is perhaps the most evoked evidence supporting the importance of reducing SFA intake, considering that elevated LDL-C concentrations are the cardiometabolic hallmark of a high CHD risk. But the etiology of atherosclerosis and consequent CHD is not only reliant on variations in LDL-C concentrations. Indeed, there is now undisputable evidence that other cardiometabolic risk factors, particularly those associated with the metabolic syndrome (MetS), also play a key role in determining one's risk to have CHD [12].

Although MetS is a state associated with an increased risk of CHD, it does not necessarily comprise elevated LDL-C concentrations. The typical dyslipidemic state associated with MetS includes elevated TG levels (in fasting and postprandial states), increased number of small dense LDL particles and low HDL-C concentrations [13]. Pro-inflammatory and pro-thrombotic states, insulin resistance, hypertension, vascular dysfunction, and abdominal obesity also often co-segregate in MetS [12, 13]. All of these cardiometabolic risk factors have been associated with an increased risk of CHD individually or in combination with one another. Ascribing the impact of dietary SFA on CHD to its sole effect on LDL-C concentrations is therefore likely to be an oversimplification of a much more complex problem.

The impact of dietary SFA intake on a wide spectrum of cardiometabolic risk factors other than LDL-C concentrations has been less well documented. But in general, available evidence suggests neutral effects of dietary SFA and perhaps even "beneficial" effects in some instances. In the metaanalysis by Mensink et al. [11], intake of dietary SFA has been shown to increase plasma HDL-C concentrations compared with most of the other nutrients. The inverse association between HDL-C concentrations and CHD risk is undisputable [14–16]. In that context, the HDL-C raising effect of SFA compared with dietary carbohydrates appears as paradoxical when considering the recommendations to lower SFA intake. However, it is unclear if this increase in HDL-C counteracts the LDL-raising effects of SFA compared with dietary CHO. The research that has documented the effect of having very high HDL-C levels through CETP inhibition on CHD risk has also been deceiving [17, 18], raising the possibility that HDL-C may not be such an important risk factor after all. Nevertheless, the neutral effect of dietary SFA on the Chol/HDL-C ratio, a powerful lipid risk factor for CHD [14], implies neutral effects in terms of CHD risk.

The impact of dietary SFA on TG concentrations is variable, depending on the comparator nutrient. Specifically, SFA tend to increase TG compared with polyunsaturated fat but tend to reduce TG compared with carbohydrates [11]. Accumulating evidence suggests that serum apoB concentration may be superior to LDL-C in predicting the risk of CHD [19]. The concentration of apoB reflects the number of atherogenic particles in the blood (not just LDL but also VLDL) because there is only one apoB molecule per lipoprotein [20]. Data from Mensink et al. suggested that replacing dietary carbohydrates by SFA had no significant impact on apoB concentrations [11]. Chiu et al. [21] have recently shown that a high-fat DASH diet (14% of calories as SFA from dairy) resulted in significantly lower plasma triglycerides, large and medium VLDL concentrations, and significantly larger LDL particle size compared with a low-fat DASH diet (8% of calories as SFA). There were no differences between diets in LDL-C, apoB and HDL-C concentrations.

Dietary SFA come from a variety of sources, not just one single food. Cheese is one of the most important sources of dietary SFA in the US diet, followed by beef, milk, and other fats and oils [22]. Butter and margarine come in seventh place on that list. This is an important consideration because individuals do not consume SFA alone, but rather as part of complex foods that also contribute other nutrients. There is emerging evidence that the foods from which SFA are derived may influence their impact on cardiometabolic risk. In a meta-analysis of five RCTs, Goede et al. [23] have summarized the effect of hard cheese vs. butter consumption on blood lipids and lipoproteins in healthy adults. Despite similar P/S ratios between the two foods, consumption of hard cheese led to significantly lower LDL-C and HDL-C concentrations (-6.5% and -3.9%, respectively) when compared with consumption of butter. Neither cheese nor butter was shown to have an impact on TG concentrations [23]. As expected, consumption of tofu or reduced fat cheese led to lower LDL-C levels compared with regular fat cheese due its lower content in SFA. This, of course, does not imply that SFA are a safe and sound nutrient in terms of cardiovascular health. It does imply, however, that we need in the future to be mindful of the complex interaction among nutrients and how these interactions may influence their impact on health. In that regard, focusing dietary guidelines solely on nutrients, which has been the case until recently, certainly does not address these complex interactions. The most recent 2015 DGAC Guidelines do put more focus on whole foods and dietary patterns [24].

It is beyond the scope of this chapter to provide an extensive review of the association between dietary SFA and cardiometabolic health. Nevertheless, research so far seems to be pointing towards a relatively neutral effect of SFA on many cardiometabolic risk factors [25], while its LDL-C raising effects may be attenuated depending on dietary sources [23]. This information is important because a large trial on SFA (vs. any other nutrient) is very unlikely, and future guidelines will need to be informed based on clinical data from surrogate markers, while also weighing the importance of changes in LDL-C and other cardiometabolic risk features. In that regard, the Institute of Medicine (IOM) has expressed concerns regarding the extrapolation of epidemiological data and data from RCTs of statins to nutrition research. While the lowering of LDL-C with statin certainly represents a

valid surrogate of clinical outcomes, IOM stated that that the effects of food on LDL-C should not be used alone to predict CHD risk change [26]. It is currently only speculated that a diet-induced change in plasma LDL-C will yield cardiovascular benefit similar to those yielded by statin therapy. Although probably highly disputed, the evidence from the Minnesota Coronary Experiment do provide an example where replacement of SFA with a vegetable oil rich in linoleic acid, which did reduce serum cholesterol levels, did not in turn yield evident cardiovascular benefit [27].

Evidence from Epidemiology and Large Clinical Trials

Epidemiological research is informative as it provides further insight into the relationship between nutrients, foods, and complex dietary patterns and clinical outcomes *per se*. This strength also becomes its greatest weakness. Indeed, despite sophisticated mathematical modeling and adjustment for the largest possible array of concurrent risk factors, epidemiological research only reflects associations between a risk factor and a disease outcome [28]. The nature of nutrition makes this particularly difficult, because as indicated above, individuals consume whole foods, not nutrients, as part of even more complex dietary patterns. This complexity is furthered by the fact that epidemiological studies are for the most part based on self-reports of dietary intake, which can be on its own a significant source of bias and uncertainty [28]. This context makes it particularly hard to dissect out the association between one single nutrient, such as SFA, and the risk of CHD. This also partly explains why results from epidemiological research on SFA have been so inconsistent and controversial. The next paragraphs summarize some of this research over the last 10 years or so.

The meta-analysis of epidemiological cohort studies published in 2010 by Siri-Tarino et al. [29] has been particularly influential in stimulating the debate on SFA and cardiovascular health. It was based on data from more than 340,000 individuals followed up over a period ranging from 5 to 23 years and concluded that higher intakes of dietary SFA were not associated with an increased risk of CHD, stroke, or CVD. The meta-analysis of prospective cohort studies by Chowdhury et al. [30] came to similar conclusions and provided further evidence on this "apparent" neutral association between dietary SFA and CHD risk. Indeed, using data from studies in which fatty acid levels in blood had been reported, they showed again a null association between total SFA concentrations and CHD risk. It should be stressed that SFA concentrations in the plasma or in red blood cells are not an accurate indicator of dietary intake because concentrations are confounded by endogenous synthesis of SFA, for which dietary carbohydrates are a significant determinant. Interestingly, however, plasma concentrations of the 17:0 fatty acid, which is found primarily in dairy fat and therefore a marker of dairy fat intake, showed inverse association with CHD risk [30]. This was further evidence that specific SFA may show different associations with CHD risk depending on food source. Accordingly, analyses of data from the MESA cohort study also suggested inverse associations between SFA from dairy specifically and risk of CVD in a population of approximately 5200 individuals followed over a period of 10 years [31]. SFA from meat showed a nonsignificant trend towards an increased risk of CVD in this population.

Of course, the meta-analyses on this topic can be criticized for a number of considerations, including for over-interpreting the data and for having excluded important studies on the topic [32]. Nevertheless, data from large interventions studies in which SFA were replaced by other fats, mostly n6-PUFA, are also at odds. In a meta-analysis of eight randomized clinical trials (RTC), Mozaffarian et al. have shown that a 5% isocaloric replacement of SFA by PUFA significantly reduced the incidence of CHD events by 19% but not of all-cause mortality [33]. The majority of these RCTs were conducted before 1980 and there have been criticisms that the cardiovascular benefits of substituting SFA for n6-PUFA was overestimated to some extent because of missing data in the analysis and due to the fact that *trans* fat consumption was also reduced in the intervention groups in some of those studies [34]. More recent meta-analyses on this topic provided conflicting results. For example, Ahmadi-Abhari et al. [35] summarized the association between dietary fatty acids from self-reported records as well as in blood and risk of CHD based on observational studies as well as RCTs of fatty acid supplementation [35]. Consistent with previous observational studies, they have shown no significant association between dietary intake of SFA and risk of CHD based on data from 20 studies comprising 283,963 subjects. Data also revealed no association between total SFA concentration in blood and CHD risk (8 studies; 15,590 subjects). More recently, Ramsden et al. [27] have reassessed the diet-heart hypothesis by amalgamating retrieved data from the Minnesota Coronary Experiment and data from RCTs that lowered serum cholesterol by providing vegetable oil rich in linoleic acid in place of SFA, without confounding by concomitant interventions. The meta-analysis, which comprised five RCTs including 10,808 participants, showed no benefit of replacing SFA by n6-PUFA (mostly linoleic acid) on total mortality and on mortality from CHD, despite reductions in blood cholesterol by 6–13% [27]. This study has just been published and will most likely be closely scrutinized. In a Cochrane-type review of the RCTs on fat reduction and fat modification and their impact on CVD-related outcomes, Hopper et al. [36] have shown that reducing SFA intake by reducing and/ or modifying dietary fat was associated with a significant reduction in the risk of CVD, based on studies with 24 comparisons and 65,508 participants followed for a minimum of 6 months [36].

This brief, nonsystematic review of the literature reveals the rather inconsistent conclusions that researchers have reached while analyzing essentially the same pool of cohort and intervention trials. Methodological considerations, such as study selection criteria and also data analysis, can obviously lead to different observations and conclusions. Nevertheless, such discrepant results have generated a lot of confusion within the population as well as the scientific community. It is fair to say that the association between dietary SFA and CHD risk is not a matter a consensus.

SFA: Friend or Foe? Conclusions

This question remains plagued with some degree of controversy. On the one hand, there is limited data to indicate that SFA are in fact a "friend." Dietary SFAs may be better than trans fat - or not as bad depending on one's perspective on the topic. SFA may overall also be "better" than dietary carbohydrates [37]. However, SFAs represent an enemy nutrient if one considers LDL-C lowering as the main target in preventive nutrition. In that context, replacement with unsaturated fat is certainly to be privileged. Data from clinical outcome studies have been inconclusive, but then again this depends on one's perspective on this topic. On the other hand, if one considers that the etiology of CHD involves many other risk factors than just raised LDL-C, then SFA may be considered as being a relatively neutral nutrient (neither friend nor foe). The picture gets blurrier because as indicated above SFA are not consumed in isolation. Its impact on cardiovascular and cardiometabolic health therefore needs to be contextualized as part of more complex food patterns. For example, a recent systematic review by our group has shown no evidence of harmful effects of high-fat dairy consumption on cardiovascularrelated clinical outcomes [38]. Cheese intake, which does contribute significantly to SFA intake in the US population, even showed inverse association with risk of stroke and T2D. This apparent paradox emphasizes the importance to consider the impact of foods rather than individual nutrients on health, as in the most recent 2015 DGAC [39].

Will we ever reach consensus regarding the impact of dietary SFA on cardiovascular health? That is doubtful. Many researchers have strong opinions in both camps of the debate. Nevertheless, more research will be informative in solving some of this controversy. For example, we currently do not have data on the differential association of the low vs. regular fat versions of a dairy product, say cheese, with the risk of CVD [38]. This is important because consumption of low-fat dairy is advocated in current dietary guidelines. This question and several others currently go unanswered. In the

meantime, dietary guidelines that focus on whole foods and dietary patterns, without overly emphasizing the importance of SFA in the diet, seem entirely reasonable and appropriate.

Disclosures The author is Chair of Nutrition at Laval University. This Chair is supported by unrestricted endowments from Royal Bank of Canada, Pfizer, and Provigo/Loblaws. He has received funding in the last 5 years for his research from the CIHR, NSERC, Agriculture and Agrifood Canada, the Canola Council of Canada, Dairy Farmers of Canada (DFC), Dairy Research Institute (DRI), Atrium Innovations, the Danone Institute, and Merck Frosst. He has received speaker honoraria over the last 5 years from DFC and DRI. He is Chair of the Expert Scientific Advisory Panel of DFC and was a member of the ad hoc committee on saturated fat of Heart and Stroke Foundation of Canada.

References

- 1. Hunt CL. Food for young children. Washington, DC: US Department of Agriculture, Farmer's Bulletin;1916. p. 717.
- Gifford KD. Dietary fats, eating guides, and public policy: history, critique, and recommendations. Am J Med. 2002;113:89–106.
- 3. Mansfield HC. A short history of OPA. Washington, DC: Office of Temporary Controls OoPA; 1947. p. 332.
- Welsh SO, Davis C, Shaw A. 1993. USDA's Food Guide: Background and Development. United States Department of Agriculture. Human Nutrition Information Service. Miscellaneous Publication No. 1514. Hyattsville, MD, p 1–38.
- 5. Keys A. Diet and the epidemiology of coronary heart disease. JAMA. 1957;164:1912-9.
- Aravanis C, Corcondilas A, Dontas AS, et al. Coronary heart disease in seven countries. IX. The Greek islands of Crete and Corfu. Circulation. 1970;41:188–100.
- 7. Yerushalmy J, Hilleboe HE. Fat in the diet and mortality from heart disease; a methodologic note. NY State J Med. 1957;57:2343–54.
- Committee USS. Dietary goals for the United States. US Senate Select Committee on Nutrition and Human Needs. Washington, DC; 1977.
- 9. Committee USS. Dietary goals for the United States, 2nd ed. US Senate Select Committee on Nutrition and Human Needs. Washington, DC; 1977.
- Hite AH, Feinman RD, Guzman GE, et al. In the face of contradictory evidence: report of the Dietary Guidelines for Americans Committee. Nutrition. 2010;26:915–24.
- Mensink RP, Zock PL, Kester ADM, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. Am J Clin Nutr. 2003;77:1146–55.
- 12. Despres JP, Lemieux I. Abdominal obesity and metabolic syndrome. Nature. 2006;444:881-7.
- 13. Desroches S, Lamarche B. The evolving definitions and increasing prevalence of the metabolic syndrome. Appl Physiol Nutr Metab. 2007;32:23–32.
- 14. Lamarche B, Moorjani S, Lupien PJ, et al. Apolipoprotein A-I and B levels and the risk of ischemic heart disease during a five-year follow-up of men in the Quebec cardiovascular study. Circulation. 1996;94:273–8.
- Lamarche B, Despres JP, Moorjani S, et al. Triglycerides and HDL-cholesterol as risk factors for ischemic heart disease. Results from the Quebec cardiovascular study. Atherosclerosis. 1996;119:235–45.
- Gordon T, Castelli WP, Hjortland MC, et al. High density lipoprotein as a protective factor against coronary heart disease: the Framingham study. Am J Med. 1977;62:707–14.
- 17. Barter PJ, Caulfield M, Eriksson M, et al. Effects of torcetrapib in patients at high risk for coronary events. N Engl J Med. 2007;357:2109–22.
- Schwartz GG, Olsson AG, Abt M, et al. Effects of dalcetrapib in patients with a recent acute coronary syndrome. N Engl J Med. 2012;367:2089–99.
- Sniderman AD, Furberg CD, Keech A, et al. Apoproteins versus lipids as indices of coronary risk and as targets for statin therapy: analysis of the evidence. Lancet. 2003;361:777–80.
- 20. Sniderman AD, Shapiro S, Marpole D, et al. Association of coronary atherosclerosis with hyperapobetalipoproteinemia [increased protein but normal cholesterol levels in human low density (beta) lipoproteins]. Proc Natl Acad Sci U S A. 1980;77:604–8.
- Chiu S, Bergeron N, Williams PT, et al. Comparison of the DASH (Dietary Approaches to Stop Hypertension) diet and a higher-fat DASH diet on blood pressure and lipids and lipoproteins: a randomized controlled trial. Am J Clin Nutr. 2016;103:341–7.

- 22. Huth PJ, Fulgoni VL, Keast DR, et al. Major food sources of calories, added sugars, and saturated fat and their contribution to essential nutrient intakes in the U.S. diet: data from the National Health and Nutrition Examination Survey (2003–2006). Nutr J. 2013;12:116.
- de Goede J, Geleijnse JM, Ding EL, Soedamah-Muthu SS. Effect of cheese consumption on blood lipids: a systematic review and meta-analysis of randomized controlled trials. Nutr Rev. 2015;73:259–75.
- Millen BE, Abrams S, Adams-Campbell L, et al. The 2015 Dietary Guidelines Advisory Committee Scientific Report: development and major conclusions. Adv Nutr. 2016;7:438–44.
- Drouin-Chartier JP, Côté JA, Labonte ME, et al. Comprehensive, evidence-based review of the impact of dairy products and dairy fat on cardiometabolic risk factors. Adv Nutr. 2016. In press 2016.
- 26. (Medicine) CoQoBaSEiCDIo. Evaluation of biomarkers and surrogate endpoints in chronic disease. National Academy of Sciences; 2010.
- Ramsden CE, Zamora D, Majchrzak-Hong S, et al. Re-evaluation of the traditional diet-heart hypothesis: analysis of recovered data from Minnesota Coronary Experiment (1968–73). BMJ. 2016;353:i1246.
- Maki KC, Slavin JL, Rains TM, Kris-Etherton PM. Limitations of observational evidence: implications for evidence-based dietary recommendations. Adv Nutr. 2014;5:7–15.
- 29. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Meta-analysis of prospective cohort studies evaluating the association of saturated fat with cardiovascular disease. Am J Clin Nutr. 2010;91:535–46.
- Chowdhury R, Warnakula S, Kunutsor S, et al. Association of dietary, circulating, and supplement fatty acids with coronary risk: a systematic review and meta-analysis. Ann Intern Med. 2014;160:398–406.
- 31. de Oliveira Otto MC, Mozaffarian D, Kromhout D, et al. Dietary intake of saturated fat by food source and incident cardiovascular disease: the Multi-Ethnic Study of Atherosclerosis. Am J Clin Nutr. 2012;96:397–404.
- 32. Scarborough P, Rayner M, van Dis I, Norum K. Meta-analysis of effect of saturated fat intake on cardiovascular disease: overadjustment obscures true associations. Am J Clin Nutr. 2010;92:458–9. author reply 459.
- Mozaffarian D, Micha R, Wallace S. Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: a systematic review and meta-analysis of randomized controlled trials. PLoS Med. 2010;7:e1000252.
- 34. Ramsden CE, Zamora D, Leelarthaepin B, et al. Use of dietary linoleic acid for secondary prevention of coronary heart disease and death: evaluation of recovered data from the Sydney Diet Heart Study and updated meta-analysis. BMJ. 2013;346:e8707.
- 35. Ahmadi-Abhari S, Luben RN, Powell N, et al. Dietary intake of carbohydrates and risk of type 2 diabetes: the European Prospective Investigation into Cancer-Norfolk study. Br J Nutr. 2014;111:342–52.
- Hooper L, Martin N, Abdelhamid A, Davey Smith G. Reduction in saturated fat intake for cardiovascular disease. Cochrane Database Syst Rev. 2015;(6):CD011737.
- Siri-Tarino PW, Chiu S, Bergeron N, Krauss RM. Saturated fats versus polyunsaturated fats versus carbohydrates for cardiovascular disease prevention and treatment. Annu Rev Nutr. 2015;35:517–43.
- Drouin-Chartier JP, Brassard D, Tessier-Grenier M, et al. Systematic review of the association between dairy product consumption and risk of cardiovascular-related clinical outcomes. Adv Nutr. 2016. (In press 2016).
- Hauk L. DGAC makes food-based recommendations in the 2015–2020 Dietary Guidelines for Americans. Am Fam Physician. 2016;93:525.

Part VII Nutrition and Public Policy Issues

Chapter 22 Public Policy and Environmental Supports for Healthy Eating

Zaida Cordero-MacIntyre, Hildemar Dos Santos, and Christy Mota

Key Points

- The Healthy People 2010 goals for the USA were not met; in order to meet the Healthy People 2020 goals, the built environment can make the difference for this accomplishment. Built environment means the existence and affordability of health foods in a neighborhood.
- Due to the residential segregation in the United States, it is likely that there is a difference in food choices as a result of the environment. Neighborhoods that have large-chain food stores or medium-sized non-corporate-owned food stores have only 21 % obesity level, while the neighborhoods that have no access have 32–40 % obesity level.
- It has been recommended by the state and local governments to require nutrition and calorie labels on restaurant menus; the 2010 Health Care Reform Law requires chain restaurants to label their menus.
- Foods high in calories such as fats, sugar, and sugary drinks should be taxed, and the tax money should be used for programs to promote good nutrition and to subsidize healthy foods to low-income populations.
- ChooseMyPlate is an online dietary assessment tool that provides information on one's diet serving portions, nutritional messages, and links to nutrient information.

Loma Linda University School of Public Health, Nichol Hall, Loma Linda, CA 92350, USA e-mail: zcordero-macintyre@llu.edu; hdossantos@llu.edu; cm.mota74@gmail.com

The national Healthy People 2020 Nutrition and Weight Status objectives are to reduce the proportion of children and adolescents aged 2-9 who are considered obese from 16.1 to 1.6%. The goal is to promote health and reduce chronic disease risk through the consumption of healthful diets and to promote health for all through a healthy environment.

Z. Cordero-MacIntyre, PhD, PharmD, MPH, REHS, MS, RD • H. Dos Santos, MD, DrPH, CNS, CHES, EPC () C. Mota, MPH

Introduction

According to the Centers for Disease Control and Prevention (CDC), more than one-third, 78.6 million, of US adults are obese; obesity is common, serious, and costly [7, 10]. In 2014, the mean total fat intake for men and women were larger than the mean protein intake, that is, 33.0% kcal of fat versus 16% kcal of protein intake for men and 32.9% kcal of fat versus 15.5% kcal of protein intake for women [8, 9]. Approximately 17% or 12.7 million of children and adolescents aged 2–19 years are obese [8, 9]. Most young US people do not meet the recommendations for eating 2.5 cups to 6.5 cups of fruits and vegetables each day, but rather eat more than the recommended maximum daily intake of sodium, 1,500–2,300 mg each day. Currently, research suggests childhood dietary habits are influenced by various environmental factors that include the availability of fast foods and soft drinks, availability of soda and unhealthy foods on school campuses, and limited access to healthy foods compared to the ready availability of unhealthy foods [41].

Although most Americans know the relationship between diet and health, due to environmental barriers, low-income communities have constraints (or difficulties) to maintain a healthy and affordable diet [16]. Consequently, public policy and environmental behavior change, and not just individual behavior, have become a major focus to improve healthy eating. To increase a healthy nutrient diet, environmental and public policy favor an increase in fruit and vegetable food security, provide more access, and target healthcare and worksite settings.

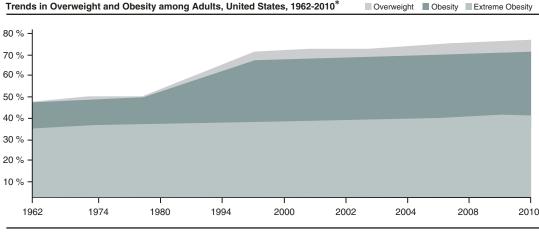
Overview of Healthy Eating Trends

Morbidity and Obesity

Twenty-nine million Americans have diabetes, and the number is significantly rising particularly among Hispanics. Currently, African-American and Hispanic adults are 1.3–1.9 times more likely to have diabetes than non-Hispanic white adults [31]. Currently, childhood obesity is at epidemic levels in the United States. Nearly a third of youths are overweight or obese [34]. In the past 20 years, there has been twice as much increase in childhood obesity. African-American and Hispanic children are twice as likely to be obese than non-Hispanic white and non-Hispanic Asian children [27].

The current evidence shows that the traditional ways to prevent and treat obesity, which focus on behavioral change, have not been successful in controlling the epidemic of obesity. In 1980, the US Public Services proposed that by improving nutrition and physical fitness, there would be a reduced rate of overweight among men and women by 10 and 17%, respectively, in 1990. The goal made in 1990 for the Healthy People 2000 was no more than 20% of overweight and obesity for adults. In 2000, the aim for 2010 was to increase healthy weight among adults to 60%. None of these goals were reached according to the image below. By 2010, 70% of the population was overweight and obese (Fig. 22.1) [35].

There is good news, however. Growing evidence has shown that one factor may give good results, the built environment. Increasing the opportunities for exercise in the community and increasing the availability of fresh fruits and vegetables could have some impact. Also, making fast food availability more difficult or more expensive could also be another promising strategy.



*Data for 1960-1980 are for adults ages 20 to 74: data for 1988-2010 are for adults age 20 and older.

Overweight

Fig. 22.1 Trends in overweight and obesity among adults in the United States, 1962-2010 (Image: NIDDK (National Institute of Diabetes and Digestive and Kidney Diseases))

Availability and Affordability of Healthy Foods

Healthy eating recommendations, such as substituting "lite" bread and diet soft drinks, made by associations such as the American Diabetes Association, do not mean that healthier eating choices are available to all neighborhoods. There is evidence that shows that grocery stores in low-income and non-white neighborhoods have limited supply of healthy eating choices. The lack of availability of healthy foods makes it challenging for clinicians to prevent and control chronic diseases such as diabetes, that requires an increased intake of healthier foods, fruits, and vegetables [28]. The affordability of these foods is also a concern as refined grains, sugar, and fats are more affordable than the recommended diets based on lean meats, fish, and fresh vegetables and fruits [16].

Furthermore, grocery stores in predominantly black and lower-income population place the food behind glass storages making it harder to be accessed by the costumers. As a healthcare professional, it is important to understand that environment support is necessary for the change in dietary practices to occur. Statistics show that obesity and overweight prevalence is higher in lower-income neighborhoods [45]. Similarly, the growing number of fast food restaurants is alarming. McDonald's goal is that every person in the United States should be at a 4-min distance to one of its restaurants [4].

Health Behavior in Healthy Eating

A study conducted in 2007 found that "the health concern of consuming too many calories from food has a greater effect on healthy eating attitudes than did health concern of developing diseases" [42]. This study showed that food choice motives are not associated with healthy eating attitude. It also indicates that health concern affects the food choice motives of mode and familiarity. It is important that healthcare professionals take a practical approach in encouraging healthy eating by focusing on the importance of health and calorie control instead of weight control. The eating behavior of an adolescent is important in the maintenance of their health and in their physical development, which is why the intake of nutrients needs to be higher than in any other growth period. Similarly, adolescence is a time where behavior patterns are developed and consequently have an impact on their behaviors in the future, as there is an increase of independence and autonomy. Studies show that the eating practices one develops as an adolescent have long-term effects on health status in adulthood. It is critical as a healthcare professional that one incorporates healthy eating behaviors into the daily routines of adolescence to reduce the risk of chronic diseases such as heart disease, diabetes, and particularly obesity in adulthood. Although there are standards and guidelines set out by nutrition professionals of a diet low in saturated fat and cholesterol and high in of fruits, vegetables, and whole grains, adolescents are having a nutritional problem due to their inconsistent eating behavior and unhealthy eating.

Studies show that adolescents have a lack of concern for healthy eating patterns, which is attributed to their perception of healthy eating recommendations and the health implications. In 2001, a study was done to determine the perception of healthy and unhealthy foods, eating behaviors, and the importance of healthy eating. Most adolescents associated unhealthy eating with their peers, but healthy foods were associated with eating at home with their families. However, despite their awareness of healthy eating, adolescents are not consistently following nutrition recommendations, and this may be due to barriers that prevent them from developing healthy eating practices. It is important to understand that peer norms play a role in the healthy eating choices of adolescents. For instance, a study in California revealed that 85% of teen girls had a health concern for weight control, but only 8.5% had a concern about healthy eating. This data was then compared to the *National Health Interview Survey* and *Teenage Attitudes and Practice Survey*, which showed that weight was the second most important concern, whereas healthy eating was ranked last as a concern [13].

These findings suggest that the healthy eating messages based on the *Dietary Guidelines for Americans* are reaching the adolescents, but interventions are needed to assist adolescents with the translation of this knowledge into healthy behaviors. Interventions should help make healthy eating easy for youths to apply and explain the consequences of unhealthy eating in terms that they value, stressing meaningful short-term benefits [13]. "Eating behavior is carried out with little regard to intention or self-regulation. However, deliberating over a menu seems to be an occasion when cognition plays a role and thus when rational food decisions might be made" [39].

Disparities/Inequities in Healthy Eating

In 2005, the *Dietary Guidelines for Americans* took note of the low intake of fresh fruits and vegetables, low-fat dairy products, and whole-grain food in the US population. It noted that race and income might be contributing factors to the level of healthy food intake. Due to residential segregation in the United States, it is likely that there is a difference in food choices as a result of their environment. For example, the type and amount of food stores in a neighborhood vary by race and income. A study conducted in 2008 found that there were more supermarkets that carried healthy food in predominantly non-Hispanic white and high-income neighborhoods compared to non-Hispanic black lowincome neighborhoods. There are disparities in obesity rates existing among ethnic groups. Non-Hispanic black, Hispanic, and Native American children and adolescents have higher rates of diabetes and obesity than do non-Hispanic white children and adolescents [28].

According to a recent study in 2010, there is no statistically significant difference in obesity prevalence between 2001 and 2007. However, the income disparity in obesity prevalence more than doubled between 2001 and 2007 when adolescents with family incomes below the poverty line were compared with those with family incomes at or above 300% of the federal poverty line. It also found that male adolescent obesity rates were higher than female rates across the income groups, especially among Hispanic adolescents in the United States [3]. Currently, the increasing disparity in obesity prevalence by Socioeconomic Status (SES) places lower-income adolescents at a disproportionate risk for adulthood obesity as well as related conditions such as diabetes, hypertension, heart disease, stroke, and cancer. It is important to point out that physical inactivity, sweetened beverages, and skipping breakfast are targets of intervention to reduce the disparities in obesity prevalence.

Breaking down the barrier of accessing resources for those who have a low SES, particularly African-Americans and Latinos, would require environmental and public policy support. Without which, eating healthier will remain a challenge, ultimately preventing the reduction of childhood obesity [3]. Nonetheless, studies show that there are disparities in neighborhood access to healthy foods based on income, race, ethnicity, and urbanization [33].

Environmental Factors

Food environment is affected by many different factors, including the price of food, food distribution channels, the awareness and knowledge of store managers, and policies concerning the location of various types of stores [45]. The physical environment has an effect on the individual weight status by impacting both energy intake and energy expenditure. The two most important environmental influences are the availability of food retail and food establishment [21].

Availability of Full-Service Grocery Stores

A study conducted in East Harlem, *Barriers to Buying Healthy Foods for People with Diabetes: Evidence of Environmental Disparities*, explored the difference in availability and cost of healthy food in low-income, minority neighborhoods and affluent all-white neighborhood in New York [28]. The results showed that although East Harlem had twice as many food stores per capita than the Upper East Side, only 18% of the food stores carried recommended food items such as fruits, vegetables, and whole grains compared to 58% of the food stores on the Upper East Side of New York [28].

In areas that have a smaller population, there are food stores that are conveniently located in the neighborhood. In neighborhoods that have small grocery stores, the average amount of shelf for fruits and vegetables is smaller than the shelf space for snack food. Furthermore, the quality of the fruits and vegetables tends to be poor in food stores in lower-income areas. Consequently, it is difficult to eat healthier if there are not many quality food options. Research has shown that small grocery stores are prevalent in poorer urban areas, making access to healthy food more difficult [21].

There was a revision of studies and abstracts by Papas et al. in 2007 about the built environment and obesity [38]. About 1,500 articles were analyzed, and 84% of them reported a significant positive association between some aspects of the built environment and obesity. However, most of the studies were cross-sectional, there was inconsistency in the methods to assess the built environment, and the measures for behaviors were not used comprehensively; one study will use diet only and another exercise only. "While there is a strong intuitive appeal to the notion that the built environment must be contributing to the obesity epidemic, existing scientific evidence does not tell a clear story" [18]. There are individual studies that clearly show the influence of the built environment, but others do not, and also, most of the studies have different designs. Therefore, it is not easy to conclude that the built environment really affects behavior. Suggestions were made to improve the research design of studies mostly by having a better understanding of environmental metrics, more longitudinal studies, more interdisciplinary cooperation, and a better understanding of places [18].

Food Purchasing Options

Retail Food Stores

Supermarkets tend to have the most variety of high-quality food at the lowest cost, whereas convenience stores typically sell high-calorie, prepared food and are limited in fresh fruits and vegetables which are at a higher cost. Studies have shown that having the option of a supermarket where you can buy high-quality, low-cost fresh produce is associated with increased daily intake of healthy food. It has been shown that purchasing food at a convenience store is associated with lower intake of fresh fruits and vegetables. Neighborhoods that have large-chain food stores or medium-sized non-corporate-owned food stores have only a 21 % obesity level compared to neighborhoods that have no access at 32-40 % obesity level. Low-income and rural residents often have limited access to supermarkets or chain grocery stores that provide healthy food options. Rural and farm areas have 14 % fewer chain supermarkets compared to urban areas [33].

Similarly, some findings showed that a greater number of convenience stores were located in secondary schools in lower-income neighborhoods.

Restaurants

Currently, the majority of our meals and snacks are purchased at restaurants, which tend to serve food high in calorie and low in quality. Food that is eaten at restaurants typically does not provide nutritional information to encourage healthy eating choices. Studies show that frequently eating outside the home leads to a higher intake of fat, sodium, and sugary beverages and lower intake of fruits and vegetables. However, full-service restaurants offer lower food prices, encourage higher food consumption, yet have shown to be associated with fruit and vegetable intake. Full-service restaurants in affluent neighborhoods have more healthy options on the menu. For example, a study conducted in South Los Angeles showed that 40 % of restaurants in high-income areas provide at least five healthy food choices. In fact, there was a study that showed proximity to fast food restaurants increases the likelihood of gaining more than 20 kg during pregnancy [21].

Frequent consumption of fast foods is related to an increased intake of calories, mostly from fat and sugary drinks, and inversely related to the intake of fruits and vegetables. Even though there is no clear connection with weight gain, the relationship between fast food consumption and the risk factors for obesity is real [19].

Several mechanisms have been proposed to control the influence of fast food restaurants. One of them was to provide nutrition labels. This was implemented by the *Patient Protection and Affordable Care Act* in 2010, whereby all chain restaurants having 20 or more locations must provide nutrition information of their foods. Whether the *Patient Protection and Affordable Care Act* method really produced less calorie consumption was not clear. However, Swartz et al. [43] made a review studies and concluded that nutrition labeling has not been successful in reducing caloric intake.

Another method would be to develop a restaurant labeling similar to what is done with the ABC system used to check for food-borne diseases. As Americans consume 41% of their food outside of their homes, and both adults and children consume one-third of their calories from eating out [48], it is imperative that restaurants, especially fast foods, be controlled by the healthy quality of their foods.

The *Health Eating Index* would be a good way to promote this. Today, most of the fast food restaurants claim that they are improving their menus and having much more healthy options. However, a

study by Hearst et al [25] concluded that after 14 years of follow-up, the fast food restaurant menus had little change in them. The *Health Eating Score* of the restaurants started with 45 in 1997–1998 and increased to 48 in 2009–2010, based on a total score of 100, representing the healthiest score. They checked eight fast food restaurants and figured out that there was an increase in the meat and beans and a decrease in saturated fats and sugar. The authors concluded that the improvement was small and that further improvements are needed.

The *Health Eating Index 2005* was calculated for each restaurant menu and is based on the *Dietary Guidelines of America*. They found the score of 48 being lower than the average American score for individuals using the index, which is a score of 55; this was considered not optimal by the United States Department of Agriculture (USDA). We propose that the *Health Eating Index* be applied to restaurants across the country and publicized in their front window, similarly to what is being done by the *ABC Food Safety System*.

Just to illustrate the issue, there was a study that evaluated children's hospitals with fast food restaurants compared to hospitals without those restaurants. The researchers confirmed that the clients from the fast food restaurants would more likely purchase fast food from the hospital, thinking that the fast food restaurant supporting the hospital and that food from those restaurants was very healthy. This is the impact that health professionals and institutions have in the community when they associate their practice with services that do not provide healthy options. There are those who are waking up, however, such as the Cleveland Clinic that closed down its McDonald's restaurant when its lease contract ended. On September 18, 2016, the McDonald's restaurant at the Cleveland Clinic, the world's most renowned heart clinic, was closed as the restaurant was not allowed to renew the lease contract [2].

There are still 26 hospitals in the country that have fast food restaurants in their building, but the trend is decreasing. "This has to happen. We know about the causes, effects, and costs of overweight and obesity. So it is indefensible for hospitals to continue to provide space for a restaurant chain that serves food linked to the health problems clinicians are treating just a few steps away. It's not just a mixed message. It's the wrong message" (John Commins, Senior Editor at HealthLeaders Media) [12].

List of Other Hospitals that Ousted Fast Food Restaurants in Recent Years

- Children's Hospital of Philadelphia, Philadelphia, PA
- Lurie Children's Hospital of Chicago, Chicago, IL
- · Kosair Children's Hospital, Louisville, KY
- · Children's Mercy Hospital, Kansas City, MO
- · Thomas Medical Center, Kansas City, MO
- Parkland Hospital, Dallas, TX
- Mount Sinai Hospital, New York, NY
- University of California San Francisco, San Francisco, CA
- Vanderbilt Medical Center, Nashville, TN [6, 17, 22, 26]

Food Vending

Studies show that the number of vending machines present at school is negatively associated with healthy eating as "preferred food" like sweetened drinks and high-fat foods are being provided to the students. With more machines in schools, the low-nutrient snack selections and an easier opportunity to purchase such items become available for the students [32]. Similarly, this relationship is noticeable with mobile food vendors, though they sell a much smaller range of food options.

The mobility of the vendor means that it can reach areas that otherwise would not have access to food stores and establishments. However, mobile vendors are more prevalent in rural and low-income communities, especially in large Latino immigrant-populated neighborhoods. Rather than selling

nutritious food, more high-fat, energy-dense foods are sold, contributing to the low intake of five servings of fruits and vegetables. Historically, food vendor catered the poor and foreign immigrants by offering affordable prices, while indoor retail stores catered the middle- and upper-class customers. Since the 1800s, mobile vendors face challenges of competing with local business and being prohibited to sell in upscale neighborhoods. Currently, efforts are being made to change permits, fees, and regulations to encourage "healthy food" vendors to meet the nutritional needs of the residents [44].

Healthy Food Policy

School

Studies show that there is an association between school "à la carte" programs and the average daily consumption of fruits, vegetables, and saturated fats. Those who do not have an à la carte program tend to eat more closely to the dietary recommendations, and thus, the quality and variety of food in à la carte programs have been in need of attention. Schools have become more dependent on the revenue of "à la carte" sales, and students enjoy the tastiness and convenience of fast food, making it challenging to improve healthy eating. School food programs are being encouraged to provide more fruits and vegetables [32]. In 2005, the *California Endowment's Healthy Eating, Active Communities Program* was established to prevent childhood obesity and diabetes in low-income communities. There was a set of policies proposed that targeted improving the availability and quality of healthy foods.

The following are some of the policies adopted as a result of the *California Endowment's Healthy Eating, Active Communities Program* [28]:

- 1. Setting standards for quality of nutrition and physical education on campus
- 2. Regulations on food and beverage marketing and advertisement
- 3. Quality of food offered at fundraisers and classroom parties
- 4. Eliminating sports drinks
- 5. Eliminating sale of trans-fat foods

Currently, policy makers and public officials are focused on sustainable environmental improvements despite the fact that the current economic state has limited the number of resources in public school's access to improve healthy eating. However, there is an increasing demand for effective environmental and policy strategies that focused on mobilizing more efforts in community-based intervention strategies to improve healthy eating.

Moreover, organizational policy, such as policy changes in a school or work environment, has been shown effective through the *Students Today Achieving Results for Tomorrow* program (Sacramento Start). The program targeted high-risk populations and low-income and ethnic minority children to increase fruit and vegetable intake during snack time. The snacks contained (1) a dairy product, (2) a serving of meat, (3) a serving of vegetables or fruits or vegetable or fruit juice, and (4) a serving of whole grain. By using what was called "five-a-day" guidelines, they were able to increase the intake of five daily servings of fruits and vegetables by 83 % [40]. This was accomplished by changing the food vendor at their school.

Geographic Availability of Full-Service Food Stories

Studies show that neighborhoods that have a density of small groceries are significantly related to obesity and body mass index of its residents in urban areas. This raises the question of whether

policies that offer incentives for food retail stores to increase the availability of healthy food or other policies that foster the creation of neighborhood stores specializing in healthy food would be expected to improve food choices and health outcomes for neighborhood residents [21]. There have been policies proposing a tax on high-fat foods at grocery stores to decrease the purchase of high-fat foods and to increase the intake of fresh fruits and vegetables (see the "Food Taxation" section).

Food Labels

It is recommended that food and beverage manufacturers limit levels of saturated fats, trans-fatty acids, free sugars, and salt in products sold. It is also recommended that they develop new products with more nutritional value and limit the amount of promotion and marketing of processed products. For instance, PepsiCo permits advertisement of its products to children of all ages although its products are high in fat, saturated fat, cholesterol, sugar, and sodium that exceeds the limits recommended by the World Health Organization. Food and beverage advertisement is encouraging the consumption of high levels of calories in soft drinks, overeating, and snacking instead of eating a full balanced meal and eating while in front of the television. Rather, there needs to be an effort in encouraging cooking and discouraging the fact that happiness does not equate to spending.

Restaurant Menus

The United States is spending nearly half of food expenditures in meals in restaurants, which tend to be higher in calories and fat. Unfortunately, the food descriptions provided on menus do not enable the consumer to make healthier food choices. It is becoming increasingly difficult to determine which food items on the menu contain the most calories, and in fact most tend to underestimate calorie content, specifically in high-calorie items. Although nearly half of the chain restaurants in the United States publicize nutrition information, customers typically do not notice where they are displayed. It has been recommended by the state and local governments to require nutrition and calorie labels on restaurant menus; the 2010 Health Care Reform Law requires chain restaurants to label their menus.

A study was conducted to see if labeling menus influenced eating choices in restaurants. The results showed that restaurant menus were associated with calorie, fat, and sodium reduction [39]. Menu labeling would permit health-based food choices in restaurants, but its absence makes such choices virtually impossible. However, menu descriptions alone are inadequate for guiding nutrient-based food decisions. People are typically biased in judging which food items on the menu are unhealthy. There is a need for healthy nutrition information together with description of menus.

It is also important to take into consideration that, for instance, a Reuben sandwich can range from 480 to 1,730 calories, 19 to 83 g of fat, and 39 to 182 g of carbohydrates. The variation comes from the differences in the side dish, portion size, and ingredients [39].

According to the *CDC State Indicator Report on Fruits and Vegetables 2009 National Action Guide*, the following are key policy recommendations [15]:

1. Promote the availability of healthier food retail in communities

Strategies and policies to improve the food environment can aid fruit and vegetable access, availability, and affordability. The indicators in the subsequent text represent key areas in which policy and environmental support can help make changes in people's community by having access to fruits and vegetables.

2. Promote the availability of healthier foods and nutrition services in schools

Schools are uniquely positioned to model and reinforce healthful eating behaviors such as increasing knowledge of and access to fruits and vegetables on the school campus and at school-

related activities. Schools have the ability to provide fruits and vegetable not only to youth but also to teachers, other school staff, parents, and community members.

3. Encourage food system support

A systems approach to food considers the many factors involved in getting fruits and vegetables from farm to the consumer including aspects of food production, processing, and distribution. Also included in a food system approach are the participants in that system, including farmers, processors, industries, workers, governments, retailers, institutional purchasers, communities, and consumers.

Food Taxation

Other strategies to curtail the consumption of fast foods and promote healthy foods include taxation. Foods high in calories such as fat, sugar, and sugary drinks should be taxed, and the money should be used for programs to promote good nutrition and to subsidize healthy foods to be affordable to low-income populations. This has a high potential to be successful as it is similar to what was done with smoking, and as a result, there was a reduced rate of smoking in the country.

"A significant increase in tobacco product taxes and prices has been demonstrated to be the single most effective and cost-effective intervention for reducing tobacco use, particularly among the young and the poor" [29]. Increasing taxes on cigarettes by 10% resulted in decreased consumption of cigarettes by 4.5% [20], and more recent studies confirmed this decrease to be between 2 and 6% [29]. The increase in smoking taxes has been applied to promote health activities to help people stop smoking and campaigns to prevent children from starting smoking. Rates of smoking that were around 33.7% in 1970 dropped to 17.8% in 2013. Cigarette prices increase because taxation has been one of the main factors implicated on dropping those rates (Fig. 22.2).

A similar increase in taxation could be developed for unhealthy foods and being directed to develop prevention and treatment programs to curtail the obesity epidemics or to subsidize the costs of healthy foods [4]. States are taxing some unhealthy foods such as California that have 7.25 % sales tax on soft drinks which brings about \$200 million per year [36]. However, most of the taxes do not go for obesity prevention.

Nestle and Jacobson [30] estimated that each of the those strategies below for taxation would generate revenues of 1 billion per year:

- A 2/3 cent tax per 12 oz. can of soft drink
- A 5% tax on new televisions and video equipment
- A \$65 tax on each new motor vehicle
- A penny tax per gallon of gasoline

There was a tentative plan of doing so with the California tax on soft drinks that the money from the tax should go to the below activities; however, the bill was passed without the tax provisions [37]:

- 50% to school districts that stop selling soft drinks on school campuses
- 25% to the state health department for programs to promote nutrition and physical activity
- 25% to hospitals, emergency and trauma care, and clinics

Six states impose special taxes or fees on soda and soft drinks, and ten states, including Connecticut, exclude certain high-fat and high-sugar edibles, junk food or soda, and other soft drinks from their sales tax exemptions for food [30]. So there is a concern and states are moving forward with

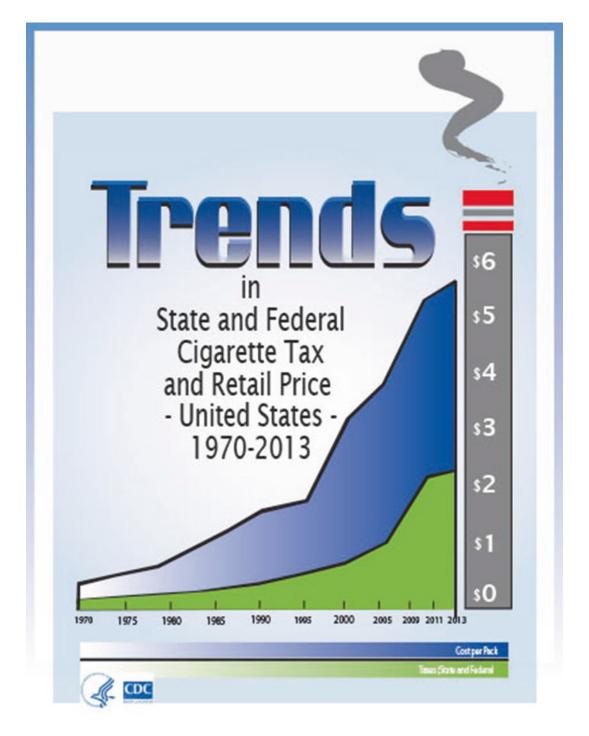


Fig. 22.2 Trends in state and federal cigarette tax and retail price – United States – 1970–2013 (Image: CDC, Economics Infographics (CDC [11]))

legislation on junk foods. What is needed is that the revenues from these taxes go to promote more preventive activities in the community. Also, food taxation should be accompanied by policies to decrease the price of healthy foods; otherwise, low economic classes will be severely affected by the general high-price foods.

Food Affordability

Food Subsidy

Foods that are subsidized directly or indirectly by the government, in general, are animal foods or foods consumed in its processed forms. The commodity crops are the foods the government describes as fundamental for consumption and, therefore, the most subsidized. This is done to maintain its prices at a level that the population can purchase that is still profitable for the producers. Commodity products in the United States are corn, wheat, soybeans, cotton, and rice. Between 1995 and 2009, the government spent 246 billion dollars subsidizing this and other commodity products (Agriculture and Health Policies in Conflict) [1].

However, if we take a closer look at these crops, we will notice that corn and soybeans are mostly produced for the livestock to produce meat and dairy products. Wheat and rice are main products consumed mostly in its processed forms, being devoid of vitamins, minerals, and fiber. Only regarding dairy products, the government spent 4.8 billion dollars between 1999 and 2009 to subsidize its production allowing producers to have lower costs in their production to maintain milk prices stable and affordable.

Special crops are mostly fruits and vegetables, and they do not get any subsidy, and even the farmers who deal with the commodity crops are forbidden to plant special crops so as not to use the subsidy money for other types of crops not approved. Having in mind the current disease crisis and the obesity epidemic in the country, we suggest that authorities should consider changing those policies to make provisions for special crops. If farmers are given subsidy, maybe the same as what they get from producing dairy products and milk, there would be a bigger production of fruits and vegetables in the country with affordable prices – prices that would be controlled by the government just like milk is (*Agriculture and Health Policies in Conflict*) [1].

Food Losses

About six billion pounds of fruits and vegetables are wasted every year in the United States because they do not reach the patterns of beautiful food, according to a report by the *Natural Resources Defense Council*. About 20–40% of the produce grown for human consumption goes uneaten because of its appearance. A company called "Imperfect" is trying to decrease this waste by working with the farmers in order to get and sell these wasted foods in a lower price to the community. The California-based start-up began its service in Oakland and Berkeley in the summer of 2015. This is something that could be developed or amplified around the country, as the waste of food is enormous [5].

"Farmers should profit, making money off crops that would have otherwise been trashed in landfills. Customers benefit because the fruit and veggies cost 30–50% less than they would at the grocery store, and tastes exactly the same as its physically attractive counterparts, the company pledges" [5] (Fig. 22.3). This something that should be done in the rest of the country to favor communities that cannot afford the high prices of fruits and vegetables. This would also help the farmers to keep up with their profits and everybody will be happy.



Fig. 22.3 Logo of the imperfect produce company that works with produce that are not perfect to be marketable to usual vendors (Picture given and authorized by Imperfect Produce)

Other initiatives that have helped farmers to save money and offer more produce to the population are the farmer's markets – in the last 10 years, they have doubled in number, selling more high-quality produce that might not meet the supermarket criteria of size, appearance, and shelf life. Another initiative was the recently approved California bill that gives growers to receive a tax credit when they donate their leftovers to food banks. Arizona, Oregon, and Colorado have already those bills in place [23].

Healthy Eating Recommendations

Self Nutrition Assessment

In addition to fruits and vegetables, a healthy diet also includes whole grains, fat-free or low-fat milk and milk products, lean meats, poultry, fish, dry beans, eggs, and nuts and is low in saturated fats, trans-fats, cholesterol, salt, and added sugars (CDC) (Fig. 22.4) [46]. An effective tool to help people integrate the practical eating guide is *ChooseMyPlate* developed by the US Department of Agriculture; visit http://www.choosemyplate.gov for more information on this project.

ChooseMyPlate is an online dietary assessment tool that provides information on one's diet serving portions, nutritional messages, and links to nutrient information. Once one provides a day's worth of dietary information, one will receive an overall evaluation by comparing the amounts of food eaten to current nutritional guidance. It gives a better understanding of diet over time, which can be tracked using their *Super Tracker* app.

Sales Promotion at Grocery Stores

It is important to understand that food is marketed to consumers, which influences their dietary behavior, for instance, "buy-one-get-one-free" foods that are displayed in the front of the store, free samples provided while grocery shopping, or even "reduced price with purchase" and

EAT THE MYPLATE WAY

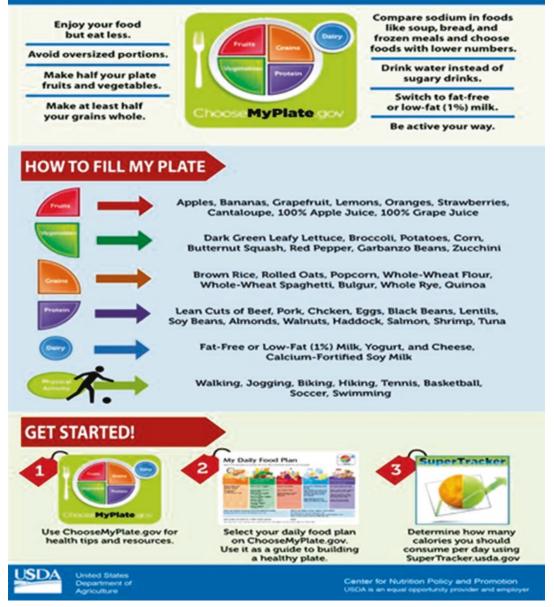


Fig. 22.4 Eat the ChooseMyPlate way (Image: USDA, ChooseMyPlate (USDA))

"bonus-size" packages. These sale promotion strategies are having more of an impact on what foods we buy and how much we eat. This makes behavior modification more challenging since the sales typically are not on low-fat, healthy food items [24]. Low-income individuals tend to be attracted to the sales and advertising and more often choose these forms of dietary behaviors rather than fresh fruits and vegetables.

Conclusion

Given the obesity epidemic, more attention has been given to how the food environment plays a role. There is a need for better access to healthy food and limits on energy-dense, high-fat food with poor nutritional value. The availability of healthy food in local food stores is associated with a higher intake of fresh fruits and vegetables and a limit on processed food. While there are methods to increase the purchase of fruits and vegetables within large corporate supermarkets and grocery stores, sales promotions and food advertisements make it challenging by encouraging overeating. Although supermarkets typically sell fresh produce, they are less available and affordable in low-income and ethnic minority neighborhoods. Similarly, residents in rural areas have even less access to healthy food at supermarkets compared urban neighborhoods. Areas without supermarkets instead have convenience stores and corner stores that typically do not carry food with high nutritional value. It is unreasonable to expect behavior change in eating healthier when there are social, cultural, and environmental factors that do not support such change [14].

References

- Agriculture and Health Policies in Conflict. Government support for unhealthful foods. n.d. Retrieved from committee for responsible medicine: http://www.pcrm.org/health/reports/agriculture-and-health-policies-unhealthful-foods.
- Aubrey A. So long, big Mac: Cleveland Clinic ousts McDonald's from cafeteria. 2015. Retrieved from the salt: what's on your plate: http://www.npr.org/sections/thesalt/2015/08/19/432885995/so-long-big-mac-clevelandclinic-ousts-mcdonalds-from-cafeteria.
- 3. Babey SH. Income disparities in obesity trends among California adolescents. Am J Public Health. 2010;100(11):2149–55.
- Battle KB. Confronting a rising tide of eating disorders and obesity: treatment vs. prevention and policy. Elsevier. 1996;21(6):755–65.
- 5. Bratskeir K. 6 Billion pounds of perfectly edible produce is wasted every year, simply because it's ugly. 2015. Retrieved from Huffpost Taste: http://www.huffingtonpost.com/2015/05/19/food-waste-ugly-fruits-and-vegs-dont-judge_n_7309432.html.
- CBS. Group pushes to oust McDonald's from hospital lobbies. 2012. Retrieved from CBS DFW. http://dfw.cbslocal.com/2012/04/16/group-pushes-to-oust-mcdonalds-from-hospital-lobbies/.
- 7. CDC. Adult obesity facts. 2015. Retrieved from http://www.cdc.gov/obesity/data/adult/html.
- 8. CDC. Diet/Nutrition. 2014. Retrieved from http://www.cdc.gov/nchs/fastats/diet.htm.
- CDC. Division of nutrition, physical activity, and obesity. 2014. Retrieved from http://www.cdc.gov/obesity/data/ childhood.html
- CDC. Leading causes of death. 2015. Retrieved January 25, 2016, from http://www.cdc.gov/nchs/fastats/leadingcauses-of-death.htm.
- 11. CDC EI. Trends in state and federal cigarette tax and retail price United States-1970-2013. 2013. Retrieved from http://www.cdc.gov/tobacco/data_statistics/tables/economics/infographics/pdfs/linegraph2013_TAG508.pdf.
- 12. Commins J. Prognosis grim for McDonald's restaurants in hospitals. 2012. Retrieved from HealthLeaders Media: http://healthleadersmedia.com/page-3/HR-278987/Prognosis-Grim-for-McDonalds-Restaurants-in-Hospitals.
- 13. Croll JN-S. Healthy eating: what does it mean to adolescents? J Nutr Educ. 2001;33:193–8.
- Davis RC. A community resilience approach to reducing ethnic and racial disparities in health. Am J Public Health. 2005;95(12):2168–73.
- 15. Department of Health and Human Services, C. State indicator report on fruits and vegetables. National action guide. 2009. Retrieved from www.cdc.gov/nutrition/downloads/NationalActionGuide2009.pdf.
- 16. Drewnowski A. Can low-income Americans afford a healthy diet? Nutr Today. 2010;44(6):246–9.
- Farthing H. American hospitals need to stop offering fast food, quick! 2015. Retrieved from Disruptive Women in Health Care: http://www.disruptivewomen.net/2015/08/26/american-hospitals-need-to-stop-offering-fast-food-quick/
- 18. Feng JT. The built environment and obesity: a systematic review of the epidemiologic evidence. Health Place. 2010;16:175–90.
- French SJ. Pricing and promotion effects on low-fat vending snack purchases: the CHIPS study. Am J Public Health. 2001;91(1):112–7.

- 20. Fuji E. The demand for cigarettes: further empirical evidence and its implication for public policy. Applied Economics. 1980;479–89.
- 21. Gibson D. The neighborhood food environment and adult weight status: estimates from longitudinal data. Am J Public Health. 2011;101(1):71–8.
- 22. Gordon E. Hospital bids bye-bye to Big Macs, others may follow suit. 2012. Retrieved from The Salt: What's on your plate: http://www.npr.org/sections/thesalt/2012/12/17/167463286/hospital-bids-bye-bye-to-big-macs-others-may-follow-suit.
- Gunders D. Wasted: How America is losing up to 40 percent of its food from farm to fork to landfill. NRDC Issue Paper. 2012;12(6):1–26.
- 24. Hawkes C. Sales promotions and food consumption. Nutr Rev. 2009;67(6):333-42.
- 25. Hearst MH. Nutritional quality at eight U.S. fast-food chains: 14-year trends. Am J Prev Med. 2013;44(6):589–94.
- 26. Herman B. 20 Hospitals urged to end food contracts with McDonald's. 2012. Retrieved from Becker's Hospital Review: http://www.beckershospitalreview.com/hospital-management-administration/20-hospitals-urged-to-endfood-contracts-with-mcdonalds.html
- Holmes LJ. Racial-ethnic differences in childhood higher body mass index: insurance status explanatory model. Int J Hum Rights Healthc. 2015;8(1):45–56.
- Horowitz CC. Barriers to buying healthy foods for people with diabetes: evidence of environmental disparities. Am J Public Health. 2004;94(9):1549–54.
- IARC. Effectiveness of tax and price policies for tobacco control, vol. 14. IARC; 2011. Retrieved from http://publications.iarc.fr/Book-And-Report-Series/Iarc-Handbooks-Of-Cancer-Prevention/Effectiveness-Of-Tax-And-Price-Policies-For-Tobacco-Control-2011. International Agency for Research on Cancer (IARC), World Health Organization, Lyon CEDEX 08, France.
- 30. Jacobson MB. Small taxes on soft drinks and snack foods to promote health. Am J Public Health. 2000;90(6):854–7.
- 31. Jovanovic LH. Advances in diabetes for the millennium: diabetes in minorities. Medscape General Med. 2004;6(3):2.
- Kubik ML. The association of the school environment with dietary behaviors of young adolescents. Am J Public Health. 2003;93(7):1168–73.
- Larson NS. Neighborhood environments: disparities in access to healthy foods in the U.S. Am J Prev Med. 2009;36(1):74–81.
- 34. National Collaborative on Childhood Obesity Research. Childhood obesity in the United States. n.d. Retrieved from NCCOR Layout 1: http://www.nccor.org/downloads/ChildhoodObesity_020509.pdf.
- 35. National Institute of Diabetes and Digestive and Kidney Diseases, N. Trends in overweight and obesity among Adults. NIH.
- 36. Nestle JJ. Halting the obesity epidemic: a public health policy approach. Public Health Rep. 2000;115(1):12–24.
- 37. Ortiz. Childhood obesity prevention: tax on sweetened beverages. Senate Health and Human Services Committee Analysis. SB 1520: Revenue and Taxation/Appropriations. 2002.
- 38. Papas MA. The built environment and obesity. Epidemiol Rev. 2007;29:129-43.
- 39. Pulos EL. Evaluation of a voluntary menu-labeling program in full-service restaurants. A J Public Health. 2010;100(6):1035–9.
- Sacramento Start. City of Sacramento Start Program. n.d. Retrieved January 2016, from http://portal.cityofsacramento.org/ParksandRec/Recreation/START.
- 41. Story MN. Schools and obesity prevention: creating school environment and policies to promote healthy eating and physical activity. Milbank Q. 2009;87(1):71–100.
- 42. Sun Y. Health concern, food choice motives, and attitudes towards healthy eating: the mediating role of food choice motives. Appetite. 2008;51:42–9.
- Swartz JJ et al. Calorie menu labeling on quick-service restaurant menus: an update systematic review of the literature. Int J Behav Nutr Phys Act. 2011;8(135):1–8.
- 44. Tester JS. An analysis of public health policy and legal issues relevant to mobile food vending. Am J Public Health. 2010;100(11):2038–46.
- 45. Troy LM. Hunger and obesity: understanding a food insecurity paradigm. Food and Nutrition Board; Institute of Medicine. 2011.
- 46. USDA. ChooseMYPlate.gov
- 47. WHO. The Top 10 causes of death. 2014. Retrieved from http://www.who.int/mediacentre/factsheets/fs310/en/.
- 48. Wootan ME. Poor nutrition on the menu: children's meal at America's top chain restaurants. Child Obes. 2012;8(3):251–4.

Chapter 23 Food Safety

Carol Byrd-Bredbenner

Key Points

- The incidence of foodborne illness is needlessly high.
- All food handlers—at every stage in the food chain—share responsibility for protecting the safety of the food supply.
- At the home level, there is considerable room for improving food safety practices.
- Most consumers are interested in food safety and want to learn how to keep their food supply safe which bodes well for the success of food safety education efforts and progress toward national food safety goals.

Keywords Food safety • Foodborne illness

Introduction

"Food, glorious food! Is it worth the waiting for?" [1] Yes, when it is both healthful and safe. The 2015 Food and Health Survey found that these qualities are important to the vast majority of Americans – with about half putting a great deal of thought and effort into controlling the healthfulness and safety of the foods they eat [2]. Yet, according to estimates from the Centers for Disease Control and Prevention (CDC), about one in every six US residents suffers a bout of foodborne illness every year. Of the estimated 48 million Americans who are victims of foodborne illness annually, 128,000 become so seriously ill they require hospitalization and 3000 die [3, 4].

Rutgers University, New Brunswick, NJ 08901, USA

e-mail: bredbenner@aesop.rutgers.edu

C. Byrd-Bredbenner, PhD, RD, FAND

Incidence of Foodborne Illness

Total US population estimates of foodborne illness are based on Foodborne Diseases Active Surveillance Network (FoodNet) data from ten states. FoodNet is a collaborative program among the CDC, ten state health departments, and US Department of Agriculture's Food Safety and Inspection Service that actively observes about 15% of the US population (currently more than 48 million people) with regard to key bacterial and parasitic pathogens commonly transmitted through food [5]. As shown in (Fig. 23.1), FoodNet data indicate that the incidence of culture-confirmed bacterial infections and laboratory-confirmed parasitic infections cases has averaged about 40 cases per 100,000 people since 2000.

Of the 19,542 culture-confirmed bacterial infections and laboratory-confirmed parasitic infections cases identified in FoodNet surveillance during 2014, nearly one-quarter resulted in hospitalization. The likelihood of hospitalization is associated with both the type of foodborne pathogen and the age of the individual. For example, Table 23.1 shows that *Listeria* infection is far more likely to result in hospitalization than infection with other pathogens. *Salmonella* and *Listeria* were responsible for 42% and 25%, respectively, of the 71 deaths caused by foodborne illness in 2014 [6].

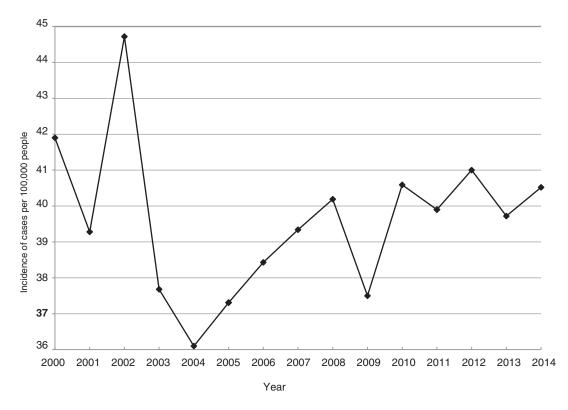


Fig. 23.1 Incidence of culture-confirmed bacterial infections and laboratory-confirmed parasitic infections cases per 100,000 people* [9]. *Infections include *Campylobacter, Listeria, Salmonella, Shigella*, Shiga toxin-producing Escherichia coli (STEC) 0157 and non-0157, *Vibrio, Yersinia, Cryptosporidium, Cyclospora*

Pathogen	Hospitalizations (N)	Percent hospitalized	
Campylobacter $(n=6486)$	1080	17	
Listeria $(n=118)$	108	92	
Salmonella $(n=7452)$	2141	29	
Shigella $(n=2801)$	569	20	
$STEC^{a} O157 (n = 445)$	154	35	
STEC non-O157 (<i>n</i> =690)	104	15	
Vibrio $(n=216)$	40	19	
<i>Yersinia</i> $(n = 133)$	30	23	
Cryptosporidium (n = 1175)	217	18	
Cyclospora (n=26)	2	8	

Table 23.1 Hospitalizations in 2014 resulting from culture-confirmed bacterial infections and laboratory-confirmed parasitic infections cases [7]

^aShiga toxin-producing Escherichia coli (STEC) 0157

 Table 23.2
 Hospitalizations by age group in 2014 resulting from culture-confirmed bacterial infections and laboratoryconfirmed parasitic infections cases [7]

Pathogen	Percent hospitalized					
	Age <5 years n=3557 total cases; 544 hospitalizations	Age 5–9 years n=1738 total cases; 274 hospitalizations	Age 10–19 years $n = 1976$ total cases; 350 hospitalizations	Age 20–64 n=9754 total cases; 2249 hospitalizations	Age ≥ 65 n=2517 total cases; 1028 hospitalizations	
Campylobacter	7	11	12	16	30	
Listeria	100	100	100	89	90	
Salmonella	21	22	23	28	50	
Shigella	9	12	19	31	36	
STEC ^a O157	35	30	34	32	50	
STEC non-O157	7	9	12	17	37	
Vibrio	0	11	0	15	37	
Yersinia	17	20	13	20	35	
Cryptosporidium	6	12	10	22	36	
Cyclospora	0	0	0	11	0	
Total	15	16	18	23	41	

^aShiga toxin-producing Escherichia coli (STEC) 0157

Table 23.2 reports that older adults were far more likely to be hospitalized as a result of foodborne illness than other age groups [7]. Similarly, the incidence of death from foodborne illness is highest among adults age 65 years and older, accounting for 56% of the confirmed foodborne illness deaths in 2014 [6].

Compared to data from the mid-1990s, the relative combined rates of confirmed infections from monitored foodborne pathogens (i.e., *Campylobacter*, Shiga toxin-producing *Escherichia coli* (STEC) O157, *Listeria*, *Salmonella*, *Vibrio*, and *Yersinia*) were lower throughout the 2000s [8]. However, when examined individually, *Vibrio* is currently much higher than in the mid-1990s and is on an upward trend while *Salmonella* incidence has remained fairly unchanged over time.

A comparison of the incidence of culture-confirmed bacterial infections and laboratory-confirmed parasitic infections cases to Healthy People 2010 objectives (Fig. 23.2) shows that the incidence of STEC O157 and *Listeria* were closely congruent with objectives whereas both *Salmonella* and *Campylobacter* incidence exceeded goals [9]. The most recent incidence data (i.e., 2014) compared to the newer Healthy People 2020 objectives in (Fig. 23.3) indicates close agreement for *Yersinia* and

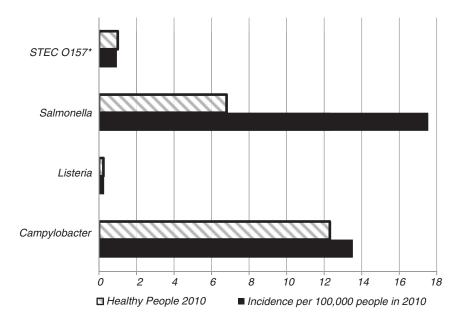


Fig. 23.2 Comparison of Healthy People 2010 objective targets for incidence of foodborne infections per 100,000 people to incidence in 2010* [9]. *Shiga toxin-producing *Escherichia coli* (STEC) 0157

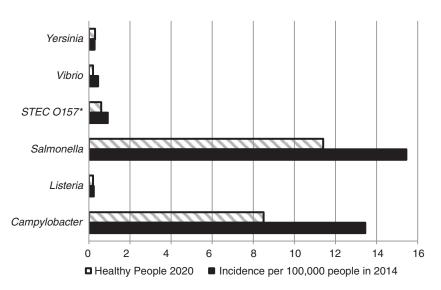


Fig. 23.3 Comparison of Healthy People 2020 objective targets for incidence of foodborne infections per 100,000 people to incidence in 2014 * [9]. *Shiga toxin-producing *Escherichia coli* (STEC) 0157

Listeria while *Vibrio* and STEC O157 need improvement and *Salmonella* and *Campylobacter* incidence need considerable reductions to meet the objectives [9].

Although strides have been made toward reducing foodborne illness, greater and more concentrated efforts are warranted. Indeed, the suffering caused by foodborne illness is largely needless because nearly all foodborne illness is preventable [10]. Knowing foods frequently associated with foodborne illness and applying safe food-handling practices at every point in the food chain can help reduce the incidence of foodborne illness.

Foodborne Illness Pathogens and Common Food Sources

There are over 250 foodborne illness pathogens. FoodNet monitors key foodborne bacteria (i.e., *Campylobacter*,STEC O157, STEC non-O157, *Listeria, Salmonella, Yersinia, Vibrio*) and parasites (i.e., *Cryptosporidium, Cyclospora*). In addition to these, there are numerous other pathogenic bacteria (e.g., *Staphylococcus aureus, Clostridium perfringens, Clostridium botulinum*), parasites (e.g., *Toxoplasma gondii, Trichinella spiralis*, anisakis, and tapeworms), viruses (e.g., norovirus, hepatitis A), prions, and toxins (e.g., mycotoxins, seafood toxins, plant toxins) that can cause foodborne illness, hospitalizations, and deaths. Table 23.3 describes the foods most frequently associated with common foodborne illness pathogens and toxins.

Knowing the food types most commonly associated with foodborne illness can help food producers, food processers, transporters, wholesalers, retailers, consumers, and regulators target efforts to safeguard the food supply. However, it is important to remember that any food can become contaminated with disease-causing agents at any point in the food chain.

In the decade spanning 1998–2008, produce was the food commodity most commonly implicated in foodborne illness outbreaks – accounting for 46% of all foodborne illnesses and 23% of deaths from foodborne illness [11]. Produce includes fruits, vegetables, nuts, sprouts, and mushrooms. Nearly all of the other cases of foodborne illnesses were caused by contaminated animal products as were about 50% of related deaths. That is, more than one in five foodborne illnesses and almost one-third of deaths were attributable to meat and poultry. Dairy and eggs contributed to 20 and 15% of foodborne illnesses and deaths, respectively. About 6% of foodborne illnesses were attributed to fish and shellfish as well as 6% of foodborne illness deaths. Very low percentages of foodborne illnesses and deaths were caused by foods other than produce, meat, poultry, dairy, eggs, fish, and shellfish. However, one in four foodborne illness-related deaths were not attributed to commodities and were mostly caused by pathogens *Toxoplasma* and *Vibrio*.

Safe Food-Handling Responsibilities

Food safety is the responsibility of everyone in the food chain. Laws and good manufacturing practices help protect the food supply from field to food retailer. For example, the US Department of Agriculture created standards to help industry reduce contamination of chicken with *Salmonella* and *Campylobacter* [12]. The US federal government passed extensive food safety legislation known as the Food Safety Modernization Act (FSMA) in 2011 intended to protect the US food supply [13, 14]. The FMSA gives FDA the responsibility for requiring food facilities to create plans for identifying and minimizing food safety hazards for food for humans and animals, establishing standards for USA and foreign farms growing and handling produce, requiring importers to verify foods entering the USA were produced according to US standards, accrediting foreign facilities producing human and animal food entering the USA, requiring sanitary practices for transporting food, and mandating USA and foreign facilities have procedures for preventing intentional adulteration of food [15].

Although food safety laws typically do not extend into private homes, home food preparers are the last line of defense. In fact, 15–33% of foodborne illness cases originate in the home [16, 17]. The incidence of foodborne illness from foods prepared at home is likely because more food is prepared at home than eaten out [18]; home kitchens are used for many activities not related to food preparation (e.g., pet care, laundry, house plants) which increase risk of contamination of kitchen areas with *Salmonella, Campylobacter*, and other foodborne pathogens [19–22], and many home food preparers are not handling food as safely as they could be [23–29] (e.g., 39% thaw raw meat or poultry on the kitchen counter; 16% do not wash hands with soap and water before food preparation; 25% do not

Foodborne illness causes	Common food sources
Bacterial	
Campylobacter	Raw or undercooked poultry, unpasteurized (raw) dairy products, contaminated water
Escherichia coli	Food or water contaminated with human or animal feces, unpasteurized milk and juice, soft cheeses made from unpasteurized milk, raw fruits and vegetables (e.g., sprouts), undercooked meat, especially ground meat
Listeria	Deli meats, hot dogs, meat spreads, unpasteurized dairy products, soft cheeses (e.g., queso fresco, Brie, Camembert), smoked seafood, raw sprouts
Salmonella	Raw or undercooked poultry, eggs, meat; unpasteurized milk or juice, contaminated produce, spices, and nuts
Yersinia	Raw or undercooked pork, especially chitterlings (intestines); contaminated water, unpasteurized milk
Vibrio	Raw or undercooked seafood, especially oysters (<i>Vibrio</i> naturally occurs in ocean waters and multiplies when water warms up in summer months)
Staphylococcus	Foods prepared with hand contact and not further cooked (e.g., moist salads like egg, tuna, ham, chicken, potato, and macaroni salad), cream-filled bakery items (e.g., pastries, eclairs), sandwiches
Clostridium perfringens	Foods prepared in large quantities and kept warm for more than 2 h; beef, poultry, gravy
Clostridium botulinum	Home-canned vegetables and fruits, home-canned or fermented fish, herb- infused oils, cheese sauce Infants: corn syrup and honey
Shigella	Water contaminated with human or animal feces; unwashed fruits and vegetables, foods handled by infected person
Bacillus cereus	Rice, leftover foods, sauces, soups
Brucella	Raw meat, unpasteurized dairy products
Parasites	
Cryptosporidium	Water contaminated with human or animal feces; unwashed fruits and vegetables
Cyclospora	Water contaminated with human or animal feces; unwashed fruits and vegetables
Toxoplasma gondii	Raw or undercooked contaminated meat, contaminated water
Trichinella spiralis	Raw or undercooked contaminated pork, horse, dog, wild animals
Giardia intestinalis	Contaminated water, raw or undercooked contaminated meat
Anisakis	Raw or undercooked contaminated seafood
Tapeworms	Raw or undercooked contaminated beef or pork
Viruses	
Norovirus	Produce, shellfish, foods prepared by an infected person
Hepatitis A	Contaminated water, raw or undercooked shellfish from contaminated water, raw produce, foods prepared by an infected person
Astrovirus	Contaminated water and shellfish, food prepared by an infected person
Rotavirus	Contaminated water, food prepared by an infected person
Sapovirus	Contaminated water and shellfish, food prepared by an infected person
Prions	Meat/tissue from animals prion disease (e.g., infected cattle, sheep, deer, elk)
Toxins	
Mycotoxins	
Aflatoxin	Molded peanuts, nuts, corn, wheat, cottonseed
Ergot	Molded rye
Seafood toxins	
Ciguatera	Large warm water contaminated fish (e.g., predatory fish like barracuda)
Scombroid	Certain fish when they start to decompose (e.g., tuna, bonito, mackerel, marlin, herring, sardines)

(continued)

Table 23.3 (continued)

Foodborne illness causes	Common food sources
Tetrodotoxin	Pufferfish (fugu) liver, skin, ovaries
Paralytic, diarrheic, neurotoxin and amnesic shellfish toxins	Contaminated shellfish
Plant toxins	
Safrole	Sassafras, nutmeg, mace
Solanine	Green potatoes
Herbal preparations	Senna, comfrey
Lectins	Undercooked legumes
Mushrooms	Some mushrooms, such as amanita

wash cutting boards with soap and water or bleach) [2, 30]. Unsafe food handling at home may be because consumers are not concerned about food safety issues (e.g., one in ten have no food safety issue concerns), lack equipment (e.g., 40% lack food thermometers) or do not use it regularly (e.g., 70% do not use a food thermometer to check meat and poultry doneness), lack awareness of foodborne pathogens (e.g., 89% have not heard of *Campylobacter*), lack knowledge of how to prevent foodborne illness (e.g., 12 believe washing *Salmonella*-contaminated food would make it safe to eat) [31], and six in ten are confident in the safety of the food supply and believe their chance of getting foodborne illness is extremely low [32].

Food Safety Education and Practices

Knowledge of safe food handling is requisite to keeping foods safe at home. Audits of home kitchens based on criteria applied in retail food establishments indicate that the average home would fail the inspection [28, 33–35].

Several resources are available to help consumers quickly and easily develop the knowledge and skills needed to reduce their foodborne illness risk. Mostly all food safety education campaigns focus their messages on keeping hands and kitchen surfaces clean, cooking foods sufficiently, refrigerating foods promptly, and preventing cross-contamination. For example, the US Department of Agriculture campaign is Cook, Clean, Chill, and Separate [36]. The World Health Organization has five keys to safer food: Keep clean, Separate raw and cooked, Cook thoroughly, Keep food at safe temperatures, and Use safe water and raw materials [37, 38].

Hands are a primary vehicle for transferring pathogens to foods and kitchen surfaces [39, 40]. Washing hands with soap for 20 s before food preparation, after handling raw produce and raw animal proteins (e.g., meat, eggs, poultry), after using the toilet or changing diapers, and after handling garbage is critical to preventing foodborne illness [41, 42]. The vast majority of individuals report washing their hands before preparing food and after handling meat, however research indicates consumers' handwashing practices are often insufficient to prevent cross-contamination with pathogenic bacteria [23, 32, 40, 43–46].

Kitchen counters, food preparation equipment (e.g., knives, cutting boards), handles on sinks and refrigerators, and cleaning cloths and sponges also are primary vehicles for cross-contamination [27, 47–51]. Pathogenic bacteria identified on these areas include *Campylobacter*, *Salmonella*, *Staph*, *E. coli*, and *Listeria*. In one study, more than one in eight foodborne illnesses were traced to insufficiently washed knives and cutting boards [24]. Insufficient cleaning of kitchen equipment has been observed in numerous studies [26–29].

Controlling the temperature of food is a key strategy for preventing foodborne illness. Temperature includes those associated with cooking food as well as temperatures of foods stored and held for later

consumption. Consumers realize that undercooked foods pose great risk for foodborne illness and are aware the meat and poultry should be cooked to sufficiently high temperatures, yet many do not cook these foods to high enough temperatures, do not realize that color is unsuitable for detecting adequate doneness, and do not use a cooking thermometer to check doneness [25, 30]. On the plus side, most consumers indicate they do not let prepared foods sit at room temperatures for more than 2 h and tend to thaw foods in the refrigerator [25, 46]. However, home refrigerator temperatures often are too warm, few consumers check refrigerator temperatures regularly or even have a refrigerator thermometer available [24, 46].

As discussed previously, handwashing practices often are inadequate to prevent cross-contamination. Other cross-contamination prevention practices, however, tend to be somewhat better. For example, the majority of consumers report they keep raw meat, poultry, and seafood separated from foods that are ready to eat and nearly all indicate they use different plates for raw and cooked meat [25].

Conclusion

The incidence of foodborne illness is needlessly high. All food handlers – at every stage in the food chain – share responsibility for protecting the safety of the food supply. At the home level, there is considerable room for improving food safety practices. Most consumers are interested in food safety and want to learn how to keep their food supply safe which bodes well for the success of food safety education efforts and progress toward national food safety goals [23, 52, 53].

References

- 1. Bart L. Food, glorious food. 1968 Contract No.: Document Number.
- International Food Information Council Foundation. What's your health worth? Food & Nutrition Survey 2015. Washington, DC; 2015 Contract No.: Document Number.
- CDC. Estimating foodborne illness: an overview. Atlanta: Centers for Disease Control and Prevention; 2014 [updated 2014; cited 15 Mar 2016]; Available from: www.cdc.gov/foodborneburden/estimates-overview.html.
- Foodborne Diseases Active Surveillance Network (FoodNet). Number and incidence of infections by Year, 1996– 2014; Table 2a. Atlanta: Centers for Disease Control and Prevention; 2015 [updated 2015; cited 16 Mar 2016]; Available from: www.cdc.gov/foodnet/trends/2014/number-of-infections-by-year-1996-2014.html#table2a.
- Foodborne Diseases Active Surveillance Network (FoodNet). Tables and figures-2014 preliminary data. Atlanta: Centers for Disease Control and Prevention; 2015 [updated 2015; cited 16 Mar 2016]; Available from: www.cdc. gov/foodnet/trends/tables-2014.html.
- 6. Foodborne Diseases Active Surveillance Network (FoodNet). Number of deaths and case fatality ratio by age group and pathogen, 2014; Table 14. Atlanta: Centers for Disease Control and Prevention; 2015 [updated 2015; cited 16 Mar 2016]; Available from: www.cdc.gov/foodnet/trends/2014/number-of-deaths-cfr-by-age-group-pathogen-2014. html.
- Foodborne Diseases Active Surveillance Network (FoodNet). Number and percentage of hospitalizations by age group and pathogen, 2014, Table 12e. Atlanta: Centers for Disease Control and Prevention; 2015 [updated 2015; cited 16 Mar 2016]; Available from: www.cdc.gov/foodnet/trends/2014/number-percentage-of-hospitalizations-by-age-group-pathogen-2014.html#table12a.
- Foodborne Diseases Active Surveillance Network (FoodNet). Figures 2014 preliminary data, figure 15. Atlanta: Centers for Disease Control and Prevention; 2015 [updated 2015; cited 16 Mar 2016]; Available from: www.cdc. gov/foodnet/trends/figures-2014.html#ui-id-29.
- Foodborne Diseases Active Surveillance Network (FoodNet). Number and incidence of infections by year, 1996–2014; Table 2b. Atlanta: Centers for Disease Control and Prevention; 2015 [updated 2015; cited 16 Mar 2016]; Available from: www.cdc.gov/foodnet/trends/2014/number-of-infections-by-year-1996-2014.html#table2a.
- Crim S, Griffin P, Tauxe R, Marder E, Gilliss D, Cronquist A et al (2015) Preliminary incidence and trends of infection with pathogens transmitted commonly through food foodborne diseases active surveillance network, 10 U.S. Sites, 2006–2014. Morb Mortal Wkly Rep 64(18):495–9

- 11. Painter J, Hoekstra R, Ayers T, Tauxe R, Braden C, Angula F et al (2013) Attribution of foodborne illnesses, hospitalizations, and death to food commodities by using outbreak data, United States, 1998–2008. Emerg Infect Dis 19(3):407–15
- CDC. Campylobacter. Atlanta: Centers for Disease Control and Prevention; 2014 [updated 2014; cited 15 Mar 2016]; Available from: www.cdc.gov/foodsafety/diseases/campylobacter/index.html.
- Foodborne Diseases Active Surveillance Network (FoodNet). Questions and answers about the 2014 FoodNet MMWR. Atlanta: Centers for Disease Control and Prevention; 2015 [updated 2015; cited 16 Mar 2016]; Available from: http://www.cdc.gov/foodnet/trends/mmwr-questions-and-answers-2014.html.
- Food and Drug Administration. FDA Food Safety Modernization Act (FSMA). Washington, DC: U.S. Government;
 2016 [updated 2016; cited 19 Mar 2016]; Available from: www.fda.gov/Food/GuidanceRegulation/FSMA/ ucm242500.htm.
- Food and Drug Administration. Frequently asked questions on FSMA. Washington, DC: U.S. Government; 2016 [updated 2016; cited 19 Mar 2016]; Available from: www.fda.gov/Food/GuidanceRegulation/FSMA/ucm247559. htm.
- 16. CDC. Table 3. Number of reported foodborne disease outbreaks and outbreak-associated illnesses, by etiology and place where food was eaten* United States, 2008; 2011 Contract No.: Document Number.
- European Food Safety Authority. Trends and sources of zoonoses and zoonotic agents and food-borne outbreaks in the European Union in 2008. Parma; 2010 Contract No.: Document Number.
- Carlson A, Kinsey J, Nadav C (2002) Consumers' retail source of food: a cluster analysis. Fam Econ Nutr Rev 14:11–20
- Redmond E, Griffith C, Riley S (2009) Contamination of bottles used for feeding reconstituted powdered infant formula and implications for public health. Perspect Public Health 129:85–94
- Byrd-Bredbenner C, Maurer J, Wheatley V, Cottone E, Clancy M (2007) Food safety hazards lurk in the kitchens of young adults. J Food Prot 70:991–6
- Josephson K, Rubino J, Pepper I (1997) Characterization and quantification of bacterial pathogens and indicator organisms in household kitchens with and without the use of a disinfectant cleaner. J Appl Microbiol 83:737–50
- 22. Rusin P, Orosz-Coughlin P, Gerba C (1998) Reduction of faecal coliform and heterotropic plate count bacterial in the household kitchen and bathroom by disinfection with hypochlorite cleaners. J Appl Microbiol 85:819–28
- de Jong A, Verhoeff-Bakkenes L, Nauta M, de Jong R (2008) Cross-contamination in the kitchen: effect of hygiene measures. J Appl Microbiol 105:615–24
- 24. Kennedy J, Jackson V, Blair I, McDowell D, Cowan C, Bolton D (2005) Food safety knowledge of consumers and the microbiological and temperature status of their refrigerators. J Food Prot 68:1421–30
- 25. American Dietetic Association, ConAgra Foods. A Benchmark survey: consumer knowledge of home safety practices, a workplace food survey: "Desktop Dining"; 2011 Contract No.: Document Number.
- 26. Jay L, Comar D, Govenlock L (1999) A national Australian food safety telephone survey. J Food Prot 62(8):921–8
- 27. Redmond E, Griffith C (2003) Consumer food handling in the home: a review of food safety studies. J Food Prot 66:130–61
- Byrd-Bredbenner C, Maurer J, Wheatley V, Cottone E, Clancy M (2007) Observed food safety behaviors and skills of young adults. Br Food J 107:519–30
- Anderson JB, Shuster TA, Hansen KE, Levy AS, Volk A (2004) A camera's view of consumer food-handling behaviors. J Am Diet Assoc 104(2):186–91
- 30. United States Department of Health and Human Services. Healthy people 2020: food safety. In: United States Department of Health and Human Service, editor. Washington, DC: Office of Disease Prevention and Health Promotion; 2010.
- 31. International Food Information Council Foundation. 2014 Food & Nutrition Survey: the pulse of America's diet from beliefs to behaviors. Washington, DC; 2014 Contract No.: Document Number.
- 32. International Food Information Council Foundation. 2012 Food & Nutrition Survey: consumers attitudes toward food safety, nutrition, & health. Washington, DC; 2012 Contract No.: Document Number.
- 33. Kuo T, Dela Cruz H, Redelings M, Smith L, Reported R, Simon P et al (2010) Use of a self-assessment questionnaire for food safety education in the home kitchen – Los Angeles County, CA 2006–2008. MMWR CDC Surveill Summ 59(34):1098–101
- 34. Daniels R (1998) Home food safety. Food Tech 52(2):54-6
- 35. Daniels R, Daniels B, Gilmet P, Noonan D. Audits International 2000 Home Food Safety Study Report: Audits International; 2001 Contract No.: Document Number.
- CDC. Food Safety. Atlanta: Centers for Disease Control and Prevention; 2015 [updated 2015; cited 15 Mar 2016]; Available from: www.cdc.gov/foodsafety/groups/consumers.html.
- 37. World Health Organization. Prevention of foodborne disease: five keys to safe food. 2009 [updated 2009; cited 5 Nov 2012]; Available from: www.who.int/foodsafety/publications/consumer/manual_keys.pdf.

- World Health Organization. Five keys to safer food. Geneva; undated [updated undated; cited 18 Mar 2016]; Available from: www.who.int/foodsafety/publications/consumer/en/5keys_en.pdf?ua=1.
- 39. Fischer A, de Jong A, van Asselt E, de Jong R, Frewer L, Nauta M (2007) Food safety in the domestic environment: an interdisciplinary investigation of microbial hazards during food preparation. Risk Anal 27:1065–82
- Kennedy J, Gibney S, Nolan A, McMahon M, McDowell D, Fanning S et al (2011) Identification of critical points during domestic preparation: an observational study. Br Food J 113:766–83
- van Asselt E, de Jong A, de Jong R, Nauta M (2008) Cross-contamination in the kitchen: estimation of transfer rates for cutting boards, hands, and knives. J Applied Microbio 105(5):1392–401
- 42. van Asselt E, Fischer A, De Jong A, Nauta M, De Jong R (2009) Cooking practices in the kitchen observed versus predicted behavior. Risk Anal 29:533–40
- 43. Food Marketing Institute. U.S. Grocery shopper trends 2011. Arlington; 2011 Contract No.: Document Number.
- 44. Hoelzl C, Mayerhofer U, Steininger M, Bruller W, Hofstadter D, Aldrian U (2013) Observation trial of safe food handling behavior during food preparation using the example of Campylobacter spp. J Food Prot 76(3):482–9
- 45. U.S. Census Bureau. State & County QuickFacts. Washington, DC: U.S. Department of Commerce; 2012 [updated 2012; cited 19 Sep 2012]; Available from: http://quickfacts.census.gov/qfd/states/00000.html.
- 46. ConAgra Foods. A Benchmark Survey: consumer knowledge of home food safety practices compared to those of registered dietitians; 2012 Contract No.: Document Number.
- Rossi E, Scapin D, Tondo E (2013) Survival and transfer of microorganisms from kitchen sponges to surfaces of stainless steel and polyethylene. J Infect Dev Ctries 7(3):229–34
- Redmond E, Griffith C, Slader J (2004) Humphrey. Microbiological and observational analysis of cross contamination risks during domestic food preparation. Br Food J 106:581–97
- Redmond E, Griffith P (2009) The importance of hygiene in the domestic kitchen: implications for preparation and storage of food and infant formula. Perspect Public Health 129:69–76
- 50. Griffin P, Worsfold D, Mitchell R (1998) Food preparation, risk communication, and the consumer. Food Control 9:225–32
- Scott E, Bloomfield S (1990) The survival and transfer of microbial contamination via cloths, hands, and utensils. J Applied Bacteriol 68:271–8
- Quick V, Corda K, Byrd-Bredbenner C (2013) Determinants of safe food handling among middle school youth. Nutr Food Sci 43:543–53
- Abbot J, Policastro P, Bruhn C, Schaffner D, Byrd-Bredbenner C (2012) Development and evaluation of a university campus-based food safety media campaign for young adults. J Food Prot 75:1117–24

Index

A

ABC Food Safety System, 403 Academy of Nutrition and Dietetics (AND), 130-131, 163-164, 218 Acceptable macronutrient distribution range (AMDR), 26,30 Acceptance and commitment therapy (ACT), 94 Accreditation Council for Education in Nutrition and Dietetics (ACEND), 123 Action for Health in Diabetes, 180 Actual energy intake (AEI), 110 ADA. See American Diabetic Association (ADA) Added sugars and health, 38 cardiovascular disease, risk factors, 377 diabetes, 378 energy-regulating hormones, 374-375 fructose composition of, 371 metabolism, 372-374 HFCS composition of, 371 metabolism, 372-374 liver fat accumulation, NAFLD and, 379 metabolic syndrome, 378-379 and neurologic response, 379-380 nutritive sweeteners, 371 and obesity, 375-376 public health/public policy considerations, 381 sucrose composition of, 371 metabolism, 372-374 sugar consumption, 371-372 taxation, 370 Adequate intakes (AI), 22-23, 25, 31, 356 Adjustable gastric banding (AGB), 221, 222 Ad libitum strategy, 93, 287, 375, 376 Adolescent athletes behavioral techniques meal timing, 277 promoting healthy environments, 277 weight status and eating disorders, 276-277 beverages, 273-275 dietary needs (see Adolescents' dietary needs) dietary supplements and ergogenic aids, 275 Adolescents' dietary needs calorie needs, 269

early adolescence, 268 late adolescence, 268 macronutrients carbohydrates carbohydrates: added sugars, 270 carbohydrates: brain metabolism, 269 carbohydrates: fibers, 270 carbohydrates: SSB consumption, 269 fats, 271 protein, 271 micronutrients calcium, 273 iron, 272 vitamin D. 272 middle adolescence, 268 nutrient-dense foods, 268 Adult Learning theory, 73 Adults, eating behaviors environmental food cues and influences food marketing communications, 91 portion sizes, 90-91 sensory food cues, 90 external environment, healthy food choices, 95-96 food choice and energy intake, managing strategies, 91 mindful and attentive eating techniques, 94 neurophysiological factors brain's reward system, 87-88 genetic factors, 87 hunger and satiety signaling system, 87 nutritional counseling and education, 94-95 portion control strategies, 93 psychological and cognitive factors disinhibition and impulsivity, 88-89 mood and emotional status, 88 stress, 88 psychosocial factors habit. 89 social norms and social modeling, 89-90 supportive eating environment improve cooking skills/knowledge, 92 nutrient-rich foods, 91–92 portion-controlled meal and snack replacements, 92 Aesthetic health, 314, 319 Affordable Care Act, 4

Aging process, 289, 360 AHA. See American Heart Association (AHA) Allergen labeling, 40, 42 Alpha-linolenic acid (ALA), 200 American Academy of Pediatrics, 274 American Cancer Society (ACS) diet, 56 American College of Cardiology (ACC), 141, 361 American College of Lifestyle Medicine (ACLM), 4 American College of Obstetricians and Gynecologists (ACOG), 299 American College of Sports Medicine (ACSM), 275 American Diabetic Association (ADA), 56, 163-164, 399 American Heart Association (AHA), 4-6, 56, 230, 270, 361, 370, 377 diet and lifestyle recommendations and goals, 138 added sugars, moderate intake of, 145 alcoholic beverages, moderate intake of, 146 "away" food, 146-147 desirable lipid profile, 141 fish and other seafood, consumption of, 144-145 fruits and vegetables, consumption of, 144 healthy body weight, 140-141, 143-144 normal blood pressure, 141-142 overall healthy diet, consumption of, 140 physical activity, 142-144 saturated and trans fat, limited intake of, 145 sodium consumption, reduction of, 146 tobacco products, avoidance of, 143 whole grain, high fiber foods, 144 heart healthy nutrition plans, implementation of, 147 2020 Strategic Plan, 139, 143 American Society of Human Genetics (ASHG), 130 American Stroke Association (ASA), 307 American Thyroid Association, 301 AND. See Academy of Nutrition and Dietetics (AND) Anisakis, 417 Antioxidant nutrients, 27 Appreciation, 61-63 Appreciative inquiry (AI), 68 Arginine vasopressin (AVP), 283 Asymptomatic pregnant women, 302 Atherosclerotic cardiovascular disease (ASCVD), 141 Atherosclerotic vascular disease (ASVD), 199 Atkins Diet, 184, 185 Atrial natriuretic peptide (ANP), 283 Attentive eating, 94 Autosomal-dominant disorder, 240

B

Baby bottle tooth decay. *See* Early Childhood Caries (ECC) Balanced hypo-caloric diets, 235 Bariatric surgery, 156–157 Behavioral principles, 217 Behavior change and nutrition counseling. *See* Nutrition counseling Biggest Loser Diet, 184, 185 Bioelectrical impedance analysis (BIA), 215 **Biomarkers** BOND project, 22 functional/intermediary, 21-22 of intake, 20, 21 limitations, 20 nutritional status, 21 of risk/outcome, 22 sources of, 20 Biomarkers of Nutrition in Development (BOND) project, 22 Body mass index (BMI), 140, 174, 214-215, 230, 243, 256 Body water deficits, 287 Bone mineral content (BMC), 273 Borg scale, 288 Brain's reward system, 87 Brainstorming sessions, 71 Breakfast in the Classroom (BIC), 256

С

Calcium, 201-202 California Endowment's Healthy Eating, Active Communities Program, 404 Campylobacter, 303, 415-417, 419 Candidate nutrients, 23 Cardiodiabesity, 5 Cardiometabolic risk, 389-391 Cardiovascular disease (CVD), 5 mortality rates, 138 nutritional guidelines AHA guidelines (see American Heart Association (AHA)) consensus statements, 138 DASH diet, 139 dietary patterns, 139 implementation, 139-140 periodontitis, 199 Cardiovascular Health Integrated Lifestyle Diet (CHILD), 241 Cardiovascular risk factors behavioral factors diet, 232 environmental factors, 233 physical activity, 232 screen time, 232 sleep, 232 childhood obesity, intervention of comprehensive multidisciplinary intervention, 235 prevention plus, 233-234 structured weight management, 234-235 tertiary care intervention, 235 dyslipidemias, 239-241 hypertension, 242-244 ideal cardiovascular health, 230 pediatric dyslipidemias, 241-242 pediatric obesity assessment of, 231 health problems, 231-232

prevention, recommended diet physical activity, 238 screen time, 239 sugar-sweetened beverages, 238 USDA Dietary Guidelines, 236-237 USDA Plate Model, 237-238 Centers for Disease Control and Prevention (CDC), 214, 231, 259, 298, 398, 413 Chewing gum, 196 Childhood Determinants of Adult Health (CDAH), 230 Childhood obesity comorbidities, 215-216 critical weight gain, 221 intervention of comprehensive multidisciplinary intervention, 235 prevention plus, 233-234 structured weight management, 234-235 tertiary care intervention, 235 measurement guidelines and methodologies, 214-215 social marketing method, 222-223 treatment strategies history, 216-218 primary prevention, 219 secondary prevention, 219-220 tertiary prevention, 220-222 Child Nutrition Act, 252 ChooseMyPlate, 35-36, 409, 410 Chronic hypertension (cHTN), 307 Chronic obstructive pulmonary disease (COPD), 59 Chylomicrons, 239 Clostridium C. botulinum, 417 C. perfringens, 417 COACH approach. See Curiosity, openness, appreciation, compassion, honesty (COACH) Coefficient of variation (CV), 24-25 Cognitive behavioral therapy (CBT), 94 Community Eligibility Provision (CEP), 253, 256 Compassion, 63-64 'Complicit' masculinities, 314 Continuous glucose monitoring (CGM), 166 Cookbooks, 76 Coordinated School Health (CSH) model, 259 Coronary artery disease (CAD), 240 Coronary heart disease (CHD), 5 C-reactive protein (CRP), 200 Curiosity, openness, appreciation, compassion, honesty (COACH), 53, 54, 78 appreciation, 61-63 chronic conditions, 59 compassion, 63-64 connection, 58, 59 curiosity, 60-61 health and wellness coaching, 59-60 honesty, 64-65 motivation, 67 openness, 61 CVD. See Cardiovascular disease (CVD) Cystic fibrosis (CF), 126

425

D Daily Va

Daily Value (DV), 37-38 DASH. See Dietary Approaches to Stop Hypertension (DASH) Dean Ornish's Spectrum diet, 56 Deficiency disorders, 15, 16 Dehydration, 16 De novo lipogenesis process, 373, 379 Dental caries bacteria, 195 definition, 194 diet quality, 196-197 nutrition counseling, 204 oil pulling, 194–195 oral hygiene, maintenance of, 194 S-ECC, children with, 195 sugar alcohols, 196 sugar intake, restriction of, 195 Dental disease dental caries (see Dental caries) indirect and direct costs, 194 periodontitis (see Periodontal diseases) Depression, 360 Detox diet, 56 Diabetes Control and Complications Trial (DCCT), 158 Diabetes mellitus, 5 bariatric surgery, 164 blood pressure goals, 157, 158 diagnostic criteria, 153 education/counseling and support, 166 glycemic control, recommendation for, 157, 158 monitoring, 166 nutrition therapy ADA evidence ratings, 163-164 blood glucose correction insulin factors, 160 carbohydrate intake, 160-161 effectiveness of, 158-159 fats. 161-162 glycemic index, 161 goals of, 153 insulin-to-carbohydrate ratios, 160 macronutrient percentages, 159 protein, 161 AND recommendations ratings, 163-164 weight loss interventions, 162 weight loss medications, 162, 164, 165 periodontitis, 198-199 pharmacological therapy, 166-167 physical activity, 164 exercise precautions, 165-166 recommendations, 165 prevalence of, 153 Diabetes Prevention Program (DPP), 154 Diabetes self-management education (DSME), 159, 166 Diabetes self-management support (DSMS), 159, 166 Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), 276 Dietary acculturation model, 340

Dietary Approaches to Stop Hypertension (DASH), 32, 33, 241, 243, 307, 362 cardiovascular risk reduction, 139 weight loss, 179 Dietary Guidelines Advisory Committee, 361 Dietary Guidelines for Americans (DGAs), 14, 22, 31-33, 241, 252, 370, 400 Dietary Guidelines Scientific Advisory Committee's (DGAC) report, 32 Dietary intake assessment of dietary recommendations, 18 dietary supplement use, 18 **FFOs**, 18 food records and 24 h dietary recalls, 17-18 limitations, 19 SQFF questionnaires, 18 use of, 19-20 dietary recommendations world war I, 14 DRI (see Dietary Reference Intakes (DRIs)) nutritional status, component of, 15 Dietary Reference Intakes (DRIs), 14, 300 adequate intake, 22-23, 25 AMDR, 26 chronic disease, prevention of, 27-28 criteria, 23-24 dietary risk assessment, 26 EAR, 22-25 excessive intakes, 28 exportability of, 29-30 individuals vs. populations, recommendations, 28-29 RDA, 22-24 recent report on, 27 revision, 30-31 suggestions and improvements, 30 upper intake level, 22-23, 25-26 upper reference level, 22-23 uses of, 31 Dietary status limitations, 19 methods, 17-18 use of, 19-20 Dietary supplement, 18 Dietitians, 121 Directly to consumers (DTC) testing, 129-130 Disinhibition, 88-89 Docosahexaenoic acid (DHA), 200, 304 Dual-energy x-ray absorptiometry (DEXA), 215 Dublin Standard Table of Heights and Weights, 217

Е

Early Childhood Caries (ECC), 195, 197 Eating disorders, 197 EFSA. *See* European Food Safety Authority (EFSA) Eicosapentaenoic acid (EPA), 200 Empathy, 63 'Emphasised femininities,' 316 Empty calories, 106, 107 Enteral nutrition (EN), 305 Environmental Protection Agency (EPA), 304 Epigenetics, 120 Escherichia coli, 303, 419 Estimated average requirement (EAR), 22–25, 31 Estimated Energy Requirement (EER), 235 Estimated fetal weight (EFW), 299 Ethical, legal, and social issues (ELSI), 119 European Food Safety Authority (EFSA), 283, 370 Evidence Analysis Library (EAL), 233 Examine, X-ray, plan, explain, repeat and review, tell and sell (EXPERT) approach, 58–59

F

Familial Combined Hyperlipidemia (FCHL), 240 Familial hypercholesterolemia (FH), 240 Familial hypertriglyceridemia (FHTG), 240 FDA. See Food and Drug Administration (FDA) Federal Trade Commission (FTC), 183 FFVP. See Fresh Fruit and Vegetable Program (FFVP) First-line pharmacotherapy, 305 Fish, 144-145 Fit Fans, 320 Flexitarian diet, 185, 186 Fluoride, 201 Food Allergen Labeling and Consumer Protection Act (FALCPA), 40 Food and Drug Administration (FDA), 43, 220, 370 Food and Nutrition Board (FNB), 388 Food-borne disease, 16 Foodborne Diseases Active Surveillance Network (FoodNet) data, 414 Foodborne illness food safety incidence of, 414-416 pathogens and common food sources, 417-419 food sources, 418-419 Food frequency questionnaire (FFQ), 18, 52 Food records, 17-18 Food Research and Action Center (FRAC), 256, 261 Food safety CDC, 413 education and practices, 419-420 foodborne illness incidence of, 414-416 pathogens and common food sources, 417-419 healthy pregnancy foodborne pathogens, 303 lead, 304 methylmercury, 304 safe food-handling responsibilities, 417, 419 Food Safety Modernization Act (FSMA), 417 Football Fans in Training, 320 FRAC. See Food Research and Action Center (FRAC) Framingham Children's Study, 244 Fresh Fruit and Vegetable Program (FFVP), 252, 256 Front-of-pack labeling, 42 Fructose

composition of, 371 metabolism, 372–374 Functional magnetic resonance imaging (fMRI), 380

G

Gastro-esophageal reflux (GERD), 306 Gay masculinities, 314 GDM. See Gestational diabetes (GDM) Genetically modified organisms (GMO), 43 Genetic Metabolic Dietitians International (GMDI), 129 Genetic testing Academy of Nutrition and Dietetics, 130-131 DTC testing, 129-130 genetic metabolic dietitians, 126, 128 newborn screening, 126-128 phenylketonuria, 126, 128-129 Genome wide association studies (GWAS), 132 Genomic medicine, 120 Gestational diabetes (GDM), 299, 306-307 Gluten-free labeling, 44 Glycemic control, 157, 158 Glycemic index (GI), 161 Goals, reality, options, way (GROW) model, 73 Grant funding, 258 Green tea, 204

H

Health books, 76 2010 Health Care Reform Law, 405 Health Eating Index, 402, 403 Healthy eating childhood dietary habits, 398 disparities/inequities, 400-401 environmental factors food vendors, 403-404 full-service grocery stores, 401-402 restaurants, 402-403 retail food stores, 402 food losses, 408-409 food subsidy, 408 food taxation, 406-408 health behavior, 399-400 healthy food policy food labels, 405 full-service food stories, geographic availability of, 404-405 restaurant menus, 405-406 school. 404 healthy foods, availability and affordability of, 399 morbidity and obesity, 398-399 recommendations sales promotion, grocery stores, 409-410 serving sizes, 409 Healthy Eating Index (HEI), 14, 36-37, 196-197, 363 Healthy food policy food labels, 405

full-service food stories, geographic availability of, 404-405 restaurant menus, 405-406 school, 404 Healthy, Hunger-Free Kids Act of 2010 (HHFKA), 253-255, 258, 261 Healthy People 2020, 14-15, 45 Healthy US Style Eating Pattern (HUSEP), 179, 180 Hedonic eating, 174 Hegemonic masculinities, 314 Hepatitis A, 417 HHFKA. See Healthy, Hunger-Free Kids Act of 2010 (HHFKA) High-density lipoprotein (HDL), 141, 230, 239, 271 High fructose corn syrup (HFCS), 370-374 Hispanics acculturation process, 333-335 culture characteristics, 332 collectivistic culture, 331 communication, 331 health care, 332 relationship-orientation, 331 time orientation, 331 dietary behavior acculturation, 339 attributes, 337, 338 nutrition transition, 337-338 western diet, 339 dietary intake alcohol consumption, 341 dairy, 339-340 eating outside, 341 fruits and vegetables, 339 processed foods, meats and sugar, 340-341 SSB, 341 environmental factors, 342 health, Hispanic population diabetes, 334 healthcare access and health literacy, 336 heart disease, 336 Hispanic paradox, 336-337 hypertension, 336 overweight and obesity, 335-336 prevalent cancers, 336 Latin America, 337, 338 nutritional considerations and practical implications dietary behaviors, assessing and addressing, 343-344 evidence-based dietary interventions, 344-348 psychosocial factors, 341-342 USA, Hispanic population in, 329-330 HMR Program, 184-186 Home Food Inventory (HFI), 91-92 Homeostatic system, 87, 174 Homocysteine (tHcy), 21 Honesty, 64-65 Host modulatory therapy (HMT), 200

Human Genome Education Model (HuGEM) Project, 123 Human Genome Project (HGP), 118-119 Hydration over hydration, 291 water balance healthy lifestyle, 289-291 recreational activity, 288-289 status and performance, 285-288 25 Hydroxy vitamin D (25 OH D), 21 Hyperemesis gravidarum (HG), 305 Hypertension intervention, approaches to DASH dietary pattern, 243-244 dietary sodium, 244 weight loss, overweight, 243 pediatric hypertension, 242, 243 pre-hypertension, 242 primary/essential hypertension, 242 secondary hypertension, 242 Hypohydration, 284-286, 289

I

Imbalances, 16 Institute of Medicine (IOM), 234, 253, 274, 299, 370, 390 International Diabetes Federation (IDF), 153 Intrauterine growth restriction (retardation) (IUGR), 307

J

Jefferson Scale of Empathy, 63 Jenny Craig, 183, 184 Joint National Commission VII (JNC VII), 141–142 Junk food, 107

L

Laparoscopic sleeve gastrectomy (LSG), 221 LDL. *See* Low-density lipoprotein (LDL) Lifestyle medicine, 4 Lipoprotein metabolism, 239–241 *Listeria*, 414–417, 419 Listeria infection, 303, 414 *Listeria monocytogenes*, 303 Low-density lipoprotein (LDL), 141, 271 Low-density lipoprotein receptor (LDLR), 239

М

Magnesium, 202–203 Malnutrition deficiency disorders, 15, 16 food intake alterations, 15 form and cause of, 15–17 nutritional status, indicators of, 15, 17 'Marginalised' masculinities, 314 'Masculine foods,' 317 Masculine stereotypes, 313 Massachusetts Modifying Eating and Lifestyles at School Study (MEALS), 255 Mayo Clinic diet, 56 Medical nutrition therapy (MNT), 121, 158, 178, 194, 235 Mediterranean diet (MeDiet), 55, 56, 156, 179, 180, 185, 186.241 Medium chain triglyceride (MCT), 242 Mendelian diseases, 118 Men's health dieting and slimming, 318-320 food and health, 315–317 gender and health, 313-315 masculinity, 312-313 promoting nutritional health, 320-323 Metabolic syndrome (MetS), 378-379, 389 Metabolomics, 121 Methylenetetrahydrofolate reductase (MTHFR), 124 3-Methyl-L-histidine, 21 Methylmalonic acid (MMA), 21-22 Methylmercury, 304 Mifflin-St. Jeor equation, 178 Mindful eating, 94 Minnesota Coronary Experiment, 391, 392 MNT. See Medical nutrition therapy (MNT) Motivation, 53, 54, 320 appreciative inquiry, 68 extrinsic motivators, 67 intrinsic motivation, 67 motivational interviewing, 65–67 Motivational Interviewing (MI) acceptance, 65 change talk, 66 collaboration, 65 compassion, 65 elicit-provide-elicit model, 66-67 evocation, 65, 66 **MINT. 67** OARS framework affirmations, 65 open-ended questions, 65 reflections, 65-66 summaries, 66 self-determination theory, 67 Motivational Interviewing Network of Trainers (MINT), 67 Motivational interviewing techniques, 235 Motivators, obstacles, strategies, strengths (MOSS), 60 Mycotoxins, 417 Myocardial infarction (MI), 199 MyPlate, 14, 34-36 MyPlate for Older Adults, 356, 358 MyPyramid, 35

N

NAFLD. See Non-alcoholic fatty liver disease (NAFLD) National Cholesterol Education Program guidelines (NCEP), 378 National Coalition for Health Professional Education in Genetics (NCHPEG), 122, 124

Index

National Health and Nutrition Examination Survey (NHANES), 28, 230, 269 National Health Interview Survey and Teenage Attitudes and Practice Survey, 400 National Heart, Lung and Blood Institute (NHLBI), 231.233 National Institutes of Health, 217 National School Lunch Act, 252 National School Lunch Program (NSLP), 252, 255 Natural Resources Defense Council, 408 Neural tube defects (NTD), 300 Newborn screening (NBS), 126-128 Newborn Screening Saves Lives Act, 126 NHANES. See National Health and Nutrition Examination Survey (NHANES) Non-alcoholic fatty liver disease (NAFLD), 370, 379 Norovirus, 417 Nutrient labeling allergen labeling, 40, 42 "certified organic" label, 42-43 front-of-pack labeling, 42 gluten-free, 44 health claims, 39-42 ingredient labeling, 37 menu labeling, 42 natural, 44 non-GMO labels, 43 nutrient content claims, 39 Nutrition Facts labels, 37-39 structure-function claims, 39 supermarket icons and scoring systems, 44-45 whole grain labeling, 44 Nutrisystem, 184-186 Nutrition challenge, 4 individual behavior, 6-7 community food influence, 8-9 family food influences, 7-8 individual influences, 7 macro public environment, 9 mortality, risk factors, 5 non-nutritive substances, healthy pregnancy alcohol, 304-305 artificial sweeteners, 305 caffeine, 305 nutritional guidelines, implementation of, 9-10 nutritional status assessment, 15 biomarkers, 20-22 dietary intake assessment, 17-20 health status, component of, 15 malnutrition, 15-17 nutrition-related complaints, healthy pregnancy constipation, 306 GERD, 306 nausea and vomiting, 305-306 scientific organizations, guidelines, 6 Nutritional epigenomics, 120 Nutritional genomics

DNA, discovery of, 118 ELSI, 119 genetic testing Academy of Nutrition and Dietetics, 130-131 DTC testing, 129-130 genetic metabolic dietitians, 126, 128 newborn screening, 126-128 phenylketonuria, 126, 128-129 Human Genome Project, 118-119 nutrition and dietetics education, 122-124 practice, 124-125 nutritionists and dietitians, 121 organization of health professionals, 122 research, 131-132 SNPs. 118 terminology and definitions, 119-121 Nutrition counseling diet changes, 52 goal setting theory for businesses, 73 GROW model, 73 health care provider, 77 healthy factors, 52 healthy habits, 76 logs, diaries, and books, 76 messages, 53, 54 challenges, 56 healthy eating patterns, 55, 78 medically oriented diets, 56 Mediterranean diet, 55, 56 popular diets, 56 portion control, 56, 75 serving size charts, 56-57 "shape the path," 55, 77 water consumption, 57 "wholesome foods in sensible combinations," 57 messenger advising, 57 agreeing, 58 arranging, 58 assessing, 57 assisting, 58 COACH approach, 53, 54, 58-65, 78 collaborative attitude, 58 EXPERT approach, 58-59 motivation, 53, 54 appreciative inquiry, 68 extrinsic motivators, 67 intrinsic motivation, 67 motivational interviewing, 65-67 SMART goals, 53, 74-75 stages by principles and processes of change action, 71-72 contemplation, 70-71 maintenance, 72 precontemplation, 68-70 preparation, 71 Tiny Steps, 54 Transtheoretical Model of Change, 68

Nutrition diary, 76 Nutrition Facts label, 37-39, 300 Nutritionists, 121, 123, 125, 176 Nutrition prescription, 234-235 Nutrition research association/causation, 112 averages vs. anecdotes, 112 bias, 110 causal inference, 108-109 conclusions vs. decisions, 111-112 confounding, 111 extrapolation, 113 hierarchy of evidence, 108-109 interest vs. measured/administered, exposure/outcome of. 109-110 Lanarkshire milk experiment, 110 logical fallacies, 113-115 nutrition science lemon market, 115 oversimplification of complex topics empty calories, 106, 107 junk food, 107 nutritive value, 106-107 whole vs. processed foods, 107-108 self-reported energy intake, 110 words with different definitions, 104 cholesterol, reduction of, 105 obesity as an outcome, 105-106 snacks, breakfast, and meals, 105 Nutrition Therapy Practice Guidelines (NTPG), 158 Nutritive value, 106-107

0

Obesity, 5, 16 and all-cause mortality, 174 behavior change strategies cognitive factors, 182 problem solving, 182 self-monitoring, 181-182 BMI, 174 cause of, 173, 174 chronic health conditions, risk of, 174 commercial weight loss programs AHA/ACC/TOS practice guidelines, 185, 186 Atkins Diet, 184, 185 Biggest Loser Diet, 184, 185 Flexitarian Diet, 185, 186 HMR Program, 184-186 Jenny Craig, 183, 184 Mediterranean Diet, 185, 186 Nutrisystem, 184-186 popular diets, 183 Raw Food Diet, 184-186 Slim Fast Diet, 185, 186 Vegan Diet, 185, 186 Volumetrics Diet, 184-186 Weight Watchers, 183, 184 definition, 174 diet and weight loss

DASH diet, 179 HUSEP, 179, 180 length of treatment, 180-181 meal replacements/structured meal plans, 179, 180 medical nutrition therapy, 178 Mediterranean diet, 179, 180 Mifflin-St. Jeor equation, 178 multidisciplinary approach, 181 weight cycling, 177-178 food choice clinical implications, 177 cost and convenience, 176 environmental cues, 175 environmental factors, 175 food availability and accessibility, 175-176 hedonic eating, 174 homeostatic eating, 174 metabolic factors, 175 portion size, 176 social and media environment, 176-177 nutrition weight loss myths "drinking more water can aid in weight loss," 186 "eating after 10 pm will cause weight gain," 187 "eat small meals more frequently," 187 "skipping breakfast causes weight gain," 187 periodontitis, 199 prevalence, 174 Office of Dietary Supplements (ODS), 300 OGTT. See Oral glucose tolerance test (OGTT) Oil pulling, 194-195 Older adults chronic diseases cancer. 362-363 cardiovascular disease, 361 dentition and associated senses, 361 glucose intolerance and Type 2 diabetes, 362 hypertension, 362 immune function, 362 osteoporosis, 361-362 dietary considerations food fads and nutrient supplements, 360 physiological changes, 356-358 social factors, 359-360 taste and smell, 357, 359 vision, dexterity and mobility, 359 dietary guidance, 363 RDA, 356 Omega-3 fatty acids, 200 Open-ended questions, 65 Oral glucose tolerance test (OGTT), 200, 307 Oral health, 206-207 Call to Action to Promote Oral Health, goals of, 193 cariogenic and anticariogenic foods, 195-196 dental caries (see Dental caries) dietary fiber, 200-201 eating disorders, 197 fluoride, 201 lifestyle factors, 205-206

micronutrients calcium and vitamin D, 201–202 functional foods, 204 magnesium, 202–203 polyphenols, 203–204 probiotics, 203 vitamin C, 203 nutritional deficiencies, 197 periodontal diseases (*see* Periodontal diseases) polypharmacy, 206 tooth loss and nutrition, 197–198 Organic food products, 43 Orlistat, 220, 221

P

Parenteral nutrition (PN), 306 Pathogenic bacteria, 419 Patient Protection and Affordable Care Act, 402 Pediatric obesity cardiovascular risk factors assessment of, 231 health problems, 231-232 comorbidities, 218 physical activity, 232 Periodontal diseases calcium and vitamin D. 201-202 cardiovascular disease, 199 diabetes, 198-199 green tea, 204 magnesium, 202-203 nutritional deficiencies, risk for, 194 nutrition counseling, 204-205 obesity, 199 omega-3 fatty acids, 200 risk factors, 198 Personalized nutrition (PN), 120, 121, 125 Pharmacogenomics, 120 Phenylketonuria (PKU), 126, 128-129 Physical activity cardiovascular risk reduction, 142 diabetes mellitus, 164 exercise precautions, 165-166 recommendations, 165 hydration over-hydration, 291 recreational activity, 288-289 status and performance, 285-288 occupational activity, 284, 285 Physical Activity Guidelines for Americans, 142 Plant toxins, 417 Polypharmacy, 206 Polyphenols, 203-204 'Pop' psychology, 312 Popular diets, 183 Portion control, 56-57, 75 Portion-size measurement aids (PSMAs), 93 Post-exercise rehydration strategy, 287 Prader-Willi syndrome (PWS), 128

Prediabetes bariatric surgery, 156-157 diagnostic criteria, 153 lifestyle interventions, 157 nutrition therapy, 157 alcohol, 156 carbohydrate and dietary fats, 155 effectiveness of, 154 goals of, 155 Mediterranean diets, 156 vitamin D, 156 weight loss interventions, 155 whole grains and fiber, 155 physical activity, 156 Pregnancy calorie and protein needs, 300 CDC, 298 complications GDM, 306-307 hypertensive disorders, 307 food safety foodborne pathogens, 303 lead, 304 methylmercury, 304 gestational weight gain, 299 micronutrients folate, 300-301 iodine, 301 iron, 301-302 non-nutritive substances alcohol, 304-305 artificial sweeteners, 305 caffeine, 305 nutrition-related complaints constipation, 306 GERD, 306 nausea and vomiting, 305-306 obesity, 299 preconception, 298 twins/more pregnancy, 302 vegans, 302-303 Pregnancy-induced hypertension (PIH) disorders, 299 Premier League Fitness, 320 Preventing Obesity Without Eating like a Rabbit (POWER) project, 320 Prions, 417 Probiotics, 203 Processed food, 107-108 Protein calorie malnutrition, 15, 16 Proteomics, 120, 121 Psychosocial and Environmental Dietary Questionnaire, 343

R

Randomized clinical trials (RTC), 391 Randomized controlled trials (RCTs), 59, 370 Rating of perceived effort (RPE), 286, 288–289 Raw food diet, 184–186 Recommended daily intakes (RDIs), 388 Recommended dietary allowances (RDAs), 22–24, 31, 269, 300, 356 Registered Dietitian Nutritionist (RDN), 234 Resting metabolic rate (RMR), 234 Restrictive dieting, 321 Risk-risk assessments, 26 Roadmap for National Nutrition Research, 46 Roux en Y gastric bypass (RYBG), 221

\mathbf{S}

"Salmon bias," 337 Salmonella, 303, 414, 417, 419 Saturated fat CHD death, 388 dietary SFA and CHD risk epidemiology and large clinical trials, 391-392 intervention trials, 389-391 Great Depression, 388 high-fat dairy consumption, 392 low-fat dairy consumption, 392 two World Wars, 388 USDA, 388 US Senate Committee, 388-389 Saturated fatty acids (SFA), 155 School Breakfast Program (SBP), 252 School Food Authorities (SFAs), 259 School Health Policies and Practices Study (SHPSS), 255 School meal programs childhood obesity, 256 competitive foods and beverages, 257 farm to school program initiatives, 257-258 food insecurity, 256 2010 HHFKA, 253 local wellness policies, 259 meal improvements and consumption patterns, 254-255 nutrition standards, 253 reimbursement rates and school food budgets, 258-259 resources for school food improvements, 259-260 school nutrition program standards, 253-254 time to eat, 259 trends, 261 USA, 252-253 Scientific Advisory Committee on Nutrition (SACN), 370 Scientific organizations, 6 Seafood, 144-145 Seafood toxins, 417 Sedentary behaviors, 220 Self-determination theory (SDT), 53, 67 Self-efficacy, 61-62, 182, 183 Self-monitoring of blood glucose (SMBG), 166 Self-reported energy intake (SREI), 110 Serving size charts, 56-57 Severe maternal morbidity (SMM), 298 Sex role theory (SRT), 313, 314 SFSP. See Summer Food Service Program (SFSP)

Shiga toxin-producing Escherichia coli (STEC) O157, 415 Sibutramine, 221 Significant scientific agreement (SSA), 39-40 Single nucleotide polymorphisms (SNPs), 118, 121 Slim fast diet, 185, 186 Small-for-gestational (SGA) infants, 299 Smarter Lunchroom Initiative, 255 SMART goals. See Specific, measurable, action-oriented, realistic, time-sensitive (SMART) goals Smart Snacks, 257, 258 Smoking, 52, 230, 337, 360, 406 SNAP. See Supplemental Nutrition Assistance Program (SNAP) Social modeling, 89-90 Socioeconomic status (SES), 319, 339, 401 'Somatic society,' 319 Special Milk Program, 252 Special Supplemental Nutrition Program for Women Infants, and Children (WIC), 111 Special Turku Risk Intervention Program (STRIP), 241 Specific, measurable, action-oriented, realistic, timesensitive (SMART) goals, 53, 74-75 SRT. See Sex role theory (SRT) SSBs. See Sugar-sweetened beverages (SSBs) Stages by principles and processes of change action, 71-72 contemplation, 70-71 maintenance, 72 precontemplation, 68-70 preparation, 71 Standard deviation (SD), 24-25, 214 Staphylococcus aureus, 417, 419 Starvation, 16, 197, 206 Students Today Achieving Results for Tomorrow program, 404 'Subordinated' masculinities, 314 Sucrose composition of, 371 metabolism, 372-374 Sugars. See Added sugars and health Sugar-sweetened beverages (SSBs), 155, 195, 232, 269, 341, 370, 381 Summer Food Service Program (SFSP), 252, 256 Super Tracker app, 409 Supplemental Nutrition Assistance Program (SNAP), 111-112 Systolic Blood Pressure Intervention Trial (SPRINT Study), 142

Т

Tanita BF-689, 215 Tapeworms, 417 Tertiary Care Intervention, 220 Theory of Planned Behavior (TPB), 182–183 Tiny Steps, 54 Tolerable Upper intake (UL), 302 Tooth loss, 197 Toxicity, 16, 23, 26, 108, 202

Index

Toxoplasma gondii, 303, 417 Traffic Light Program (TLP), 219 Trichinella spiralis, 417 Tufts University Project Lunch Box Study, 255 Type 1 diabetes (T1D) ADA evidence ratings, 163-164 carbohydrate intake, 160-161 macronutrient percentages, 159 medical nutrition therapy, 158 pharmacological therapy, 166-167 physical activity, 165 prevalence of, 153 AND recommendations ratings, 163-164 Type 2 diabetes (T2D) bariatric surgery, 157, 164 lifestyle interventions, 157 nutrition therapy ADA evidence ratings, 163-164 alcohol, 156 carbohydrate and dietary fats, 155 carbohydrate intake, 160-161 macronutrient percentages, 159 medical nutrition therapy, 158 Mediterranean diets, 156 AND recommendations ratings, 163-164 vitamin D, 156 weight loss interventions, 162 weight loss medications, 162, 164, 165 whole grains and fiber, 155 periodontal diseases, 198 pharmacological therapy, 167 physical activity, 156, 165 prevalence of, 153

U

United States Department of Agriculture (USDA), 252, 256 ChooseMyPlate, 35–36 empty calories, definition of, 106 Farm to School Census, 257–258 Food Guide Pyramid, 34–35 Food Pattern, 33 MyPlate, 356 MyPyramid, 35 National Organic Program, 43 SuperTracker website, 300 United States Preventive Services Task Force (USPSTF), 301–302 Upper reference level (UL), 22–23, 25–26 US Department of Agriculture, Food and Nutrition Services (USDA-FNS), 252 US Food Administration (USFA), 388 US Preventive Services Task Force, 218

V

Vegan Diet, 185, 186 Vertical jumping ability, 286 Very low-density lipoprotein (VLDL), 239 *Vibrio*, 415–417 Vitamin B6 plus doxylamine, 305 Vitamin C, 203 Vitamin D, 201–202 Vitamin D deficiency, 15, 16 Vitamin E supplementation, 362 Volumetrics diet, 184–186

W

Water balance ANP, 283 AVP. 283 euhydration, 282 extracellular compartments, 282 hydration healthy lifestyle, 289-291 recreational activity, 288-289 status and performance, 285-288 intracellular compartments, 282 oxidative metabolism, 282 plasma osmolality and blood volume, 283 sweating and water turnover, 283-285 urine production, 283 Water intoxication, 291 Weighing spring scales, 217 Weight cycling, 177-178 Weight loss interventions (WLI), 162 Weight Watchers, 183, 184 Whole-food, 107-108 Whole Grains Council, 44 Whole Grain Stamp, 44 World Cancer Research Foundation, 30 World Health Organization (WHO), 195, 370, 381, 419

Y

Yersinia, 415, 417