Burkitt's Lymphoma

A Medical Dictionary, Bibliography,
and Annotated Research Guide to
Internet References

James N. Parker, M.D.
and Philip M. Parker, Ph.D., Editors

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The collective knowledge generated from academic and applied research summarized in various references has been critical in the creation of this book which is best viewed as a comprehensive compilation and collection of information prepared by various official agencies which produce publications on Burkitt's lymphoma. Books in this series draw from various agencies and institutions associated with the United States Department of Health and Human Services, and in particular, the Office of the Secretary of Health and Human Services (OS), the Administration for Children and Families (ACF), the Administration on Aging (AOA), the Agency for Healthcare Research and Quality (AHRQ), the Agency for Toxic Substances and Disease Registry (ATSDR), the Centers for Disease Control and Prevention (CDC), the Food and Drug Administration (FDA), the Healthcare Financing Administration (HCFA), the Health Resources and Services Administration (HRSA), the Indian Health Service (IHS), the institutions of the National Institutes of Health (NIH), the Program Support Center (PSC), and the Substance Abuse and Mental Health Services Administration (SAMHSA). In addition to these sources, information gathered from the National Library of Medicine, the United States Patent Office, the European Union, and their related organizations has been invaluable in the creation of this book. Some of the work represented was financially supported by the Research and Development Committee at INSEAD. This support is gratefully acknowledged. Finally, special thanks are owed to Tiffany Freeman for her excellent editorial support.

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FORWARD

In March 2001, the National Institutes of Health issued the following warning: "The number of Web sites offering health-related resources grows every day. Many sites provide valuable information, while others may have information that is unreliable or misleading."[1] Furthermore, because of the rapid increase in Internet-based information, many hours can be wasted searching, selecting, and printing. Since only the smallest fraction of information dealing with Burkitt's lymphoma is indexed in search engines, such as **www.google.com** or others, a non-systematic approach to Internet research can be not only time consuming, but also incomplete. This book was created for medical professionals, students, and members of the general public who want to know as much as possible about Burkitt's lymphoma, using the most advanced research tools available and spending the least amount of time doing so.

In addition to offering a structured and comprehensive bibliography, the pages that follow will tell you where and how to find reliable information covering virtually all topics related to Burkitt's lymphoma, from the essentials to the most advanced areas of research. Public, academic, government, and peer-reviewed research studies are emphasized. Various abstracts are reproduced to give you some of the latest official information available to date on Burkitt's lymphoma. Abundant guidance is given on how to obtain free-of-charge primary research results via the Internet. **While this book focuses on the field of medicine, when some sources provide access to non-medical information relating to Burkitt's lymphoma, these are noted in the text.**

E-book and electronic versions of this book are fully interactive with each of the Internet sites mentioned (clicking on a hyperlink automatically opens your browser to the site indicated). If you are using the hard copy version of this book, you can access a cited Web site by typing the provided Web address directly into your Internet browser. You may find it useful to refer to synonyms or related terms when accessing these Internet databases. **NOTE:** At the time of publication, the Web addresses were functional. However, some links may fail due to URL address changes, which is a common occurrence on the Internet.

For readers unfamiliar with the Internet, detailed instructions are offered on how to access electronic resources. For readers unfamiliar with medical terminology, a comprehensive glossary is provided. For readers without access to Internet resources, a directory of medical libraries, that have or can locate references cited here, is given. We hope these resources will prove useful to the widest possible audience seeking information on Burkitt's lymphoma.

The Editors

[1] From the NIH, National Cancer Institute (NCI): **http://www.cancer.gov/cancerinfo/ten-things-to-know**.

CHAPTER 1. STUDIES ON BURKITT'S LYMPHOMA

Overview

In this chapter, we will show you how to locate peer-reviewed references and studies on Burkitt's lymphoma.

The Combined Health Information Database

The Combined Health Information Database summarizes studies across numerous federal agencies. To limit your investigation to research studies and Burkitt's lymphoma, you will need to use the advanced search options. First, go to **http://chid.nih.gov/index.html**. From there, select the "Detailed Search" option (or go directly to that page with the following hyperlink: **http://chid.nih.gov/detail/detail.html**). The trick in extracting studies is found in the drop boxes at the bottom of the search page where "You may refine your search by." Select the dates and language you prefer, and the format option "Journal Article." At the top of the search form, select the number of records you would like to see (we recommend 100) and check the box to display "whole records." We recommend that you type "Burkitt's lymphoma" (or synonyms) into the "For these words:" box. Consider using the option "anywhere in record" to make your search as broad as possible. If you want to limit the search to only a particular field, such as the title of the journal, then select this option in the "Search in these fields" drop box. The following is what you can expect from this type of search:

- **Effects of Cancer Therapy on Dental and Maxillofacial Development in Children: Report of Case**

  Source: Journal of Dentistry for Children. 67(3): 218-222. May-June 2000.

  Contact: Available from American Society of Dentistry for Children. John Hancock Center, 875 Michigan Avenue, Suite 4040, Chicago, IL 60611-1901. (312) 943-1244.

  Summary: This article reports the case of a child with cancer, in order to evaluate the long term effects of cancer therapy on dental and maxillofacial development and to determine how to maintain the oral function of this patient (and thus improve the quality of his daily life). The patient presented at five years of age in March 1987 to the periodontic department for the treatment of dental caries. One year prior, the child had

been diagnosed with **Burkitt's lymphoma** and received serial chemotherapy for nine months. In addition, the child received localized radiation therapy. The irradiated areas included the pituitary gland, maxilla, mandibular condyle, ramus and body, but not the anterior portion of the mandible. The authors report the child's dental findings and treatment through age twelve. Treatment was designed to preserve his teeth as long as possible, including oral hygiene instruction; caries prevention; and avoidance of excessive occlusal force and traumatic injury. In this case, the accumulated dosages of irradiation on the dentition caused irreversible damage. At the time of this article's publication, the patient is in his early puberty, and the treatment goal is to preserve his natural dentition and rehabilitate his normal jaw relations and functions. How long the teeth with shortened roots can be retained and what therapy should be used after loss of these teeth are yet to be determined. 6 figures. 1 table. 10 references.

Federally Funded Research on Burkitt's Lymphoma

The U.S. Government supports a variety of research studies relating to Burkitt's lymphoma. These studies are tracked by the Office of Extramural Research at the National Institutes of Health.[2] CRISP (Computerized Retrieval of Information on Scientific Projects) is a searchable database of federally funded biomedical research projects conducted at universities, hospitals, and other institutions.

Search the CRISP Web site at **http://crisp.cit.nih.gov/crisp/crisp_query.generate_screen**. You will have the option to perform targeted searches by various criteria, including geography, date, and topics related to Burkitt's lymphoma.

For most of the studies, the agencies reporting into CRISP provide summaries or abstracts. As opposed to clinical trial research using patients, many federally funded studies use animals or simulated models to explore Burkitt's lymphoma. The following is typical of the type of information found when searching the CRISP database for Burkitt's lymphoma:

- **Project Title: ANALYSIS OF POLYAMINE METABOLISM IN EBV LYMPHOMAGENESIS**

  Principal Investigator & Institution: Scott, Rona S.; Louisiana State Univ Hsc Shreveport P. O. Box 33932 Shreveport, La 71103

  Timing: Fiscal Year 2003; Project Start 30-SEP-2003; Project End 30-JUN-2008

  Summary: Epstein-Barr virus (EBV) is a human tumor virus discovered in **Burkitt's lymphoma** (BL) and subsequently linked to multiple cancers. In vitro, EBV infection confers on B lymphocytes an unlimited growth potential termed immortalization. The long-term goal of this research is to understand the mechanisms by which EBV causes cancer. Toward this end, our laboratory has developed a novel cell system in which EBV episomes can be eradicated from **Burkitt's lymphoma** cells by treatment with low-dose hydroxyurea. Burkitt cells "cured" of EBV lose their malignant growth phenotype despite retaining the hallmark t(8;14) in which c-myc is translocated into an immunoglobulin locus. Using gene arrays to compare global gene expression patterns in paired EBV-positive and EBV-negative BL clones, a 3-fold reduction in

[2] Healthcare projects are funded by the National Institutes of Health (NIH), Substance Abuse and Mental Health Services (SAMHSA), Health Resources and Services Administration (HRSA), Food and Drug Administration (FDA), Centers for Disease Control and Prevention (CDCP), Agency for Healthcare Research and Quality (AHRQ), and Office of Assistant Secretary of Health (OASH).

spermidine/spermine N1-acetyltransferase (SSAT) in EBV-positive (malignant) cells was among the very limited changes that distinguished these closely related clones. Because the translocation and subsequent deregulation of c-myc in BL result in overexpression of ornithine decarboxylase (an enzyme involved in biosynthesis of polyamines essential to cell growth), the finding that EBV infection downregulates SSAT (an enzyme involved in polyamine catabolism) formed the basis for our hypothesis: EBV infection manipulates the polyamine metabolic pathway to provide a growth advantage that not only favors viral persistence but also contributes to tumorigenesis in B cells. Specific aims to test this hypothesis are: 1) Quantify differences in polyamine catabolism between EBV-positive BL cells and their EBV-negative subclones by examining transcriptional and translational controls involved in SSAT regulation and by measuring polyamine levels; 2) Identify the type 1 latency viral gene product(s) that affect(s) SSAT expression, by stable transfection techniques; 3) Determine whether forced expression of SSAT reverses the malignant phenotype in EBV-positive cells, using a conditional expression SSAT construct or treatment with a polyamine analogue that is a potent inducer of SSAT.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ANTI VIRAL THERAPEUTIC FOR EBV MALIGNANCIES**

  Principal Investigator & Institution: Faller, Douglas V.; Director, Cancer Research Center; None; Boston University Medical Campus 715 Albany St, 560 Boston, Ma 02118

  Timing: Fiscal Year 2002; Project Start 07-FEB-2001; Project End 30-JUN-2004

  Summary: Epstein-Barr Virus is a common and worldwide pathogen. While exposure usually results in a self-limited lymphoproliferative syndrome, infectious mononucleosis, the virus is causative, or associated with, a number of malignancies. The latent virus is detected in 2 endemic tumors: 95% of African **Burkitt's lymphoma,** and 90-100% of nasopharyngeal carcinoma. Many B-lymphomas, some T-lymphomas, and approximately 50% of Hodgkin's lymphomas have also been found to contain latent EBV. 40% of lymphomas arising in AIDS, and nearly all lymphomas arising in transplant recipients (post-transplant-associated lymphoproliferative disease (PT-LPD) harbor EBV. PT-LPD is especially difficult to treat unless the immunosuppression can be reversed, and is typically refractory to radiation therapy and chemotherapy. Similar to herpes simplex virus and varicella-zoster virus, EBV encodes a thymidine kinase (TK) enzyme. In a rate-limiting step, the viral TK converts nucleoside analogues to their monophosphate form, eventually leading to premature termination of the nascent DNA and cell death. Latently-EBV-infected B-cells and epithelial cells, including tumor cells, do not express TK. We have found that exposure of these cells to the experimental drug Arginine Butyrate results in induction of TK expression. Preliminary in vitro studies demonstrated that induction of EBV-TK in patient-derived tumor cells by Arginine Butyrate is possible, and that these previously-resistant cells are rendered susceptible to Ganciclovir (GCV) therapy. We have years of clinical experience in the administration of Arginine Butyrate to adults and children in studies to induce fetal hemoglobin as therapy for sickle cell anemia and thalassemia. We hypothesized that treatment of patients with EBV- associated tumors with arginine butyrate (to induce the EBV-TK) and GCV (to eliminate EBV-TK expressing cells) might be an effective, nontoxic therapy. We have treated eight patients with Arginine Butyrate plus ganciclovir in an FDA-registered pilot study with documented responses in the majority of patients, and no adverse outcomes related to this regimen. Our Specific Aims are: (1) To determine if treatment with Arginine Butyrate plus Ganciclovir will result in clinical responses in a significant proportion of patients with EBV-associated lymphomas and

lymphoproliferative disease (LPD); (2) To determine toxicity or side effects of the combination therapy; and (3) To determine if tumor specimens and cell lines derived from patients demonstrate the same response to Arginine Butyrate and Ganciclovir (with respect to TK gene induction and synergistic susceptibility) as the EBV(+) cell lines we have studied to date.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ANTIGEN-INDUCED B LYMPHOCYTE DEVELOPMENT**

  Principal Investigator & Institution: Fearon, Douglas T.; Director; University of Cambridge Cambridge, England Cambridge,

  Timing: Fiscal Year 2002; Project Start 20-SEP-2002; Project End 30-JUN-2007

  Summary: (provided by applicant): With a long-term objective to understand the developmental stages of the B cell response to antigen, this research will focus on germinal center and memory B cells, which relate to the cardinal features of immunity, affinity maturation of the immune response and immunological memory. The specific aims are to determine: 1) the role of heparan sulfate/heparin in serving as a ligand for CD19 in the germinal center; 2) whether developmental down-regulation of SHP-1 in the germinal center B cell is required, and whether this permits signaling through a gamma-c cytokine receptor; 3) whether arrested terminal differentiation by BCL-6 is the basis for B cell memory; 4) whether signaling through a gamma-c, cytokine receptor is necessary to maintain memory B cells; and 5) whether B cells with dysregulated c-myc present as centroblast-like Burkitt lymphomas because of unique expression patterns of BCL-6 and SHP-l at this stage of development. For Aims 2-5, mice will be created in which transgenes can be inducibly expressed in a lineage-specific manner. A bacterially-derived transactivator transgene, rtTA, will be expressed only in B cells and will drive transcription of "target" transgenes in the presence of doxycycline. The target transgenes are SHP-1, dominant negative (DN) gamma-c, and wild-type and DN BCL6, all linked via an IRES to EGFP to permit visualization of transgene expression. With doubly transgenic, Fl mice, giving doxycycline will 1) up-regulate SHP-1 and DN gamma-c in germinal center B cells, allowing analysis of the role of low SHP-1 and cytokine receptor signaling in these cells; and 2) up-regulate DN gamma-c, and wild-type and DN BCL-6 in memory B cells, allowing analysis of the role of BCL-6 and cytokine receptor signaling in these cells. The understanding gained from these studies of normal B cell development will be applied to a murine model of Burkitt lymphoma to discover why translocation of c-myc at an early stage of B cell development leads to a B cell tumor at a much later stage.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ANTIVIRAL MEDIATED APOPTOSIS OF NON HODGKIN'S LYMPHOMA**

  Principal Investigator & Institution: Harrington, William J.; Professor; Medicine; University of Miami-Medical Box 248293 Coral Gables, Fl 33124

  Timing: Fiscal Year 2002; Project Start 01-APR-2000; Project End 31-MAR-2003

  Summary: (Applicant's Abstract) The applicant has found that certain high grade Herpes virus associated lymphomas are sensitive to anti-viral mediated apoptosis in vitro and in vivo. Epstein-Barr Virus positive **Burkitt's lymphoma** and Human Herpes Virus Type 8 related Primary Effusion Lymphomas undergo apoptosis when cultured in the presence of Azidothymidine (AZT) or AZT and Interferon Alpha (IFN Alpha). He has investigated the mechanisms by which this therapy causes apoptosis in these

lymphoma subtypes. He has found that incubation of **Burkitt's lymphoma** cells with AZT results in upregulation of CD95 and apoptosis. Primary Effusion Lymphoma requires Interferon Alpha to potentiate AZT mediated apoptosis. He has also found that Interferon Alpha induces the death receptor ligands, TRAIL and Fas Ligand in B cell lymphomas. In contrast to **Burkitt's lymphoma** and Primary Effusion Lymphoma, EBV positive large cell immunoblastic lymphomas and Epstein-Barr virus negative lymphomas were resistant to AZT and Interferon Alpha. These initial findings indicate that some lymphomas might be selectively sensitive to anti-viral therapy. In susceptible lymphomas, AZT and Interferon Alpha mediated apoptosis does not occur solely through Fas/Fas-Ligand interaction and likely involves activation of additional mechanisms of apoptosis. The applicant will investigate the role of viral and cellular pro- and anti-apoptotic proteins in blocking or facilitating AZT and Interferon Alpha induction of apoptosis in primary lymphoma specimens and cell lines developed from these tumors. A mechanism of inducing apoptosis in aggressive lymphomas would benefit patients with these diseases.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: B LYMPHOCYTES DIFFERENTIATION AND TRIGGERING**

  Principal Investigator & Institution: Huber, Brigitte T.; Professor; Pathology; Tufts University Boston Boston, Ma 02111

  Timing: Fiscal Year 2004; Project Start 01-APR-1978; Project End 30-APR-2009

  Summary: (provided by applicant): This competitive renewal application is based on the discovery that the env gene of HERV-K18 (Human Endogenous Retrovirus) encodes a superantigen, which is transcriptionally activated by EBV (Epstein-Barr Virus) and IFN-alpha (type I interferon). The working hypothesis is that the T cell stimulation elicited by the superantigen is not only essential for establishing life-long persistent infection with EBV in healthy individuals, but also plays a crucial role in EBV-associated diseases and malignancies. The following specific aims are proposed to test this model: I) The control of HERV-K18 expression will be defined. The role of CD21 engagement in the initiation of superantigen expression will be analyzed, based on the observation that IFNalpha and EBV infection lead to superantigen expression, both acting through CD21 on resting B cells. Furthermore, the role of EBV LMP-2A in sustained superantigen expression will be evaluated, based on the finding that this EBV latent gene product is sufficient to transactivate HERV-K18 env, leading to superantigen activity. II) HERV-K18 superantigen-reactive cells will be delineated in vivo and in vitro. For this purpose, an HLA.DR/HERV-K18 Env tetramer will be constructed to identify and stimulate superantigen-reactive T cells. The recent discovery of the murine TCR (T cell receptor) Vbeta specificity for the human superantigen will be exploited to define and map the TCR-superantigen interaction site. In addition, the role of CD48 in the superantigen-induced T cell activation will be tested, since EBV upregulates expression of this co-stimulatory molecule. III) The role of the HERV-K18 superantigen in EBV-infection will be determined, because the central dogma of this proposal is that the superantigen activity is required for the successful EBV life-cycle in the human host. In vitro and in vivo EBV-infection/ outgrowth/ persistent latency/lytic cycle/reactivation models will be used to address this aim. IV) The DNA of patients suffering from EBV-associated diseases will be typed for preferential expression of certain HERV-K18 alleles, compared to their respective healthy controls. This aim is based on the observation that the 3 HERV-K18 env alleles defined so far are unequally distributed in the Caucasian population and differ in their superantigen expression level. Collectively, these studies

will further the general understanding of how viruses exploit the immune system of their hosts to their own advantage.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: BLC AND BLR1 AND IMMUNE FUNCTION AND DYSFUNCTION**

  Principal Investigator & Institution: Cyster, Jason G.; Associate Professor; Microbiology and Immunology; University of California San Francisco 500 Parnassus Ave San Francisco, Ca 941222747

  Timing: Fiscal Year 2002; Project Start 01-APR-1999; Project End 31-MAR-2004

  Summary: Chemokines are well characterized as small chemotactic cytokines that recruit cells from the blood to sites of infection. Recent studies have uncovered a role for chemokines in cell trafficking through lymphoid tissues. In mice deficient in **Burkitt's Lymphoma** Receptor 1 (BLR1/CXC- R5), B cell follicles fail to form in spleen and Peyer's patches and inguinal lymph nodes do not develop. B-Lymphocyte Chemoattractant (BLC/BCA1), the only known ligand for BLR1, is expressed by follicular stromal cells in these tissues. How BLR1 and BLC function to organize cells in follicles is not understood. The long-term objective of this propose is to define the contribution of BLR1/BLC to cell migration and organization in both lymphoid and non-lymphoid tissues. The first of three aims seeks to determine if changes in BLR1 expression or BLC responsiveness contribute to the changes in B cell tropism that occur upon activation and differentiation. Similar studies will also be performed on T cells since T cell migration into follicles is essential for germinal center reactions and may also permit access to antigen trapped on follicular dendritic cells such as HIV in patients with AIDS. Aim 1 will also test if a splice variant of BLR1 in human macrophages is expressed in the mouse and will determine the BLC responsiveness of macrophage subsets. In Aim 2 the mouse BLC gene will be inactivated by gene targeting. BLC-deficient mice are important for reestablishing the role of BLC in follicular compartmentalization of B cells, for determining whether BLC is the only ligand for BLR1 and for characterizing whether BLC has functions in addition to a role in cell homing to follicles. The targeting construct will also introduce the green fluorescent protein gene (GFP) into the BLC locus to permit characterization of BLC expressing cells. In patients suffering chronic inflammatory diseases such as rheumatoid arthritis and diabetes, there are often large accumulations of B cells in the affected tissue. The third aim will explore whether BLC is expressed at sites of chronic inflammation in mouse models and, using the gene targeted mice, test to what extent BLC contributes to the inflammation. This aim will also test the effect of BLC expression to an ectopic site (the pancreatic islets) using a transgenic approach both to determine whether BLC is sufficient to promote follicle formation and to establish a system where the relationship between B cell accumulation and pathology can be studied.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: CHROMATIN REMODELING IN VIRAL TRANSFORMATION**

  Principal Investigator & Institution: Schubach, William H.; Medicine; University of Washington Grant & Contract Services Seattle, Wa 98105

  Timing: Fiscal Year 2002; Project Start 01-JUL-2000; Project End 30-JUN-2004

  Summary: (Adapted from the Investigator's abstract): The Epstein-Barr virus (EBV) protein, EBNA2, is essential for B cell immortalization which underlies the pathogenesis of EBV-related lymphomas in immunosuppressed patients. EBNA2 activates transcription of specific genes indirectly through its association with the ubiquitous

DNA binding protein, CBF1/RBP-Jk. A fraction of phosphorylated EBNA2 is associated with hSNF5/Ini1 in EBV infected B cells. hSNF5 is a component of the human SWI/SNF chromatin remodeling complex. Using chromatin immunoprecipitation, SWI/SNF components hSNF5 and BRG1 are found associated with specific EBNA2-responsive elements on an episomal construct and the cellular CD23 gene. This is the first demonstration of recruitment of the hSWI/SNF complex in vivo. The mechanism of EBNA2-associated SWI/SNF function will be investigated. Critical amino acids in EBNA2 will be identified that mediate the interaction with hSNF5, and mutants which fail to bind hSNF5 will be tested for the ability to activated transcription, alter chromatin structure, and induce cellular transformation. Components of the EBNA2-hSNF5 complex will be identified, and the possible function of the repressor EBNA3C will be studied.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: CONTROL OF C-MYC TRANSCRIPTION IN HIGH GRADE LYMPHOMA**

  Principal Investigator & Institution: Boxer, Linda M.; Associate Professor; Medicine; Stanford University Stanford, Ca 94305

  Timing: Fiscal Year 2002; Project Start 15-APR-1996; Project End 31-JAN-2006

  Summary: (Adapted from the applicant's abstract) We proposed to study the mechanism of activation of c-myc at a molecular level in human lymphoma tissue and in mouse model of the translocation. The goal is to reach a better understanding of the mechanisms of malignant transformation. 1. Completion of the characterization of the regulatory elements in the murine and human immunoglobulin heavy chain (IgH) enhancers that deregulate c-myc transcription. We will continue to identify the regulatory elements that are required for activation of c-myc, including increased transcription and P2 to P1 promoter shift. 2. Identification of the regions of the c-myc promoter that are required for the interaction with the IgH enhancers for maximun transcriptional activity and the promoter shift. Study of the mechanisms involved. 3. Determination of the mechanisms of transcriptional silencing of the normal c-myc allele in **Burkitt's lymphoma.** 4. Construction of a mouse model of the c-myc-IgH translocation. We will target the active sites of the IgH enhancers to the murine c-myc gene to recreate the Burkitt's translocation. 5.Development of strategies to interfere with c-myc transcription that is driven by the IgH enhancers. We have shown that several NF-kB sites are critical for deregulated c-myc expression, and we will first target the function of NF-kB.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: DNA SEQUENCES INVOLVED IN THE HEAVY CHAIN SWITCH**

  Principal Investigator & Institution: Dunnick, Wesley A.; Professor; Microbiology and Immunology; University of Michigan at Ann Arbor 3003 South State, Room 1040 Ann Arbor, Mi 481091274

  Timing: Fiscal Year 2002; Project Start 01-SEP-1984; Project End 31-JUL-2007

  Summary: (provided by applicant): The heavy chain switch is mediated by recombination event between switch (S) regions associated with the donor (usually mu) and recipient (gamma (g), epsilon (e), or alpha (a)) heavy chain genes. Aberrant switch recombination events lead to the chromosomal translocations found in **Burkitt's lymphoma,** AIDS-associated lymphoma, and perhaps other lymphoid tumors. Hence, understanding the heavy chain switch is important for both improving immune

responses to pathogens and to our understanding of the genesis of lymphoid tumors. Switch recombination is preceded by germline transcription through the S region and the constant region. Although switch recombination is understood to be a DNA deletion beginning and ending in S regions, almost nothing else is known about its mechanism. Switch recombination is also regulated in that it is directed by extracellular signals to one, or sometimes, two heavy chain genes. Cis-acting elements in the heavy chain locus must play a role in gene-specific switch recombination. To understand the mechanism of switch recombination, and to understand the cis elements that are important to both regulation and the recombination event itself, we have applied new technology to modify genes in a cloned version of the murine heavy chain locus. In the proposed experiments we will modify a specific heavy chain gene on a bacterial artificial chromosome with an assembled variable region and the entire heavy chain constant region locus, and test expression of various heavy chain genes in transgenic mice. We will test the role of a specific protein binding site or promoter regions, in general, for murine gamma heavy chain genes in both germline transcription and switch recombination (Aims 1 and 2). We will test how deletion or modification of the switch region changes switch recombination (Aim 3). We will examine the role of the orientation of switch sequences, and hence the role of the unusual complexes between germline transcripts and switch region DNA, in Aim 4.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: E2F TRANSCRIPTION FACTOR AND TUMOR SUPPRESSION**

  Principal Investigator & Institution: Chellappan, Srikumar P.; Associate Professor; Moffitt Cancer Center; University of South Florida 4202 E Fowler Ave Tampa, Fl 33620

  Timing: Fiscal Year 2002; Project Start 01-MAY-1994; Project End 31-JAN-2003

  Summary: (adapted from the investigator's abstract) The major focus on this proposal is to understand the mechanisms that regulate the function of the E2F transcription factor, with special emphasis on its role in tumor suppression and oncogenesis. It is clear that the retinoblastoma tumor suppressor protein exerts its growth inhibitory function at least in part through repressing the activity of E2F. Oncogenic events that disrupt the function of Rb result in the loss of the Rb-E2F interaction and increased E2F activity; further, over-expression of E2F1 itself can lead to oncogenic transformation. The experiments proposed in this application attempt to understand the biochemical mechanisms involved in regulating E2F function in response to extra-cellular stimuli. Though the steps involved in mitogenic signal transduction pathways have been elucidated, it is not clear how a signal received at the cell surface activates the cell cycle machinery. Their preliminary studies suggest that a vital signaling kinase, Raf-1, can interact with and inactivate Rb. Further, Jun Kinase (JNK) can repress E2F1 function. These observations directly link cell surface signaling with the cell cycle machinery. In this context, efforts will be made to elucidate how Jun Kinase (JNK1) regulates E2F activity and the functional consequences of such a regulation. Attempts will be made to identify the sites on E2F that is targeted by JNK1 and to evaluate whether mutations in these sites will affect the ability of E2F1 to transform cells and to induce apoptosis. Similarly, finer analysis will be conducted on the Raf-1-Rb interaction, and efforts will be made to characterize the steps involved in Raf-1 mediated repression of Rb. It will also be determined how DP1 - a dimerization partner of E2F1, is modulated by signaling pathways and what are the functional consequences of such a regulation. Finally, a novel E2F activity that is over-expressed in **Burkitt's lymphoma** cells will be characterized and its contribution to the oncogenic process will be assessed. It is hoped

that these studies will throw new light on hitherto unknown steps involved in tumor suppression and oncogenesis.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: EBNA1-SPECIFIC CD4+T HELPER 1 CELLS**

  Principal Investigator & Institution: Bickham, Kara; Lab/Cell Physiol & Immunology; Rockefeller University New York, Ny 100216399

  Timing: Fiscal Year 2002; Project Start 01-JUL-2001; Project End 01-FEB-2003

  Summary: Epstein Barr virus (EBV) is a gamma herpes virus that latently infects greater than 90% of the adult population. Despite a relatively benign course in most carriers, EBV has growth transforming potential and is associated with a number of malignancies, including nasopharyngeal carcinoma, Hodgkin's lymphoma and **Burkitt's lymphoma.** EBNA1 is a vital EBV latency antigen that maintains the viral episome and is found in all EBV-associated tumors. EBNA1-specific CD8+ T cell immunity is blocked by its glycine-alanine repeat domain, which prevents proteosomal processing for MHC class I. However, our laboratory recently showed that the normal host response to EBNA1 lies in the CD4+ TH1 T cell compartment. TH1 CD4+ T cells are known to be critical for resistance to tumors and viruses in mice. This project will characterize EBNA1-specific CD4+ lymphocytes in several ways. First, we will optimize techniques to detect EBNA1 - specific responses using intracellular cytokine staining and real time PCR and thereby have methods to follow this immune response in patients with EBV-associated malignancies. Second, we will investigate the role of the antigen-presenting cell in the polarization of the CD4+ T cells to TH1 in vivo. We will describe the phenotype of the EBNA1- specific response in blood and tumor infiltrating lymphocytes from patients with EBV-associated Hodgkin's lymphoma and nasopharyngeal carcinoma to determine if EBNA1 immunity is reduced or changed to a TH2 response. Finally, we will learn to expand EBNA1 immunity in T cells from patients with EBV-associated malignancy, including if need be redirect established TH2 responses to TH1. These experiments will set the stage for clinical studies, most likely with dendritic cells pulsed with EBNA1, to manipulate the immune response in patients with EBV-associated malignancy.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: EBV SPECIFIC THERAPY OF LYMPHOMA USING DENDRITIC CELLS**

  Principal Investigator & Institution: Dhodapkar, Madhav V.; Assistant Professor; Lab/Cell Physiol & Immunology; Rockefeller University New York, Ny 100216399

  Timing: Fiscal Year 2002; Project Start 01-APR-2000; Project End 09-JUL-2002

  Summary: Epstein Barr virus (EBV) is a ubiquitous gamma herpes virus associated with the development of several B lymphoproliferative diseases including lymphoma in human immune deficiency virus (HIV) infected individuals. Cellular immunity plays a critical role in the control of EBV and other viral infections. However it has been difficult to boost this arm of the immune response using current approaches in humans. Dendritic cells (DCs) are specialized antigen presenting cells (APCs) capable of generating strong anti-viral immune responses. Our hypothesis is that Dcs will be effective adjuvants for the generation of EBV specific immune response for therapy and prevention of lymphoma in HIV infected individuals. Maturation of DCs ex vivo leads to an increase in their potency in vitro. We have recently demonstrated that a single injection of antigen bearing mature DCs, but not unpulsed DCs or antigens alone,

generated broad CD4 and CD8+ve T cell immunity in healthy volunteers. These data provide the first controlled evidence of immunogenicity of DCs in humans. We will now examine the strength and durability of the T immunity using newer quantitative assays. In studies proposed herein, we will next determine the magnitude of EBV specific memory using DCs as APCs and EBV specific effector CTL response using newer sensitive assays (ELISPOT and MHC-tetramer binding) in patients with HIV infection, as compared to normal hosts. Using the ELISPOT assay, we will also examine the nature of CD4+ve T cell immunity to EBV in these populations. These studies will serve as a baseline for future immune therapeutic trials to boost EBV and HIV specific immune responses. Recent studies in our laboratory have demonstrated that DCs can acquire exogenous antigen from apoptotic cells and generate CD8+ CTLs. There fore we will examine if DCs are able to acquire antigen from apoptotic EBV infected cells, as a potentially novel strategy for generating EBV specific CTL responses in patients. The long term goals are to use DCs as adjuvants to boost EBV and HIV specific immune response in patients with HIV associated lymphoma. The proposed studies and career development plan will provide the necessary laboratory experience to complement the PI s prior expertise in clinical oncology research and lay the foundation for career as a physician-scientist in tumor immunology.

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- **Project Title: EBV VECTORS FOR TARGETED GENE THERAPY OF B-LYMPHOMAS**

  Principal Investigator & Institution: Pagano, Joseph S.; Professor; Medicine; University of North Carolina Chapel Hill Aob 104 Airport Drive Cb#1350 Chapel Hill, Nc 27599

  Timing: Fiscal Year 2002; Project Start 10-SEP-2000; Project End 31-AUG-2004

  Summary: (Applicant's Abstract): The goal of this application is to effectively and selectively eliminate B-cell derived lymphoma and leukemia cells in cancer patients using a naturally targeted viral vector system. To achieve this goal, the applicant proposes to develop a novel combinatorial gene therapy approach as applied to Burkett B lymphoma based on i) a minimal "gene-less" B lymphotropic Epstein-Barr virus (miniEBV) vector, and ii) a genetically enhanced "hyper-suicide" HSV1 thymidine kinase (super-TK). Specifically, the following studies will be undertaken: Aim 1) To test the efficacy of B lymphotropic miniEBV vectors to deliver and express a suicide gene into human B-cell lymphomas following oncotropic and oncolytic strategies. This approach is based on the transfer of the viral thymidine kinase (TK) gene into B-cell derived lymphomas rendering them sensitive to the prodrug ganciclovir (GCV). For this endeavor miniEBV/sTK will be used to infect B-lymphoma cells in vitro and the transiently infected cells implanted in an animal model to analyze prodrug mediated eradication of the lymphoma using ex vivo protocol. Aim 2) Development of an in vitro cultured packaging cell system to produce helper-free infectious miniEBV. This will involve cloning the genome of EBV in a BAC based vector to delete the packaging sequence by homologous recombination. In addition, EBV negative cell lines will be evaluated for their permissivity to miniEBV replication and packaging into infectious virions. Aim 3) Use the results obtained in Aims 1-2 to test the miniEBV system for its efficiency and safety in a SCID-Human lymphoma/leukemia animal model using an in vivo protocol. Pre-established human B-lymphoma in vivo by intravenous (i.v.) injection with this system will be also evaluated in order to eliminate the disseminated B-lymphoma from various organs of the animal.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: ELONGATION AND TERMINATION OF TRANSCRIPTS BY RNA POL II**

  Principal Investigator & Institution: Bentley, David L.; Professor; Biochem & Molecular Genetics; University of Colorado Hlth Sciences Ctr P.O. Box 6508, Grants and Contracts Aurora, Co 800450508

  Timing: Fiscal Year 2003; Project Start 01-JUL-2003; Project End 30-JUN-2007

  Summary: (provided by applicant): The synthesis of messenger RNA precursors by RNA polymerase II is the primary event in gene expression and is central to the life of cells. The amount of mRNA made from each gene is a key determinant of how much of each corresponding protein is made. For this reason RNA pol II function is extremely carefully regulated by the cell. Corruption of this process of transcription is a major cause of cancer and interference with the transcription program of viruses is a potential target for therapeutics. Transcription can be regulated at the level of initiation of the RNA chains or at the level of their elongation. Initiation is thought to be regulated by controlling recruitment of RNA polymerase to a gene's promoter region but it is much less clear how elongation is controlled. Elongational control is important in a number of clinically important examples. HIV regulates its transcription at the level of elongation and failure to regulate elongation of c-myc oncogene transcripts contributes to development of **Burkitt's lymphoma.** In this proposal, genetic and biochemical approaches will be used to study pol II transcriptional elongation and termination in animal cells and in budding yeast. The proteins of the transcriptional machinery are highly conserved between yeast and mammals and yeast offers significant experimental advantages for genetics. The specific aims of this work are: Aim 1: To determine how elongation factors influence transcription and termination by pol II on individual genes in budding yeast. Aim 2: To determine the role of the pol II CTD in termination of transcription at the 3' ends of genes encoding polyadenylated and non-polyadenylated RNAs. Aim 3: To determine how pre-mRNA processing factors that associate with transcribing pol II affect elongation and termination at genes that produce polyadenylated and non-polyadenylated transcripts.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: EPSTEIN BARR VIRUS INDUCED GENOMIC INSTABILITY**

  Principal Investigator & Institution: Sixbey, John W.; Professor; Microbiology and Immunology; Louisiana State Univ Hsc Shreveport P. O. Box 33932 Shreveport, La 71103

  Timing: Fiscal Year 2002; Project Start 05-APR-1995; Project End 31-MAR-2006

  Summary: (provided by the applicant): The Epstein-Barr virus (EBV) is a ubiquitous human herpesvirus that, despite the life-time rapport typically achieved with its human host, can be associated with benign (infectious mononucleosis) and malignant (Burkitt's lymphoma, Hodgkin's lymphoma, primary central nervous system lymphoma) lymphoproliferative diseases. The overall objective of this grant is to understand molecular mechanisms by which EBV causes disease and their inter-relatedness to modes of viral persistence in the memory B lymphocyte reservoir. Physiologic signaling via the B cell antigen receptor (surface immunoglobulin) has major implications for the fate of any infected B cell, leading to cell proliferation and differentiation or, conversely, apoptosis. Because we showed up-regulation of recombinase activating genes RAG1 and RAG2 upon EBV infection of mature B cells, we now hypothesize that virus diversifies the B cell antigen receptor through induction of secondary immunoglobulin gene rearrangements as a means of assuring adequate survival signaling in infected cell progeny. Renewed V(D)J recombination outside the selective environment of bone

marrow or germinal centers has potential pathogenic consequences that include auto-immunity, lymphoproliferation and chromosomal damage. The specific aims to test our hypothesis are: 1) to determine if secondary rearrangements of immunoglobulin variable region genes occur as a consequence of RAG induction by Epstein-Barr virus; 2) to determine if RAG1 and RAG2 are expressed in human peripheral blood lymphocytes in vivo as a consequence of acute EBV infection; 3) to analyze EBV DNA integration as a marker of illegitimate recombination prompted by viral induced RAG expression; 4) to determine the mechanism by which RAG1 and RAG2 are up regulated by latency protein EBNA1. The use of recombinant EBV expressing green fluorescent protein allows rapid selection of infected cells now capable of expressing RAG; concurrent analysis by flow cytometry for altered surface immunoglobulin; detection by PCR of broken DNA ends or excision circles that are byproducts of V(D)J recombination; and subsequent analysis for chromosomal abnormalities from aberrant RAG.

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- **Project Title: EPSTEIN BARR VIRUS LATENCY AND ONCOGENESIS**

  Principal Investigator & Institution: Kieff, Elliott; Princeton University 4 New South Building Princeton, Nj 085440036

  Timing: Fiscal Year 2002

  Summary: The objective of these experiments is to understand the mechanisms by which Epstein-Barr Virus (EBV) establishes latent infection, persists, and causes neoplasia. The specific aims are (i) To complete the identification of EBV reading frames and proteins that are expressed at various stages of experimental latent or lytic infection of cells, in vitro. (ii) To identify cellular genes whose expression is specifically altered during the course of latent or lytic EBV infection, in vitro. (iii) To further identify viral and cellular genes expressed in cell lines and tumor tissue from patents with EBV associated Lymphoproliferative Disease, **Burkitt's Lymphoma,** Hodgkin's Disease, Nasopharyngeal Carcinoma, and Gastric Cancer and to further explore the association of EBV with testicular cancers. (iv) To further identify EBV and cellular gene expression in latently infected lymphocytes in the peripheral blood and lymphoid organs of normal people undergoing phlebotomy, biopsy, or surgical resection. (v) To establish and maintain databases of the effects of EBV on cell gene expression.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: FUNCTIONS OF EBNA-1 IN THE STABLE REPLICATION OF EBV**

  Principal Investigator & Institution: Aiyar, Ashok A.; Microbiology and Immunology; Northwestern University Office of Sponsored Research Chicago, Il 60611

  Timing: Fiscal Year 2002; Project Start 30-SEP-1999; Project End 29-SEP-2004

  Summary: I will continue ongoing studies designed to characterize the mechanism by which the Epstein-Barr nuclear antigen 1 (EBNA-1) protein of Epstein-Barr virus (EBV) facilitates the stable replication of plasmids that bear oriP, EBV's plasmid origin of replication. Together my post-doctoral mentor, Dr. Bill Sugden, and I have demonstrated that oriP-plasmids are synthesized directly by the cell, and that EBNA-1 functions post- synthetically to stabilize oriP-plasmids. Our work also demonstrates that human cells can actively eliminate extra- chromosomal DNAs. This application experimentally addresses the process by which human cells eliminate plasmids, by defining when it occurs in the cell-cycle, and understanding the mechanism by which those viruses that maintain their genomes as plasmids in human cells overcome it. It also experimentally addresses the mechanism by which oriP-plasmids are stabilized so

that they can be partitioned faithfully to daughter cells, and the contributions of EBNA-1 to these processes.

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- **Project Title: FUNCTIONS OF VEROTOXIN RECEPTOR GB3 (CD77) IN B CELL FUNCTIONS**

  Principal Investigator & Institution: Maloney, Mark D.; Spelman College 350 Spelman Ln Sw Atlanta, Ga 303144399

  Timing: Fiscal Year 2002

  Summary: Globotriaosyl ceramide (Gb3 or CD77), and potentially other glycosphingolipids which contain Gala-4Gal residues, are receptors for verotoxins (VT's). VT's (also called Shiga-like toxins) produced by E. coli serotype 0157:H7 and other serotypes have been implicated as causative agents in the development of hemorrhagic colitis and the hemolytic uremic syndrome (HUS), which is the leading cause of pediatric acute renal failure. Gb32 is expressed on a variety of human cell types including those of pediatric glomeruli and germinal center B lymphocytes. Previously, we identified VT-like sequences on CD19 and the IFNAR-1 subunit of the interferon-alpha receptor, and we have demonstrated roles for Gb3 in CD19-mediated adhesion and interferon- alpha-induced growth inhibition in **Burkitt's lymphoma** cells, B cells with a phenotype similar to that of germinal center B cells. Recently we have identified verotoxin-like sequences on human murine MHC class II proteins. Therefore, interactions between Gb3 and MHC class II proteins could play a major role in antigen presentation by lymphocyt4s with a germinal center phenotype. The long term objective of this research is to determine in a comprehensive manner the roles of the VT receptor Gb3 in immune responses and the pathogenic effects on the immune system mediated by VT during human infection with VT-producing E. coli. The specific aims of the proposed research are to identify potential Gb3-binding sites on CD19 and MHC class II proteins using molecular modeling techniques, determine if Gb3 interacts with MHC class II molecules in **Burkitt's lymphoma** cells, and investigate the mouse as a model for the role of Gb3 in B cell functions.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: GENETIC MODIFIERS OF EPSTEIN-BARR VIRUS/HOST INTERACTNS**

  Principal Investigator & Institution: Adamson, Amy L.; Biology; University of North Carolina Greensboro 103 Foust Building Greensboro, Nc 274026170

  Timing: Fiscal Year 2002; Project Start 01-APR-2002; Project End 31-MAR-2004

  Summary: (provided by applicant): Viruses often manipulate their host cellular environment in order to create favorable conditions for viral replication and survival. Virally-induced changes include alteration of the host cell cycle progression, signal transduction cascades, and transcriptional functions, among others. Epstein-Barr virus (EBV) is a human herpesvirus that infects approximately 90 percent of the world's population. EBV is the causative agent of infectious mononucleosis, and is associated with several forms of cancer, including **Burkitt's lymphoma** and nasopharyngeal carcinoma. EBV also produces oral hairy leukoplakia in AIDS patients. Interestingly, while infection with EBV is widespread, only certain populations of people seem to be susceptible to EBV-related cancers. To understand how EBV proteins interact with host cellular proteins, and how such interactions may promote disease, we have introduced the EBV immediate-early BZLF1 protein into the model organism Drosophila, and will

perform genetic screens to identify cellular proteins that modulate Z activity. We will carry out an Fl screen of 20,000 flies, map the resulting modifiers, and determine if these modifying genes have human homologs. This work will produce a plethora of putative EBV enhancers or suppressors, which will be the basis for much future work. The results from this genetic screen will shed light onto the mechanisms by which EBV alters normal cellular functions, as well as how genetic predisposition may play a role in EBV-related diseases.

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- **Project Title: HOST IMMUNITY TO EBV INFECTION IN VITRO AND IN VIVO**

  Principal Investigator & Institution: Thorley-Lawson, David A.; Professor of Pathology; Pathology; Tufts University Boston Boston, Ma 02111

  Timing: Fiscal Year 2003; Project Start 01-SEP-1981; Project End 31-JAN-2008

  Summary: (provided by applicant): The long term objective of this study is to develop a deeper understanding of persistent infection with Epstein-Barr virus (EBV). EBV has the capacity to drive the proliferation of resting B lymphocytes and this makes it a risk factor for human cancers such as Hodgkin's disease, **Burkitt's lymphoma,** immunoblastic lymphoma and nasopharyngeal carcinoma. However, the virus is able to persist in a quiescent state in vivo where it specifically targets resting memory B cells. By understanding how EBV can persist in most individuals without causing disease we hope to gain insight into what goes wrong when the virus does cause neoplastic disease. This study wilt employ sophisticated cell fractionation techniques and quantitative RealTime DNA and RT PCR assays to address four unresolved issues around EBV persistence. 1. Does acute EBV infection, infectious mononucleosis (AIM), represent a disordered state of EBV infection or simply an amplified version of the stable, long term carrier state? 2. Does EBV, like other herpesviruses, shut off the expression of all protein coding genes when it reaches its final site of persistence - long lived memory B cells in the peripheral blood? 3. What is the nature and origin of the latently infected memory cells proposed as the site of EBV persistence? Are they bona fide memory cells? Does antigen play a role in the production and/or maintenance of these memory cells or do latent proteins perform these functions? How rapidly do the infected cells turn over? 4. Are epithelial cells of the nasopharyngeal lymphoid system e.g. tonsils infected with EBV in vivo or infectable in vitro? Previous studies have analyzed EBV infection of epithelial cell lines and tissues from sites other than the site of persistent infection - the nasopharyngeal lymphoid tissue. However, epithelial tissues are biologically diverse so we will focus our studies on the biologically relevant epithelium from the tonsil.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: IMMUNE CONTROL OF LATENT GAMMAHERPESVIRUS INFECTION**

  Principal Investigator & Institution: Blackman, Marcia A.; Associate Member; Trudeau Institute, Inc. Saranac Lake, Ny 12983

  Timing: Fiscal Year 2003; Project Start 01-DEC-2002; Project End 30-NOV-2007

  Summary: (provided by applicant): Gammaherpesviruses, such as Epstein Barr virus (EBV) and Kaposi's sarcoma-associated herpesvirus are important human pathogens, associated with lymphoproliferative disorders and various maligancies, including **Burkitt's lymphoma,** Hodgkin's disease, nasopharyngeal carcinoma and Kaposi's sarcoma. The initial lytic infection is efficiently cleared, but the virus establishes life-long latency, effectively hiding from the immune system. Periodic viral reactivation occurs

sporadically, but is kept in check by host mechanisms of immune control. CD8+ T cells have been shown to be important for control of EBV, but the mechanisms are poorly understood. In the current proposal, we will exploit a new mouse model, murine gammaherpesvirus-68, MHV-68, to study basic mechanisms of immune control of this important class of viruses. Accumulating data from our laboratory and others show that MHV-68 latency is harbored in multiple cell types and anatomical sites. Therefore, an essential first step in characterizing immune control is to characterize reservoirs of latency, and determine mechanisms for maintaining the latent load, which will be addressed in Aims 1 and 2 of the current proposal. Taking this information into account, we will then examine immune mechanisms for controlling latency and preventing viral recrudescence in Aim 3. This is important for human health, as loss of immune control as a consequence of AIDS or post-transplant immunosuppression is associated with increased latent load and the onset of disease. The availability of an easily manipulated experimental mouse model is a major advance in the field, and allows fundamental mechanisms to be addressed. It is anticipated that the basic information gathered in this proposal will provide insight into the mechanisms of immune control of the clinically-relevant human gammaherpesviruses.

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- **Project Title: IMMUNOLOGIC STUDIES OF ENDEMIC BURKITT'S LYMPHOMA**

  Principal Investigator & Institution: Moormann, Ann M.; Medicine; Case Western Reserve University 10900 Euclid Ave Cleveland, Oh 44106

  Timing: Fiscal Year 2002; Project Start 01-AUG-2002; Project End 30-APR-2007

  Summary: (provided by applicant): The long-term goal of this research is to advance knowledge of the mechanism by which malaria interacts with the host immune system and Epstein-Barr virus (EBV) to promote the development of endemic Burkitt?s lymphoma (eBL). An association between holoendemic malaria, EBV infection and eBL during childhood is well established in Africa. EBV -specific HLA class I-restricted CD8+ cytotoxic T lymphocyte responses limit proliferation of B cells infected with EBV. These and other findings suggest that malaria suppresses EBV- specific immunity and thereby promotes the emergence of eBL. The mechanisms by which malaria alters immunity to EBV and its role in the pathogenesis of eBL are poorly understood. This mentored-training award builds upon collaborations between Case Western Reserve University and the Kenya Medical Research Institute. The proposal will test the hypothesize that EBV -specific T cell immunity is not altered by malaria exposure per se but that viral immunity is transiently and profoundly suppressed during episodes of acute malaria morbidity, thereby providing the opportunity for latently infected B cells to undergo malignant transformation. Thus, in children presenting with eBL, EBV -specific T cell immunity will be intact since suppression of such preceded development of eBL. The specific aims will test the following hypotheses: Aim 1: EBV-specific T cell IFN-y and IL-I0 responses are stable over time and not influenced by the pattern of malaria transmission in a population of healthy children. Aim 2: EBV-specific IFN-y T cell responses are transiently suppressed while IL-I0 T cell responses are induced during an attack of malaria morbidity. Aim 3: EBV-specific T cell IFN-y and IL-I0 responses in children with eBL are qualitatively and quantitatively similar to that of age-matched controls. Characterization of EBV-driven T cell responses associated with malaria will have implications for EBV vaccine trials to be conducted in populations where this lymphoma has a high prevalence.

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- **Project Title: INTERACTION BETWEEN HIV-1 AND CELL CYCLE PROTEINS**

  Principal Investigator & Institution: Giordano, Antonio; Professor; Fels Institute for Cancer Rearch & Molecular Biology; Temple University 406 Usb, 083-45 Philadelphia, Pa 19122

  Timing: Fiscal Year 2002; Project Start 05-JUL-2002; Project End 30-JUN-2004

  Summary: (provided by applicant): HIV-1 has long been recognized as the etiological agent in acquired immunodeficiency syndrome (AIDS). Although neoplasms arise in HIV-1-infected patients more frequently than in other forms of immunosuppression, the role of HIV-1 as an oncogenic virus has not yet been clarified. HIV-1 encodes for the Tat transcription protein, which is essential for efficient viral replication. Tat is a likely candidate to contribute to tumor pathogenesis in HIV-1-infected patients because of its growth promoting activity, angiogenic function and regulation of the apoptotic pathway. The oncogenic role of Tat is further supported by an increased incidence of tumors in Tat-transgenic mice. 2-12 percent of AIDS patients develop primary central nervous system (CNS) lymphomas, of which 98 percent are B-cell lymphomas. Recently, a virus-linked mechanism of lymphomagenesis involving the Rb2/pl30 pathway has been proposed in AIDS-related **Burkitt's lymphoma** (BL). A deregulation of cell growth control by RB-related proteins may be the first step in lymphomagenesis in HIV-1-infected patients. However, little is known about the mechanisms by which HIV-1 gene products interact with the RB family and other cell cycle regulatory proteins. Among the latter, Cdk9 (PITALRE), identified and cloned in our laboratory, is the most likely candidate to be involved in the development of AIDS-related neoplasms. Cdk9, a cdc2-related kinase, promotes general elongation of transcription by activating the C-Terminal Domain (CTD) of RNA Polymerase II. Cdk9 is a partner of cyclin T1, which binds to the HIV Tat protein, supporting the positive involvement of Cdk9 in HIV replication and suggesting its possible role in the development of AIDS-related neoplasms. The goal of this proposal is to investigate the interaction between HIV gene products and cell cycle regulatory proteins and their role in the development/growth of the most common AIDS-related CNS and other subtype lymphomas. In particular, we will focus our attention on the cell cycle regulatory proteins, pRb2/pl30, Cdk9 and cycT1, which seem to be involved in AIDS-related tumorigenesis.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: INTERNATIONAL HERPESVIRUS WORKSHOP, CAIRNS, AUSTRALIA**

  Principal Investigator & Institution: Nelson, Jay A.; Director & Professor; Molecular Microbiology and Immunology; Oregon Health & Science University Portland, or 972393098

  Timing: Fiscal Year 2002; Project Start 17-JUL-2002; Project End 30-JUN-2003

  Summary: (provided by applicant): This proposal requests funds to enable young investigators to attend the 27th International Herpesvirus Workshop at the Cairns Convention Center, Australia, July 20-26, 2002. The International Herpesvirus Workshop is the premier scientific meeting for herpesvirus researchers, and the only meeting with an interdisciplinary focus on all the major subfamilies ofherpesviruses and all aspects of research from molecular biology to clinical studies. The strength of the Workshop rests on the cross-fertilization that results from comparison of different herpesviruses, different approaches to key questions and on the support and participation of leading researchers in the field, most significantlyincluding promising young investigators and students in training. Moreover, the forum is truly international,

with broad- based world-wide attendance. The medical importance of this meeting is clearly indicated from the wide variety of diseases caused by the now recognized eight human herpesviruses. These include skin and eye ulcerations (HSV-1), genital lesions (HSV-2), meningitis and encephalitis (HSV-1 and HSV-2), infectious mononucleosis (EBV), chicken pox and shingles (VZV). CMV is a major cause of birth defects including mental retardation, blindness and deafness due to congenital transmission but aisc) a significant opportunistic pathogen in AIDS patients and organ transplant recipients. More recently, CMV and HSV have been implicated as pathogenic contributors in the development of atherosclerosis. Cancer has also been associated with herpesvirus infections. EBV is associated with **Burkitt's lymphoma,** other B cell neoplasias and nasopharyngeal carcinoma. The most recent human herpesvirus discovered (HHV-8 or KSHV) is associated with Kaposi's sarcoma in AIDS patients and other immunosupressed persons and in other groups. All of the herpesviruses persist for life and therefore pose significant problems in the treatment of immunologically compromised persons. Diseases caused by reactivation of most human herpesviruseses are a significant cause of morbidity and mortality in various immune patient populations. Shingles and post-herpetic neuralgia are problems in the elderly. Animal herpes significant economic importance to the poultry (Marek's and others), swine (pseudorabies virus), cattle (several bovine herpesviruses) and horses (several equine herpesviruses). In addition, these animal herpes serve as Important model systems for studying herpesvirus pathogenesis. Finally, recombinant DNA technology permits the design of novel vaccines for controlling the spread of animal herpesvirus infection and the design of herpesvirus vectors for gene therapy. Workshop sessions will take an interdisciplinary approach to the following topics: virus structure, mechanism of virus entry and cell-cell spread, membrane proteins, pathogenesis and latency, DNA replication, vaccination and the immune response, transcriptional control, regulation of gene expression, chemotherapeutic targets, and virus gene therapy.

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- **Project Title: LYMPHOMAGENESIS**

  Principal Investigator & Institution: Choi, Yong S.; Laboratory Director; Ochsner Clinic Foundation New Orleans, La 70121

  Timing: Fiscal Year 2002; Project Start 01-APR-2002; Project End 31-MAR-2005

  Summary: A number of lymphoma including follicular cell, Burkitt's, and diffuse large cell lymphoma are known to originate from lymphoid tissues. Although the pathobiology of malignant B cells has been studied extensively, the role of the germinal center (GC)-microenvironment in lymphomagenesis has not been investigated in the molecular term. The GC is an unique microenvironment where antigen-activated B cells undergo clonal selection by proliferation and apoptosis, selecting memory B cells. At the same time, the genetic events such as somatic mutation and Ig-isotype switching occur, producing high-affinity antibodies. B cell lymphoma originate as a consequence of the genetic mobility and mutability. Follicular dendritic cell (FDC) is a stromal cell located inside but not outside of the GC. Furthermore, most of the GC-B cells die by apoptosis unless rescued by FDC. FDC provides the signals for survival and proliferation of GC-B cells and lymphoma cells in the early stage of lymphomagenesis. The objective is to characterize the function of FDC in B cell lymphomagenesis in the molecular term. Specific Aims are to identify the FDC-signaling molecules, using the FDC-specific monoclonal antibodies and a mammalian cell expression vector, and to characterize the function of the FDC-molecules in lymphomagenesis in vivo. The molecular identification of the FDC signaling molecule will help us understand the unique role of

the stromal cells in blast transformation of lymphoma cells. In addition, it will facilitate the development of the therapeutic monoclonal antibodies and antagonists which block the propagation of lymphoma cells. Furthermore, our in vivo experimental model will be useful in discovering the target genes for the anti-cancer drugs.

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- **Project Title: MECHANISMS OF NEOPLASTIC TRANSFORMATION BY HMG-I/Y**

  Principal Investigator & Institution: Resar, Linda M.; Pediatrics; Johns Hopkins University 3400 N Charles St Baltimore, Md 21218

  Timing: Fiscal Year 2003; Project Start 01-JUL-2003; Project End 30-JUN-2007

  Summary: (provided by applicant): The HMG-I/Ygene encodes the HMG-I and -Y protein isoforms, which function as architectural chromatin binding proteins involved in transcriptional regulation. These proteins are up-regulated in human cancer, although their role in the pathogenesis of malignancy is unclear. To understand how HMG-I/Y proteins may contribute to transformation, we are exploring their regulation and function. We discovered that HMG-I/Y is a direct c-Myc target gene important in **Burkitt's lymphoma.** We also demonstrated that HMG-I/Y is necessary for transformation because decreasing these proteins in human cancer cell lines blocks the transformed phenotype. We were the first to show that HMG-I/Y proteins have several oncogenic properties. Specifically, overexprossion of HMG-I or-Y leads to anchorage-independent cell growth in several experimental cell lines. Fibroblasts overexpressing HMG-I or-Y are tumorigenic in nude mice. We developed transgenic mice overexpressing HMG-/in lymphoid cells and all of them develop lymphoid hyperplasia and malignancy at a mean age of 8 months. HMG-I overexpression also correlates with genomic instability, which may contribute to tumor initiation or progression. Thus, I hypothesize that HMG-I/Y is an oncogene important in the pathogenesis of human cancer. The focus of this research proposal is to identify the mechanisms involved in transformation by HMG-I/Y using unique reagents developed in my laboratory. Our Specific Aims Are: 1.) Identify and characterize direct HMG-I/Y gene targets involved in neoplastic transformation using microarray analysis. 2.) Define the functional domains of HMG-I/Y involved in transformation, chromosomal instability, and cell cycle regulation. A.) Identify the functional domains of HMG-I/Y required for transformation using the soft agar transformation assay. B.) Investigate the role of HMGI/ Y in genomic instability and identify the relevant domains using spectral karyotyping analysis. C.) Investigate the role of HMG-I/Y in cell cycle regulation and identify the relevant domains using cell cycle profile analysis. 3.) Define the pathways involved in transformation using our HMG-I transgenic mice. A.) Assess transformed lymphoid cells from the HMG-I trangenic mice for overexpression of HMG-I target genes and chromosomal instability B.) Identify pathways involved in transformation by HMG-I by crossing the HMG-I transgenic mice with other genetically altered mice. 4.) Determine if HMG-I/Yexpression correlates with prognosis and clinical outcome in lymphoid malignancies and brain tumors. Study HMG-I/Y gene and protein expression in patient samples from the Johns Hopkins Leukemia and Brain Tumor Banks. This proposal is significant because the results will enhance our understanding of human malignancies with increased HMG-I/Y proteins and may lead to new treatment strategies.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MOLECULAR EPIDEMIOLOGY OF AIDS-ASSOCIATED LYMPHOMA**

  Principal Investigator & Institution: Martinez-Maza, Otoniel; Professor; Obstetrics and Gynecology; University of California Los Angeles 10920 Wilshire Blvd., Suite 1200 Los Angeles, Ca 90024

  Timing: Fiscal Year 2002; Project Start 30-SEP-1997; Project End 31-MAY-2005

  Summary: (Provided by the applicant): Non-Hodgkin' s B cell lymphoma (AIDS-lymphoma) is seen in greatly-elevated frequency in HIV-infected people, not only in North America and Europe, but worldwide. In this proposal, studies are presented to elucidate the molecular epidemiology of AIDS-lymphoma. The proposed studies will utilize the resources of the Multicenter AIDS Cohort Study of the Natural History of AIDS (MACS). In prior studies supported by this award, elevated levels of various immune system molecules that are associated with B cell activation, including IL6 and IL10, sCD23, sCD27, sCD44, and IgE, were seen prior to the clinical detection of AIDS-lymphoma. Notably, there were clear differences in the patterns of expression of such B cell-stimulatory molecules seen in different subtypes of AIDS-lymphoma (Burkitt's/SNCCL vs. other subtypes), suggesting that there are differences in the character of the immune dysfunction that precedes the development of different subsets of these cancers. In addition to this, in very recent work we saw that a single-nucleotide polymorphism (SNP) in the IL10 promoter (-592 C/C), which is known to result in increased expression of IL10, was associated with the development of AIDS-lymphoma. These findings are of great significance, since few risk factors have been identified for AIDS-lymphoma. The specific aims of the proposed studies are to determine: I) if enhanced B cell stimulation, elevated immunoglobulin isotype switch activity, detectable c-myc:Ig gene translocations, and/or detectable circulating B cells with a germinal center-like phenotype, precede the development of AIDS- lymphoma, 2) if SNPs in the genes encoding B cell-stimulatory cytokines (IL6, IL10, TNFalpha, LTalpha, RANTES) are associated with an elevated risk for the development of AIDS-lymphoma, and 3) if subjects who have a genotype that has been seen to be associated with a decreased risk for developing AIDS-lymphoma (CCR5 delta-32 heterozygotes, SDF-1 3'UTR 801 G/G SNP, or IL10 promoter -592 A/A or A/C SNP) show lower levels of B cell activation. The accomplishment of these specific aims will add valuable new information to our understanding of the molecular epidemiology of AIDS-lymphoma, as well as the role of immune dysfunction in the generation and growth of this cancer. This information could form the foundation for future studies on the pathogenesis of AIDS-lymphoma, and may lead to new screening techniques able to detect AIDS-lymphoma earlier in the course of tumor development, allowing for earlier and more effective clinical intervention.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: MYC DEPENDENT PATHWAYS IN APOPTOSIS AND LYMPHOMAGENESIS**

  Principal Investigator & Institution: Cole, Michael D.; Professor; Molecular Biology; Princeton University 4 New South Building Princeton, Nj 085440036

  Timing: Fiscal Year 2002; Project Start 01-JAN-1999; Project End 31-DEC-2003

  Summary: (adapted from the investigator's abstract) The c-myc gene is among the most frequent sites of mutation for any oncogene in human cancer. Approximately 15 percent of all cancers exhibit amplification of the c-myc gene and about 25 percent of breast cancers have similar mutations. Chromosomal translocations at c-myc occur in 100

percent of Burkitt's lymphomas, as well as in the related mouse plasmacytomas. Although these gross chromosomal abnormalities have been recognized for many years, it has only recently become apparent that missense mutations can also play a major role in the oncogenic activity of c-myc, and more that 60 percent of Burkitt's and AIDS-associated lymphomas have mutations that alter the protein structure of the already translocated c-myc gene. A major question confronting the c-myc field (and nuclear oncogenes in general) is which cellular genes are targeted ty the oncoprotein to mediate its function in cell cycle progression or apoptosis. Of broader interest is how the c-myc signaling pathway is interwoven with the signals emanating from other oncogenes. The specific goals of this project are to: 1) Dissect the role of the c-Myc protein in signaling pathways leading to cell growth or apoptosis. They will use novel cell lines that are completely deficient in endogenous c-Myc protein expression through the knockout of both chromosomal alleles. The role of phosphorylation in c-Myc activity and the function of a newly isolated Myc-interacting factor, TIP49, will also be investigated. 2) Analyze the Myc-dependent expression of previously identified as well as novel target genes. Novel targets that are linked to specific biological activities of c-Myc will be selected for by subtractive or differential display approaches. 3) Determine the functional role of mutations in the N-terminal domain of the c-Myc oncoprotein that are found in the majority of Burkitt's lymphomas. Cell lines that are reconstituted with only mutant or wt c-Myc protein expression will allow the unambiguous analysis of different functions. The relative activity of different c-Myc mutants in both B cells and fibroblasts will be analyzed.

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- **Project Title: PEDIATRIC ONCOLOGY PROGRAM**

  Principal Investigator & Institution: Garcea, Robert L.; Professor; University of Colorado Hlth Sciences Ctr P.O. Box 6508, Grants and Contracts Aurora, Co 800450508

  Timing: Fiscal Year 2002

  Summary: (Applicant's Description) The Pediatric Oncology Program is comprised of clinical oncologists, surgeons, pathologists, and basic scientists whose interests are related to the diseases of pediatric cancer patients. The overall goal of the Pediatric Oncology Program is to reduce the cancer burden in the pediatric population by its research, education, and clinical programs. To accomplish this goal, the Program has established an integrated group of members, organized in four focus areas: 1) clinical investigations, 2) a clinical and research bone marrow transplant (BMT) program, 3) clinical translational research in leukemias, cytogenetics, and brain tumors, 4) basic science research in DNA tumor viruses, lymphocyte signal transduction, and fusion protein transcription factors created by chromosomal translocations in childhood ALL. The clinical program is an active participant in the Children?s Cancer Group (CCG), and utilizes investigator-initiated protocols developed by Cancer Center members and by the cooperative group whenever possible. Each year approximately 150 new oncology patients are evaluated and/or treated by the oncology staff, and an additional 25-35 children are transplanted on various protocols in the pediatric BMT unit. Protocol development over the past year has included 13 investigator-initiated and 49 CCG protocols, with 153 patients enrolled. Program members are highly interactive with respect to clinical protocol development, translational research, and basic research, as evidenced by the fact that 36% of the 132 publications have intra-or inter-program collaborations despite the fact that most members are new. Future plans will build upon this foundation of interaction in specific areas including: 1)continue integration of Pediatric and Adult programs in Neuro-oncology and BMT; 2)development of a Cancer

Genetics Clinic to serve as a focus for new translational and basic research; 3)establishment of a clinical and basic research interest group for studying EBV-related lymphoproliferative disease; 4)increased research in cancer survivorship; and 5)faculty career development for new translational and basic scientists interested in areas related to Pediatric Oncology. This is one of a few approved Pediatric Oncology programs within NCI designated Comprehensive Cancer Centers whose growth has been supported by the UCCC and whose growth is projected to continue in the next five years.

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- **Project Title: POST-TRANSCRIPTIONAL GENE REGULATION BY EBV SM PROTEIN**

  Principal Investigator & Institution: Swaminathan, Sankar; Associate Professor; Medicine; University of Florida Gainesville, Fl 32611

  Timing: Fiscal Year 2004; Project Start 01-APR-1999; Project End 31-MAY-2009

  Summary: (provided by applicant): Epstein-Barr virus (EBV) is a human lymphotropic herpesvirus causally associated with epithelial and lymphoproliferative malignancies including **Burkitt's lymphoma,** nasopharyngeal carcinoma and AIDS-associated lymphoma. Immunosuppressed hosts have greater levels of detectable lytic EBV replication and greater viral loads. The mechanism of action of lytic replication genes is therefore important in understanding the dynamics of EBV infection. Some EBV lytic genes such as EBV SM, the subject of this study, have no human homologues and are therefore also attractive therapeutic targets. Moreover, EBV lytic proteins have extensive interactions with host genes, both regulating their expression and modulating their function. Understanding these interactions is likely to yield insights into the requirements for virus replication and persistence as well as fundamental aspects of cell growth and post-transcriptional gene regulation. EBV SM is an essential gene expressed early in EBV lytic replication that has both activating and inhibitory post-transcriptional effects on EBV and cell gene expression. SM physically interacts with cell proteins that carry out RNA processing and export functions. SM stabilizes mRNA and facilitates export of mRNA from specific EBV target genes and inhibits expression of many spliced genes, but also increases expression of a small number of spliced cellular genes. The cellular genes most highly induced by SM are members of a family of interferon-stimulated genes (ISGs). This proposal has four main objectives: The first is to determine the function of four cytoplasmic ISGs induced by SM which are likely to be important in the host response to viral infection. The second is to determine how SM increases mRNA levels in a gene-specific manner, and delineate its effects on EBV lytic gene expression. The third is to determine which cellular RNA processing proteins SM interacts with to inhibit splicing and alter splice-site selection. The last is to determine the mechanism of action and cellular function of a PML body protein known as Sp110b, which SM induces, binds to, and synergizes with to activate gene expression.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: REGULATION OF EBV TRANSCRIPTION IN BURKITTS LYMPHOMA**

  Principal Investigator & Institution: Speck, Samuel H.; Professor; Microbiology and Immunology; Emory University 1784 North Decatur Road Atlanta, Ga 30322

  Timing: Fiscal Year 2002; Project Start 01-AUG-1995; Project End 31-MAR-2004

Summary: Epstein-Barr virus (EBV) is a human lymphotropic herpesvirus which is the etiologic agent of infectious mononucleosis, a self-limiting lymphoproliferative disorder. In addition, EBV is closely associated with two human cancers, African **Burkitt's lymphoma** (BL) and nasopharyngeal carcinoma (NPC), and also appears to be associated with a significant percentage of Hodgkin's lymphoma (HD) as well as the non-Hodgkin's lymphoms that arise in immunosuppressed patients. The role of EBV in lymphomagenesis has remained enigmatic. While viral gene expression in the EBV-associated non-Hodgkin's large cell lymphomas that arise in immunosupppressed individuals mirrors that observed in B lymphocytes immortalized by EBV in tissue culture, the recently characterized restricted pattern of viral gene expression in African **Burkitt's lymphoma** (BL) raises important questions about the role of EBV in the etiology of these tumors. Addressing these questions will require a thoroug understanding of how restricted EBV latency is regulated. It is critical to determine whether restricted latency is a normal viral program, or whether it is brought about by selection during lymphomagenesis. One of the important long range goals of this research is to determine whether this form of restricted viral latency occurs in normal seropositive individuals. We propose to focus on regulation of viral gene expression in group 1 **Burkitt's lymphoma** cell lines, and to assess whether infection of some population of normal peripheral B lymphocytes results in restricted latency, as follows: 1. characterize the roles of Fp and the newly identified Qp in driving transcription of the EBNA 1 gene in group 1 BL cell lines; 2. characterize the viral genomes present in group 1 BL cell lines and clone the critical control regions of the EBNA 1 gene promoter from a representative group 1 BL cell line; 3. assay EBV infection time courses of peripheral blood B lymphocytes for presence of the restricted EBNA 1 transcription pattern; 4. investigate alternative splicing from the U exon to the EBNA 3a, EBNA 3c and EBNA 1 coding exons; and 5. generate a recombinant EBV harboring mutations in the low ffinity EBNA 1 binding sites in the viral BamHI Q fragment, and assess the impact of these mutations on EBNA 1 gene transcription in primary B cells.

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- **Project Title: REGULATION OF EPSTEIN-BARR VIRUS LATENCY**

  Principal Investigator & Institution: Lieberman, Paul M.; Associate Professor; Wistar Institute Philadelphia, Pa 191044268

  Timing: Fiscal Year 2002; Project Start 01-AUG-2002; Project End 31-JUL-2007

  Summary: (provided by applicant): Epstein-Barr virus (EBV) latent infection is associated with several human cancers, including **Burkitt's lymphoma,** Hodgkin's disease, and nasopharyngeal carcinoma. The latent viral genome exists as a multicopy episome that replicates in synchrony with the cellular chromosomal DNA. Latent cycle DNA replication initiates at OriP and EBNA1 is the only viral protein required for OriP-dependent replication and plasmid maintenance. EBNA1 binds to multiple sites in OriP, but has no intrinsic helicase or other enzyme activity associated with DNA replication function. We have used DNA affinity chromatography to isolate and identify several cellular proteins that associate with OriP in an EBNA1-dependent manner. Our preliminary data indicates that these proteins contribute to plasmid maintenance and the regulation of DNA replication. Several of these proteins have known function at human telomeres, including Telomeric Repeat Binding Factor 2 (TRF2), hRap1, and Tankyrase. TRF2 and hRap1 bind telomeric repeats and regulate chromosome stability. We now show that EBNA1 stimulates TRF2 binding to the nonamer repeats (TTAGGG) in the Dyad symmetry region of OriP. Mutation of the nonamer repeats reduced plasmid maintenance function of OriP and sensitizes OriP to genotoxic stress. We

propose that the nonamer-binding proteins function as a DNA damage checkpoint that regulates replication of OriP. Failure to regulate replication leads to a loss of stable plasmid maintenance. However, it is not clear how nonamer-binding proteins execute this function. In this application we propose to determine the structural organization of nonamer binding proteins at OriP. We will determine their protein interactions and their ability to effect single strand formation, subcellular localization, nuclear matrix attachment, and DNA looping between regions of OriP. We have also found that nonamer-binding proteins possess poly-ADP ribose activity, and we will determine how NAD levels and DNA damage may regulate the activity of PARP proteins associated with OriP. We will also determine if EBNA1 is a substrate of PARP in vivo, and if this modification regulates replication or plasmid maintenance function. We will investigate the role of nonamer-binding proteins in modifying OriP DNA and/or chromatin structure. Finally, we will determine if nonamers provide a DNA checkpoint function by arresting OriP replication in response to genotoxic stress. We hypothesize that the nonamer-binding proteins increase stability of the latent viral genome by protecting it from catastrophic recombination and degradation. The experiments proposed in this application will reveal important new insights into the mechanism of EBV latent cycle DNA replication and plasmid maintenance, and may have important implication for other latent herpesviruses, as well as the functions of cellular proteins involved in telomere maintenance.

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- **Project Title: ROLE OF ALTERNATIVE MXI1 ISOFORMS IN THE MYC NETWORK**

  Principal Investigator & Institution: Wechsler, Daniel S.; Associate Professor; Pediatrics & Communicable Dis; University of Michigan at Ann Arbor 3003 South State, Room 1040 Ann Arbor, Mi 481091274

  Timing: Fiscal Year 2002; Project Start 01-JUL-2001; Project End 30-JUN-2005

  Summary: (provided by applicant) The MYC proto-oncogene family plays a central role in the response to mitogenic stimuli and the control of proliferation in normal and malignant cells. c-Myc protein overexpression is a hallmark of **Burkitt's lymphoma,** and N-MYC amplification is a critical prognostic factor in neuroblastoma. MYC gene expression is also deregulated in many other cancers, but the molecular mechanisms that regulate Myc activity are only partly elucidated. This proposal will specifically investigate the role of Mxii and MxiO, two Myc family members, in modulating the activity of both c- and N-Myc. As a transcription factor, Myc regulates expression of genes related to cell growth, division and apoptosis. In contrast to Myc, Mxii represses transcription of some Myc-regulated genes, counteracting the effects of Myc. Thus, Mxii is a Myc antagonist. We have shown that Mxii expression results in growth arrest, suppressing cell proliferation in vitro. Therefore, we hypothesize that reduced Mxii activity is likely to potentiate Myc-dependent proliferation. We recently identified MxiO, a novel, alternatively transcribed Mxii isoform that lacks the growth suppressive activity of Mxii. While MxiO and Mxii are concomitantly expressed in many cell lines and tissues, the relative levels of MxiO are higher in tumors and tumor cell lines than in normal cells. This variation in levels of MxiO and Mxii suggests that the Myc-inhibitory activity of Mxii may be modulated by MxiO. We postulate that MxiO is an Mxii antagonist. This notion of a single gene giving rise to protein products with alternative biological activities is well established in the case of Bcl-x (Bcl-xL vs. Bcl-xS) and Ink4a (p16 vs. p14). In this proposal, we will explore the interactions of MxiO and Mxil with each other and with Myc, in the context of both cell proliferation and neoplasia, by setting the following Specific Aims: (1) Characterize the biological activity of MxiO; (2)

Determine how expression of MxiO and Mxii are coordinately regulated, using our experience with the Mxii promoter to determine the factors that regulate expression through the MxiO promoter; (3) Determine mechanisms of growth regulation by MxiO and Mxii in the context of Myc-we will use models of c-Myc-induced transformation, as well as N-Myc-dependent neuroblastoma proliferation to explore these interactions, and also study patterns of gene expression; and (4) Evaluate the in vivo effects of specifically inactivating mxii or mxiO in transgenic mice. Through these studies, we will gain a better understanding of the role of these Mxii isoforms in the complex Myc signaling pathways involved in cell growth regulation and neoplasia.

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- **Project Title: ROLE OF EBNA3C IN B LYMPHOCYTE TRANSFORMATION**

  Principal Investigator & Institution: Robertson, Erle S.; Associate Professor; Microbiology and Immunology; University of Michigan at Ann Arbor 3003 South State, Room 1040 Ann Arbor, Mi 481091274

  Timing: Fiscal Year 2002; Project Start 20-SEP-1996; Project End 31-JUL-2002

  Summary: (provided by applicant): Epstein-Barr virus (EBV) is a ubiquitous human herpesvirus which is the etiological agent of infectious mononucleosis. EBV is also associated with a number of malignancies which includes **Burkitt's lymphoma,** Nasopharyngeal carcinoma, Oral Hairy leukoplakia, Hodgkin's lymphoma, Adult T-cell lymphomas and lymphoproliferative diseases in transplant and AIDS patients. In vitro, EBV infects and growth transforms B-lymphocytes so that they proliferate continually into lymphoblastoid cell lines (LCLs). In these infected B lymphocytes EBV expresses eleven genes one of which is the Epstein-Barr nuclear antigen (EBNA) 3C. This protein is essential for B lymphocyte transformation and there is a mounting body of evidence demonstrating that EBNA3C is linked to cellular and viral transcription. However, the mechanism by which EBNA3C regulates events involved in EBV mediated B lymphocyte transformation is not fully understood. The specific aims of this proposal are: (1) To investigate the roles of EBNA3C in regulating viral and cellular gene expression in EBV transformed primary human B-lymphocytes. (2) To identify cellular proteins interacting with EBNA3C and the critical functional domains interacting with these cellular proteins. (3) To genetically characterize the EBNA3C gene by introduction of specific mutations in the open reading frame and testing for transformation of primary B-lymphocytes and to analyze the properties of the recombinant LCLs transformed by latently infected EBNA3C mutant viruses.

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- **Project Title: ROLE OF EPSTEIN-BARR VIRUS IN BURKITT LYMPHOMA**

  Principal Investigator & Institution: Sample, Jeffery T.; Associate Professor; St. Jude Children's Research Hospital Memphis, Tn 381052794

  Timing: Fiscal Year 2003; Project Start 30-SEP-1996; Project End 31-MAR-2008

  Summary: (provided by applicant): The long-term objective of the work supported by this grant is to define the role of Epstein-Barr virus (EBV) in Burkitt lymphoma (BL), a B-cell tumor that occurs in geographically distinct regions, and which is also associated with immunosuppression as a consequence of HIV infection and AIDS. The underlying hypothesis of this grant is that EBV contributes directly to BL, despite lack of expression of the known viral transforming genes within the tumor cells. This is supported by the observation that the tumorigenic potential of the BL cell line Akata is dependent on EBV infection and at least two viral gene products the EBV small RNAs EBER-1 and EBER-2.

The contribution of the EBER RNAs to tumorigenic potential, however, is partial relative to that conferred by EBV infection as a whole, indicating that additional viral genes expressed during infection of BL cells are important. The immediate goals of the proposed work are to define the mechanistic contributions of the EBER RNAs and other EBV gene products to the tumorigenic potential of BL cells and to lymphomagenesis itself. Three specific aims are proposed. Under Aim 1, we will identify the cellular targets of the EBER RNAs and define the mechanisms through which they are regulated. We will address two potential mechanisms of EBER function that are suggested by previous experimental observations. The first is that the EBER RNAs function in posttranscriptional gene silencing through direct RNA:RNA interactions with cellular gene RNAs. The second, based on known interactions of the EBERs with components of the cellular translational machinery, is that the EBERs regulate translation of specific cellular mRNAs. Under Aim 2, we will define the contributions of proteins encoded by the EBV BamHI rightward transcripts (BARTs) to BL-cell tumorigenic potential, and in particular whether any of these proteins are responsible for the enhanced survival conferred upon BL cells by EBV that is attributed to viral-enforced down-regulation of the c-MYC proto-oncoprotein under growth-limiting conditions. Under Aim 3, we will assess the importance of EBV genes expressed in BL cell lines to actual lymphomagenesis using the murine model of BL, the Emu-myc transgenic mouse, in which expression of the c-myc proto-oncogene, as in BL, is constitutively overexpressed in B lymphocytes. Specifically, we will express the EBV genes within the B cells of these mice to determine whether this accelerates c-Myc-induced lymphomagenesis, and if so, we will identify the genetic and biochemical basis for this contribution to lymphoma.

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- **Project Title: ROLE OF HERV-K18 SUPERANTIGEN IN EBV LYMPHOMAGENESIS**

  Principal Investigator & Institution: Sutkowski, Natalie A.; Pathology; Tufts University Boston Boston, Ma 02111

  Timing: Fiscal Year 2003; Project Start 30-SEP-2003; Project End 31-AUG-2006

  Summary: (provided by applicant): More than 90% of adults are latently infected throughout their lifetime with the ubiquitous herpesvirus Epstein-Barr virus (EBV). While EBV infection is usually asymptomatic during childhood, it is estimated that half of first-time infected adolescents or adults develop infectious mononucleosis, a disease characterized by polyclonal B cell activation and massive expansion of T cells. EBV is an oncogenic virus; it is associated with **Burkitt's lymphoma,** Hodgkin's disease and nasopharyngeal carcinoma. At least 1% of organ and bone marrow transplant recipients develop EBV+ lymphomas; and EBV lymphoproliferative disorders are common in AIDS patients. The tumors are often associated with vast T cell infiltrates. The SCID/hu mouse is well accepted as an animal model for EBV lymphomagenesis, because SCID mice adoptively transferred with EBV seropositive PBMC from healthy human donors develop EBV+ B cell lymphomas at a high rate. These tumors are strictly T cell dependent and can be prevented by blocking the B-T interaction. We have recently established that EBV transactivates a human endogenous retrovirus, HERV-K18, that encodes a superantigen, which strongly activates T cells. This is the first described report of a pathogen inducing a host cell superantigen. We propose that HERV-K18 Env superantigen activated T cells contribute to EBV lymphomagenesis. This proposal seeks to test whether blocking the superantigen driven T cell response prevents tumorigenesis in the SCID/hu lymphoma mouse model. We propose to block T cell activation by: I. developing monoclonal antibodies specific for the HERV-K18 superantigen; II. blocking

CD28/ICOS costimulation; III. induction of T cell anergy; and IV. ligation of immuno-inhibitory receptor PD-1. Since several other herpesviruses are associated with superantigen or superantigen-like activity, these experiments may have broad-reaching implications for herpesvirus biology. Overall, these studies represent a completely new approach towards understanding the potential role of T cells in herpesvirus oncogenesis.

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- **Project Title: STUDIES OF EPSTEIN-BARR VIRUS**

  Principal Investigator & Institution: Miller, I George.; John F. Enders Professor; Pediatrics; Yale University 47 College Street, Suite 203 New Haven, Ct 065208047

  Timing: Fiscal Year 2002; Project Start 01-JAN-1979; Project End 31-DEC-2002

  Summary: Epstein Barr virus (EBV) is associated with diverse cancers, including nasopharyngeal cancer, non-Hodgkin's lymphoma occurring in immunodeficient individuals, Hodgkin's disease and **Burkitt's lymphoma.** In all EBV-associated tumors the virus remains in a latent state of limited gene expression. Latency is maintained by regulation of the EBV BZLF1 gene, whose product ZEBRA, a b-ZIP transcriptional activator, obligates the virus to enter lytic replication. Our global objective is to understand the mechanism of this switch between latency and the lytic cycle. Studies of the functions of ZEBRA required for activation of lytic cycle gene expression focus on two groups of mutants that are discordant in their capacity to activate transcription and to disrupt latency. These mutants, containing alterations in the DNA recognition domain or in the accessory activation domain, should point to additional functions that are needed to activate the latent virus. Analysis of the downstream targets of ZEBRA include investigations of DNA context effects that permit a promoter to respond to ZEBRA, identification of cellular genes that are activated by ZEBRA, and identification of cellular and viral proteins that directly interact with the ZEBRA protein. Experiments that explore control of expression of the BZLF1 gene include determination whether Zp and Rp, the two promoters that control BZLF1 transcription, are coordinately or sequentially regulated. Clues to the relative importance of cellular or viral factors in BZLF1 regulation should come from study of well characterized EBV transformed cell lines that differ markedly in their responses to chemical inducing stimuli. The proposed experiments take a biologic perspective and utilize molecular genetic and biochemical techniques to explore a central unsolved question in the pathogenesis of this human tumor virus.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: SUPPRESSION OF B-LYMPHOMAS VIA INDUCTION OF INTERFERONS**

  Principal Investigator & Institution: Thomas-Tikhonenko, Andrei; Pathobiology; University of Pennsylvania 3451 Walnut Street Philadelphia, Pa 19104

  Timing: Fiscal Year 2002; Project Start 01-JUL-2002; Project End 30-JUN-2004

  Summary: (provided by applicant): The ability of various infections to suppress neoplastic growth is well-documented. We have previously demonstrated that tumor suppression during acute toxoplasmosis does not involve cytotoxic functions of the immune system and readily occurs in immunocompromised mice. Instead, it relies on systemic inhibition of angiogenesis by circulating factors, most likely interferons. To determine whether AIDS-related Burkitt lymphoma would succumb to infection-mediated suppression, we have established a new mouse model for this disease. It is

based on overexpression of the c-Myc oncoprotein in p53-null bone marrow progenitors. Using this model, we have found that growth of B-lymphomas during acute toxoplasmosis was completely abolished. In this proposal, we will study mechanisms whereby type I and II interferons suppress lymphomagenesis. We will use STATI-null mice in which both type I and type II interferon pathways are inactivated, and determine whether in these animals angiogenesis during infection is restored. We will also determine whether growth of neoplastic B-cells is directly inhibited by interferons. To this end, we will cross STATI- and p53-null mice and generate B-lymphomas that are deficient in STATI expression. They will be implanted into Toxoplasma gondii-infected STATl -null mice. Since in this system both host and tumor cells are refractory to interferons, we expect that B-lymphomagenesis would be completely restored. This would suggest that interferons play a dual role in lymphoma surveillance during infection: direct and aniogenesis-mediated. We will then determine whether exposure to T.gondii antigens (STAg) would lead to the induction of interferons and suppression of angiogenesis and lymphomagenesis. We will also perform tumor load studies in STAg-treated scid-beige mice, to demonstrate that anti-neoplastic properties of STAg do not rely on cell-mediated cytotoxic immunity. This anticipated result will establish that STAg or similar protozoan or bacterial antigens could be developed into new therapeutic modalities for AIDS-related Burkitt lymphoma.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: T LYMPHOCYTES FOR TREATMENT OF EBV-RELATED LYMPHOMA**

  Principal Investigator & Institution: O'reilly, Richard J.; Chairman; Sloan-Kettering Institute for Cancer Res New York, Ny 100216007

  Timing: Fiscal Year 2002

  Summary: Adoptive cell therapy with donor leukocytes can induce regressions of EBV-associated B-cell lymphomas in marrow transplant recipients. Similarly, donor leukocytes containing T cell reactive against alloantigens on host leukemic cells can induce remissions in patients with CML and certain forms of acute leukemia who relapse following marrow transplant. However, such infusions carry a significant risk of severe acute and chronic GvHD, particularly with leukocytes from an "HLA-matched" unrelated or HLA-Disparate related marrow donors are used. Genetic modifications of donor T-cells to express a gene inducing a sensitivity to drugs to which human cells are normally resistant could permit the safe use of in vitro expanded virus-specific T-cells early in their generation, even if populations still contain alloreactive T-cells. Similarly, alloreactive "suicide vector" modified T-cells could be used to eradicate leukemia through a transient reversible GvH reaction. Over the last grant period, we developed a series of dicistronic vectors encoded a mutated NGFR and HSV-TK, defined optimal orientation for expression of the genes and developed efficient techniques for transvection and selection of vector-modified human T-cells. We have shown that the NIT retroviral vector can be used to rapidly select and enrich for desired EBV-virus specific T-cells during their initial proliferation in response to sensitization and to select against contaminating alloreactive T-cells. These T-cells are HLA restricted, antigen-specific and highly cytotoxic against virus-transformed targets in vitro. Upon adoptive i.v. transfer, they specifically migrate to and induce regressions of EBV lymphomas bearing appropriate HLA restrict elements in xenografted SCID mice. Expression of NGFR and HSV-TK is sustained for over 16 weeks in vitro and in vivo. Treatment of mice with ganciclovir post adoptive transfer of human T-cells bearing HSV-TK selectively eliminates these T-cells from blood and targeted tissues. We propose to

explore novel strategies, incorporating genetically modified antigen presenting cells to develop novel, broadly accessible approaches for rapid generation of virus- antigen-specific T-cells of desired specificity and HLA restriction (Specific Aim 1). Using similar strategies we will generate suicide- vector-modified T cells specific for minor alloantigens expressed on human ALL cells (Specific Aim 2). We will then comparatively evaluate virus-specific and minor alloantigen-specific, vector modified T-cells generated by these novel strategies, for their capacities to migrate to and induce regression of targeted human EBV lymphomas and leukemias xenografted in SCID mice (Specific Aim 3). Lastly, we propose to initiate clinical trials of vector-modified and selected virus-antigen-specific and leukemia-associated minor alloantigen specific T cells in the treatment of EBV lymphomas and leukemic relapses complicating marrow allografts (Specific Aim 4).

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: TACI AND B CELL FUNCTION AND TRANSFORMATION**

  Principal Investigator & Institution: Bram, Richard J.; Chair, Division of Pediatric Research La; Mayo Clinic Coll of Medicine, Rochester 200 1St St Sw Rochester, Mn 55905

  Timing: Fiscal Year 2002; Project Start 16-DEC-1997; Project End 30-NOV-2002

  Summary: (adapted from the investigator's abstract) Latent infection by the Epstein Barr virus (EBV) has been implicated as a causative factor in the development of Burkitts lymphoma, Hodgkin's lymphoma, nasopharyngeal carcinoma, and immunodeficiency associated lymphoproliferative disease. The exact mechanisms involved in transformation remain a mystery, however. The applicant has identified a novel B-cell surface receptor whose expression in markedly increased following EBV immortalization of B lymphocytes and in EBV-infected Burkitts lymphomas. TACI (for Transmembrane Activator and CAML Interactor) was isolated by a yeast two- hybrid screen with the intracellular Ca2+ regulating protein CAML as bait. As predicted, crosslinking the TACI receptor activates the NF-AT (Ca2+dependent) transcription factor. Surprisingly, TACI also potently activates API and NF-kB transcription factors following stimulation. TACI appears to be a new member of the family of Tumor Necrosis Factor Receptors (TNFR), which include TNFR, FAS, CD40, and other receptors implicated in initiating growth and programmed cell death in lymphocytes. An attractive hypothesis is that TACI contributes to the transformation of B cells through its activation of multiple transcription factors following EBV infection. The goals of this project are to elucidate the mechanisms of action of TACI in normal and EBV-associated Burkitt lymphoma cells, and to determine its contribution to cellular transformation. Experiments will focus on determining the mechanism of action of TACI by identifying its functional domains, and identifying both extracellular and intracellular protein contacts that mediate its action. Additionally, targeted disruption of the TACI gene in mice will allow the identification of its normal role in regulation of the immune system. Lastly, the proposed project will explore the possibility that experimental manipulation of TACI signaling activating in tumor cells may provide new means to inhibit growth or accelerate death of cancer cells.

  Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: THE MIT TRANSCRIPTION FACTOR IN PEDIATRIC MALIGNANCIES**

  Principal Investigator & Institution: Fisher, David E.; Associate Professor; Dana-Farber Cancer Institute 44 Binney St Boston, Ma 02115

Timing: Fiscal Year 2004; Project Start 01-APR-2004; Project End 31-MAR-2009

Summary: (provided by applicant): The MiT transcription factor family contains four bHLHzip proteins which are critical for development of several cell lineages and exhibit overlapping DNA recognition with the Myc family. One member, Miff, is regulated by Melanocyte Stimulating Hormone (MSH) to stimulate pigment cell growth and differentiation. Another, TFE3, modulates osteoclast development together with Mitf. We recently cloned a recurring translocation in Papillary Renal Cell Carcinoma (PRCC), and identified the MiT family member TFEB as a new fusion oncogene, which places full length TFEB under the transcriptional regulation of a ubiquitous and abundant gene of unknown function reminiscent of c-Myc in **Burkitt's lymphoma.** Translocations which fuse another MiT factor, TFE3, also occur in PRCC as well as Alveolar Soft Part Sarcomas. The oncogenicity of transcriptionally dysregulated MiT factors sparked investigation of Clear Cell Sarcoma (CCS), an EWS-ATF1 translocated tumor which inexplicably expresses melanoma markers. EWS-ATF1 was seen to constitutively upregulate Miff via mimicking Melanocyte Stimulating Hormone signaling in melanocytes. Dysregulated Mitf, in turn, drives CCS growth and survival. All known MiT-dysregulated tumors are uniformly resistant to conventional chemotherapy. This proposal examines this growing family of malignancies to dissect the pathways through which they transform and identify diagnostic markers and therapeutic targets using this information. In Specific Aim 1 we employ disruption-rescue assays we developed to determine structural and post-translation requirements of MiT factors for specific oncogenic behaviors. In Specific Aim 2 we ask whether the overlapping DNA recognition properties of the MiT and Myc families reflect overlapping transformation mechanisms. Specific Aim 3 uses candidate- and microarray approaches to identify transcriptional targets of the MiT family in these cancers, and scrutinizes their functional importance in tumorigenicity. One such newly identified MiT target gene is c-MET, a finding of particular interest because human germline c-MET mutations produce hereditary PRCC-- the same malignancy in which TFEB or TFE3 translocations occur for sporadic tumors, c-MET was seen to be superactivated in all 6 MiT-associated tumor lines examined, and will be studied in primary tumors. Small molecules inhibitors of c-MET exist and will be examined (together with molecular controls) as potential targeted therapy for these incurable cancers.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

- **Project Title: TUMOR-ASSOCIATED HERPESVIRUSES CONFERENCE**

  Principal Investigator & Institution: Raab-Traub, Nancy J.; Professor; Medicine; University of North Carolina Chapel Hill Aob 104 Airport Drive Cb#1350 Chapel Hill, Nc 27599

  Timing: Fiscal Year 2002; Project Start 01-AUG-2002; Project End 31-JUL-2007

  Summary: (provided by applicant): The Tenth International Meeting of the EBV Association will be held in Cairns, Australia on July 16th-21st, 2002. The theme of the meeting will be "Tumor Associated Herpesviruses" and will focus on EBV and Human Herpevirus 8 (HHV8), but also include gamma herpesvirus infection in animal model systems. Subsequent meetings will be held in Regensburg in 2004 and Boston in 2006. The biennial EBV symposium alternates between the Far East, America, and Europe and provides the only regular forum for EBV research. The meeting encompasses both clinical studies and basic research, providing a unique opportunity to expand our understanding of the molecular basis of EBV and cancer. Each of the sessions and workshops are configured to include molecular biology, immunology, pathology and epidemiology such that every session will have relevance to all attendees, which will

promote communication and cross-fertilization of ideas. The proposed sessions will focus on: Primary EBV infections (including infectious mononucleosis, chronic fatigue and X-linked lymphoproliferative disease); Diagnosis and treatment of EBV diseases; Immunobiology and Pathology of EBV Infection; Lymphoid Tumors (including non-Hodgkin's lymphoma, Hodgkin's disease and Burkitt's lymphoma); Epithelial tumors (including nasopharyngeal carcinoma gastric carcinomas and breast cancer); Recent advances in vaccine development and Transplantation. As well there will be workshops on "Herpesviruses and AIDS", "The diagnosis, epidemiology and treatment of nasopharyngeal carcinoma"; "Animal models of Disease" and "Oral Herpesvirus Infections". The two sessions on the 21st July, Transplantation and Vaccine development, will be combined sessions with the International Herpesvirus workshop. The organization of the meeting has been arranged to 1. Stimulate communication and interactions internationally among clinical and basic scientists and students to facilitate exchange of materials and rapid movement of new basic information to clinical settings, 2. Recognize and encourage young investigators and 3. Highlight new developments in the field and identify areas for future investigation.

Website: http://crisp.cit.nih.gov/crisp/Crisp_Query.Generate_Screen

E-Journals: PubMed Central[3]

PubMed Central (PMC) is a digital archive of life sciences journal literature developed and managed by the National Center for Biotechnology Information (NCBI) at the U.S. National Library of Medicine (NLM).[4] Access to this growing archive of e-journals is free and unrestricted.[5] To search, go to **http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=Pmc**, and type "Burkitt's lymphoma" (or synonyms) into the search box. This search gives you access to full-text articles. The following is a sample of items found for Burkitt's lymphoma in the PubMed Central database:

- **5-Azacytidine up regulates the expression of Epstein-Barr virus nuclear antigen 2 (EBNA-2) through EBNA-6 and latent membrane protein in the Burkitt's lymphoma line rael.** by Masucci MG, Contreras-Salazar B, Ragnar E, Falk K, Minarovits J, Ernberg I, Klein G.; 1989 Jul;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=250871
- **A global transcriptional regulatory role for c-Myc in Burkitt's lymphoma cells.** by Li Z, Van Calcar S, Qu C, Cavenee WK, Zhang MQ, Ren B.; 2003 Jul 8;
  http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=166200
- **Antibodies to Epstein-Barr Virus in a Burkitt's Lymphoma Cell Line in Taiwan Monkeys (Macaca cyclopis).** by Chu CT, Yang CS, Kawamura A Jr.; 1971 Mar;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=377218

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[3] Adapted from the National Library of Medicine: **http://www.pubmedcentral.nih.gov/about/intro.html**.

[4] With PubMed Central, NCBI is taking the lead in preservation and maintenance of open access to electronic literature, just as NLM has done for decades with printed biomedical literature. PubMed Central aims to become a world-class library of the digital age.

[5] The value of PubMed Central, in addition to its role as an archive, lies in the availability of data from diverse sources stored in a common format in a single repository. Many journals already have online publishing operations, and there is a growing tendency to publish material online only, to the exclusion of print.

- **Antiviral effects of interferon on a somatic cell hybrid between two Burkitt's lymphoma cell lines of different interferon sensitivities.** by Lidin B, Lamon EW.; 1982 May;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=351308
- **Cellular and Epstein-Barr virus specific DNA polymerases in virus-producing Burkitt's lymphoma cell lines.** by Allaudeen HS, Rani G.; 1982 Apr 10;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=320622
- **Cloned Shiga Toxin 2 B Subunit Induces Apoptosis in Ramos Burkitt's Lymphoma B Cells.** by Marcato P, Mulvey G, Armstrong GD.; 2002 Mar;
  http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=127801
- **Cloning and sequence analysis of an Ig lambda light chain mRNA expressed in the Burkitt's lymphoma cell line EB4.** by Anderson ML, Brown L, McKenzie E, Kellow JE, Young BD.; 1985 Apr 25;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=341205
- **Cloning and sequencing of a c-myc oncogene in a Burkitt's lymphoma cell line that is translocated to a germ line alpha switch region.** by Showe LC, Ballantine M, Nishikura K, Erikson J, Kaji H, Croce CM.; 1985 Mar;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=366742
- **Cloning and sequencing of a rearranged V lambda gene from a Burkitt's lymphoma cell line expressing kappa light chains.** by Sun LH, Croce CM, Showe LC.; 1985 Jul 11;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=321835
- **c-Myc Proteolysis by the Ubiquitin-Proteasome Pathway: Stabilization of c-Myc in Burkitt's Lymphoma Cells.** by Gregory MA, Hann SR.; 2000 Apr 1;
  http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=85426
- **Complement-Fixation Tests with Cell Lines Derived from Burkitt's Lymphoma and Acute Leukemias.** by Armstrong D, Henle G, Henle W.; 1966 Mar;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=316021
- **Complement-fixing antibodies in sera of human and nonhuman primates to viral antigens derived from Burkitt's lymphoma cells.** by Gerber P, Birch SM.; 1967 Aug;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=335660
- **Epstein --Barr virus-encoded poly(A) -- RNA supports Burkitt's lymphoma growth through interleukin-10 induction.** by Kitagawa N, Goto M, Kurozumi K, Maruo S, Fukayama M, Naoe T, Yasukawa M, Hino KI, Suzuki T, Todo S, Takada K.; 2000 Dec 15;
  http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=305895
- **Epstein-Barr Virus (EBV) Nuclear Protein 2-Induced Disruption of EBV Latency in the Burkitt's Lymphoma Cell Line Akata: Analysis by Tetracycline-Regulated Expression.** by Fujiwara S, Nitadori Y, Nakamura H, Nagaishi T, Ono Y.; 1999 Jun;
  http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=112573

- **Epstein-Barr Virus and the Somatic Hypermutation of Immunoglobulin Genes in Burkitt's Lymphoma Cells.** by Harris RS, Croom-Carter DS, Rickinson AB, Neuberger MS.; 2001 Nov 1;
http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=114624
- **Epstein-Barr Virus Contributes to the Malignant Phenotype and to Apoptosis Resistance in Burkitt's Lymphoma Cell Line Akata.** by Komano J, Sugiura M, Takada K.; 1998 Nov;
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- **Epstein-Barr Virus Nuclear Antigen 1 Sequences in Endemic and Sporadic Burkitt's Lymphoma Reflect Virus Strains Prevalent in Different Geographic Areas.** by Habeshaw G, Yao QY, Bell AI, Morton D, Rickinson AB.; 1999 Feb;
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- **Generation of a variant t(2;8) translocation of Burkitt's lymphoma by site-specific recombination via the kappa light-chain joining signals.** by Hartl P, Lipp M.; 1987 Jun;
http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=365323
- **Induction of an exceptionally high-level, nontranslated, Epstein-Barr virus-encoded polyadenylated transcript in the Burkitt's lymphoma line Daudi.** by Gao Y, Smith PR, Karran L, Lu QL, Griffin BE.; 1997 Jan;
http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&rendertype=abstract&artid=191027
- **Influence of Burkitt's lymphoma and primary B cells on latent gene expression by the nonimmortalizing P3J-HR-1 strain of Epstein-Barr virus.** by Rooney C, Howe JG, Speck SH, Miller G.; 1989 Apr;
http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=248385
- **Intracellular Forms of Human NOTCH1 Functionally Activate Essential Epstein-Barr Virus Major Latent Promoters in the Burkitt's Lymphoma BJAB Cell Line but Repress These Promoters in Jurkat Cells.** by Cotter M, Callahan J, Aster J, Robertson E.; 2000 Feb 1;
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- **Isolation of Epstein-Barr virus (EBV)-negative cell clones from the EBV-positive Burkitt's lymphoma (BL) line Akata: malignant phenotypes of BL cells are dependent on EBV.** by Shimizu N, Tanabe-Tochikura A, Kuroiwa Y, Takada K.; 1994 Sep;
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- **Lambda Ig constant region genes are translocated to chromosome 8 in Burkitt's lymphoma with t(8;22).** by de la Chapelle A, Lenoir G, Boue J, Boue A, Gallano P, Huerre C, Szajnert MF, Jeanpierre M, Lalouel JM, Kaplan JC.; 1983 Feb 25;
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- **Marked, transient inhibition of expression of the Epstein-Barr virus latent membrane protein gene in Burkitt's lymphoma cell lines by electroporation.** by Gahn TA, Sugden B.; 1993 Nov;
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- **Monoclonal and polyclonal antibodies against Epstein-Barr virus nuclear antigen 5 (EBNA-5) detect multiple protein species in Burkitt's lymphoma and lymphoblastoid cell lines.** by Finke J, Rowe M, Kallin B, Ernberg I, Rosen A, Dillner J, Klein G.; 1987 Dec;
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- **Oncogenic Role of Epstein-Barr Virus-Encoded RNAs in Burkitt's Lymphoma Cell Line Akata.** by Komano J, Maruo S, Kurozumi K, Oda T, Takada K.; 1999 Dec;
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- **Partial elimination of Epstein-Barr virus plasmids from Burkitt's lymphoma cells by transfecting the BZLF1 gene.** by Takada K, Ji Z, Fujiwara S, Shimizu N, Tanabe-Tochikura A.; 1992 Sep;
  http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=289120
- **Replacement of the Epstein-Barr Virus Plasmid with the EBER Plasmid in Burkitt's Lymphoma Cells.** by Maruo S, Nanbo A, Takada K.; 2001 Oct 15;
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- **Role of bcl-2 in Epstein-Barr Virus-Induced Malignant Conversion of Burkitt's Lymphoma Cell Line Akata.** by Komano J, Takada K.; 2001 Feb 1;
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- **Separation of episomal Epstein-Barr virus from Burkitt's lymphoma host cell DNA in pulse field gels.** by Harris A, Bentley DR.; 1988 May 11;
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- **Somatic mutation and transcriptional deregulation of myc in endemic Burkitt's lymphoma disease: heptamer-nonamer recognition mistakes?** by Morse B, South VJ, Rothberg PG, Astrin SM.; 1989 Jan;
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- **The bfl-1 Gene Is Transcriptionally Upregulated by the Epstein-Barr Virus LMP1, and Its Expression Promotes the Survival of a Burkitt's Lymphoma Cell Line.** by D'Souza B, Rowe M, Walls D.; 2000 Jul 15;
  http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&artid=112177
- **The EBNA2-related resistance towards alpha interferon (IFN-alpha) in Burkitt's lymphoma cells effects induction of IFN-induced genes but not the activation of transcription factor ISGF-3.** by Kanda K, Decker T, Aman P, Wahlstrom M, von Gabain A, Kallin B.; 1992 Nov;
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- **The Epstein-Barr virus BamHI F promoter is an early lytic promoter: lack of correlation with EBNA 1 gene transcription in group 1 Burkitt's lymphoma cell lines.** by Schaefer BC, Strominger JL, Speck SH.; 1995 Aug; http://www.pubmedcentral.gov/articlerender.fcgi?tool=pmcentrez&rendertype=abstract&artid=189321
- **Three breakpoints of variant t(2;8) translocations in Burkitt's lymphoma cells fall within a region 140 kilobases distal from c-myc.** by Henglein B, Synovzik H, Groitl P, Bornkamm GW, Hartl P, Lipp M.; 1989 May; http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=363004
- **Up regulation of the Epstein-Barr virus (EBV)-encoded membrane protein LMP in the Burkitt's lymphoma line Daudi after exposure to n-butyrate and after EBV superinfection.** by Contreras-Salazar B, Ehlin-Henriksson B, Klein G, Masucci MG.; 1990 Nov; http://www.pubmedcentral.gov/picrender.fcgi?tool=pmcentrez&action=stream&blobtype=pdf&artid=248595

The National Library of Medicine: PubMed

One of the quickest and most comprehensive ways to find academic studies in both English and other languages is to use PubMed, maintained by the National Library of Medicine.[6] The advantage of PubMed over previously mentioned sources is that it covers a greater number of domestic and foreign references. It is also free to use. If the publisher has a Web site that offers full text of its journals, PubMed will provide links to that site, as well as to sites offering other related data. User registration, a subscription fee, or some other type of fee may be required to access the full text of articles in some journals.

To generate your own bibliography of studies dealing with Burkitt's lymphoma, simply go to the PubMed Web site at **http://www.ncbi.nlm.nih.gov/pubmed**. Type "Burkitt's lymphoma" (or synonyms) into the search box, and click "Go." The following is the type of output you can expect from PubMed for Burkitt's lymphoma (hyperlinks lead to article summaries):

- **A complete ileo-tumor-colic fistula in a child with Burkitt's lymphoma.**
  Author(s): Moertel CL, Watterson J, Drake DG, Johnson KW, Brennom WS.
  Source: Pediatric Radiology. 1993; 23(6): 467-8.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=8255654

- **A global transcriptional regulatory role for c-Myc in Burkitt's lymphoma cells.**
  Author(s): Li Z, Van Calcar S, Qu C, Cavenee WK, Zhang MQ, Ren B.
  Source: Proceedings of the National Academy of Sciences of the United States of America. 2003 July 8; 100(14): 8164-9. Epub 2003 June 13.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=12808131

[6] PubMed was developed by the National Center for Biotechnology Information (NCBI) at the National Library of Medicine (NLM) at the National Institutes of Health (NIH). The PubMed database was developed in conjunction with publishers of biomedical literature as a search tool for accessing literature citations and linking to full-text journal articles at Web sites of participating publishers. Publishers that participate in PubMed supply NLM with their citations electronically prior to or at the time of publication.

- **A mutant p21 cyclin-dependent kinase inhibitor isolated from a Burkitt's lymphoma.**
  Author(s): Bhatia K, Fan S, Spangler G, Weintraub M, O'Connor PM, Judde JG, Magrath I.
  Source: Cancer Research. 1995 April 1; 55(7): 1431-5.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=7882347

- **A potential role for antigen selection in the clonal evolution of Burkitt's lymphoma.**
  Author(s): Jain R, Roncella S, Hashimoto S, Carbone A, Francia di Celle P, Foa R, Ferrarini M, Chiorazzi N.
  Source: Journal of Immunology (Baltimore, Md. : 1950). 1994 July 1; 153(1): 45-52.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=8207254

- **A short history of Hodgkin's disease and Burkitt's lymphoma.**
  Author(s): Ortiz-Hidalgo C.
  Source: American Journal of Clinical Pathology. 1994 April; 101(4 Suppl 1): S27-33.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=8154453

- **A study of the association of Epstein-Barr virus with Burkitt's lymphoma occurring in a Chinese population.**
  Author(s): Chan JK, Tsang WY, Ng CS, Wong CS, Lo ES.
  Source: Histopathology. 1995 March; 26(3): 239-45.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=7797201

- **Abnormalities on 1q and 7q are associated with poor outcome in sporadic Burkitt's lymphoma. A cytogenetic and comparative genomic hybridization study.**
  Author(s): Garcia JL, Hernandez JM, Gutierrez NC, Flores T, Gonzalez D, Calasanz MJ, Martinez-Climent JA, Piris MA, Lopez-Capitan C, Gonzalez MB, Odero MD, San Miguel JF.
  Source: Leukemia : Official Journal of the Leukemia Society of America, Leukemia Research Fund, U.K. 2003 October; 17(10): 2016-24.
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- **Absence of c-kit receptor and absent proliferative response to stem cell factor in childhood Burkitt's lymphoma cells.**
  Author(s): Tomeczkowski J, Beilken A, Frick D, Wieland B, Konig A, Falk MH, Reiter A, Welte K, Sykora KW.
  Source: Blood. 1995 August 15; 86(4): 1469-80.
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- **Absence of G-CSF receptors and absent response to G-CSF in childhood Burkitt's lymphoma and B-ALL cells.**
  Author(s): Tomeczkowski J, Yakisan E, Wieland B, Reiter A, Welte K, Sykora KW.
  Source: British Journal of Haematology. 1995 April; 89(4): 771-9.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=7539624

- **Acute tumor lysis syndrome in acute lymphoblastic leukemia and Burkitt's lymphoma: experience in a single centre in Kuwait.**
  Author(s): al-Bahar S, Pandita R, Dhabhar BN, al-Bahar E.
  Source: Annals of Oncology : Official Journal of the European Society for Medical Oncology / Esmo. 1994 April; 5(4): 379.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=8075042

- **Adult Burkitt's lymphoma: clinical and prognostic evaluation of 20 patients.**
  Author(s): Zinzani PL, Gherlinzoni F, Bendandi M, Salvucci M, Tura S.
  Source: Leukemia & Lymphoma. 1994 August; 14(5-6): 465-70.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=7812206

- **Adult Burkitt's lymphoma: two patients with an unusual clinical picture.**
  Author(s): Maran R, Zeidman A, Schwartz A, Mittelman M.
  Source: Presse Medicale (Paris, France : 1983). 1994 October 22; 23(32): 1471-4. Review.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=7824466

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  Author(s): Chapman CJ, Wright D, Feizi HP, Davis Z, Stevenson FK.
  Source: British Journal of Haematology. 1998 December; 103(4): 1116-23.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=9886329

- **Variant translocation in a non endemic case of Burkitt's lymphoma: t (8;22) in an Epstein--Barr virus negative tumour and in a derived cell line.**
  Author(s): Bertrand S, Berger R, Philip T, Bernheim A, Bryon PA, Bertoglio J, Dore JF, Brunat-Mentigny M, Lenoir GM.
  Source: European Journal of Cancer (Oxford, England : 1990). 1981 May; 17(5): 577-84.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=6271555

- **Variant translocation in Burkitt's lymphoma: 8;22 translocation in a French patient with an Epstein-Barr virus-associated tumor.**
  Author(s): Fraisse J, Lenoir G, Vasselon C, Jaubert J, Brizard CP.
  Source: Cancer Genetics and Cytogenetics. 1981 March; 3(2): 149-53.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=6268290

- **Variant translocation t(8;22) and abnormalities of chromosome 15(q22) and 17(q12-21) in a Burkitt's lymphoma/leukaemia with disseminated intravascular coagulation.**
  Author(s): Daly P, Brito-Babapulle V, Lawlor E, Blaney C, Parreira A, Catovsky D.
  Source: British Journal of Haematology. 1986 November; 64(3): 561-9.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=3790445

- **Variant translocations in sporadic Burkitt's lymphoma detected in fresh tumour material: analysis of three cases.**
  Author(s): Cario G, Stadt UZ, Reiter A, Welte K, Sykora KW.
  Source: British Journal of Haematology. 2000 September; 110(3): 537-46.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=10997962

- **Variant translocations in two Burkitt's lymphoma cell lines are located in the MLV14 locus.**
  Author(s): Gallego MI, Lazo PA.
  Source: Genes, Chromosomes & Cancer. 1992 October; 5(3): 267-9.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=1384683

- **VH and VL gene analysis in sporadic Burkitt's lymphoma shows somatic hypermutation, intraclonal heterogeneity, and a role for antigen selection.**
  Author(s): Chapman CJ, Zhou JX, Gregory C, Rickinson AB, Stevenson FK.
  Source: Blood. 1996 November 1; 88(9): 3562-8.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=8896424

- **VH gene analysis in sporadic Burkitt's lymphoma: somatic mutation and intraclonal diversity with special reference to the tumor cells involving germinal center.**
  Author(s): Isobe K, Tamaru J, Nakamura S, Harigaya K, Mikata A, Ito H.
  Source: Leukemia & Lymphoma. 2002 January; 43(1): 159-64.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=11908722

- **Vimentin gene: expression in human lymphocytes and in Burkitt's lymphoma cells.**
  Author(s): Lilienbaum A, Legagneux V, Portier MM, Dellagi K, Paulin D.
  Source: The Embo Journal. 1986 November; 5(11): 2809-14.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=3792301

- **Viruses and cancer risks: outgrowth of Epstein-Barr virus-positive Burkitt's lymphoma in the immune host.**
  Author(s): Rickinson AB, Gregory CD, Young LS.
  Source: Med Oncol Tumor Pharmacother. 1987; 4(3-4): 177-86.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=2831439

- **Wanted: case reports of Burkitt's lymphoma in the U. S.**
  Author(s): Levine PH.
  Source: Med Times. 1971 February; 99(2): 124-6. No Abstract Available.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=5545985

- **What is Burkitt's lymphoma and when is it endemic?**
  Author(s): Wright DH.
  Source: Blood. 1999 January 15; 93(2): 758.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=10215347

- **What is Burkitt's lymphoma?**
  Author(s): Wright DH.
  Source: The Journal of Pathology. 1997 June; 182(2): 125-7.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=9274520

- **Xanthomatous pseudotumor of the small intestine following treatment for Burkitt's lymphoma.**
  Author(s): Ashfaq R, Timmons CF.
  Source: Archives of Pathology & Laboratory Medicine. 1992 March; 116(3): 299-301.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=1536618

CHAPTER 2. NUTRITION AND BURKITT'S LYMPHOMA

Overview

In this chapter, we will show you how to find studies dedicated specifically to nutrition and Burkitt's lymphoma.

Finding Nutrition Studies on Burkitt's Lymphoma

The National Institutes of Health's Office of Dietary Supplements (ODS) offers a searchable bibliographic database called the IBIDS (International Bibliographic Information on Dietary Supplements; National Institutes of Health, Building 31, Room 1B29, 31 Center Drive, MSC 2086, Bethesda, Maryland 20892-2086, Tel: 301-435-2920, Fax: 301-480-1845, E-mail: ods@nih.gov). The IBIDS contains over 460,000 scientific citations and summaries about dietary supplements and nutrition as well as references to published international, scientific literature on dietary supplements such as vitamins, minerals, and botanicals.[7] The IBIDS includes references and citations to both human and animal research studies.

As a service of the ODS, access to the IBIDS database is available free of charge at the following Web address: **http://ods.od.nih.gov/databases/ibids.html**. After entering the search area, you have three choices: (1) IBIDS Consumer Database, (2) Full IBIDS Database, or (3) Peer Reviewed Citations Only.

Now that you have selected a database, click on the "Advanced" tab. An advanced search allows you to retrieve up to 100 fully explained references in a comprehensive format. Type "Burkitt's lymphoma" (or synonyms) into the search box, and click "Go." To narrow the search, you can also select the "Title" field.

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[7] Adapted from **http://ods.od.nih.gov**. IBIDS is produced by the Office of Dietary Supplements (ODS) at the National Institutes of Health to assist the public, healthcare providers, educators, and researchers in locating credible, scientific information on dietary supplements. IBIDS was developed and will be maintained through an interagency partnership with the Food and Nutrition Information Center of the National Agricultural Library, U.S. Department of Agriculture.

The following information is typical of that found when using the "Full IBIDS Database" to search for "Burkitt's lymphoma" (or a synonym):

- **Activation of latent EBV via anti-IgG-triggered, second messenger pathways in the Burkitt's lymphoma cell line Akata.**
  Author(s): Department of Pharmacology, University of Massachusetts Medical School, Worcester 01655.
  Source: Daibata, M Humphreys, R E Takada, K Sairenji, T J-Immunol. 1990 June 15; 144(12): 4788-93 0022-1767

- **Altered growth phenotype of a Burkitt's lymphoma line following the introduction and stable expression of the EBNA 2A gene.**
  Source: Gordon, J Millsum, M J Finney, M Cairns, J A Guy, G R Gregory, C D Abbot, S D Rickinson, A B Wang, F Kieff, E Curr-Top-Microbiol-Immunol. 1988; 141149-56 0070-217X

- **Apoptosis and restriction of G(1)/S cell cycle by fenretinide in Burkitt's lymphoma mutu I cell line accessed with bcl-6 down-regulation.**
  Author(s): Department of Biochemistry and Molecular Biology, New York Medical College, Valhalla, New York, 10595, USA.
  Source: Hsieh, T Wu, J M Biochem-Biophys-Res-Commun. 2000 October 5; 276(3): 1295-301 0006-291X

- **Cell death induced by vincristine in the intestinal crypts of mice and in a human Burkitt's lymphoma cell line.**
  Author(s): Department of Pathology, University of Queensland Medical School, Herston, Brisbane, Australia.
  Source: Harmon, B V Takano, Y S Winterford, C M Potten, C S Cell-Prolif. 1992 November; 25(6): 523-36 0960-7722

- **Differential expression of the major histocompatibility antigen complex (MHC) on a series of Burkitt's lymphoma lines.**
  Author(s): Department of Microbiology, Fukui Medical School.
  Source: Yokochi, T Inoue, Y Iwata, H Miyadai, T Kimura, Y Microbiol-Immunol. 1987; 31(12): 1209-15 0385-5600

- **Effect of activation of the Epstein-Barr virus genome on expression of B cell differentiation antigens of Burkitt's lymphoma lines.**
  Author(s): Department of Microbiology, Fukui Medical School.
  Source: Yokochi, T Inoue, Y Iwata, H Miyadai, T Kimura, Y Microbiol-Immunol. 1988; 32(9): 957-64 0385-5600

- **Epidemiology of Burkitt's lymphoma in Enugu, Nigeria.**
  Author(s): Department of Paediatrics, University of Nigeria Teaching Hospital, PMB 01129, Enugu, Nigeria. udebue@infoweb.abs.net
  Source: Oguonu, T Emodi, I Kaine, W Ann-Trop-Paediatr. 2002 December; 22(4): 369-74 0272-4936

- **Establishment and characterization of a new human Burkitt's lymphoma cell line (WSU-BL).**
  Author(s): Department of Internal Medicine, Wayne State University School of Medicine, Detroit, MI.
  Source: Mohamed, A N Mohammad, R M Koop, B F al Katib, A Cancer. 1989 September 1; 64(5): 1041-8 0008-543X

- **Expression of normal and translocated c-myc alleles in Burkitt's lymphoma cells: evidence for different regulation.**
  Author(s): Institut fur Medizinische Mikrobiologie und Hygiene der Universitat Freiburg, FRG.
  Source: Eick, D Bornkamm, G W EMBO-J. 1989 July; 8(7): 1965-72 0261-4189

- **Expression of P0- and P3-RNA from the normal and translocated c-myc allele in Burkitt's lymphoma cells.**
  Author(s): Institut fur Klinische Molekularbiologie und Tumorgenetik, Hamatologikum der GSF, Munchen, Federal Republic of Germany.
  Source: Eick, D Polack, A Kofler, E Lenoir, G M Rickinson, A B Bornkamm, G W Oncogene. 1990 September; 5(9): 1397-402 0950-9232

- **Favorable response of pediatric AIDS-related Burkitt's lymphoma treated by aggressive chemotherapy.**
  Author(s): Institute of Hematology, Chaim Sheba Medical Center, Tel-Hashomer, Israel.
  Source: Neumann, Y Toren, A Mandel, M Martinowitz, U Varon, D Ramot, B Ben Bassat, I Rechavi, G Med-Pediatr-Oncol. 1993; 21(9): 661-4 0098-1532

- **Flavopiridol induces apoptosis and caspase-3 activation of a newly characterized Burkitt's lymphoma cell line containing mutant p53 genes.**
  Author(s): University of Maryland Greenebaum Cancer Center, 22 South Greene Street, Baltimore, MD 21201, USA. arapoport@umm.edu
  Source: Rapoport, A P Simons Evelyn, M Chen, T Sidell, R Luhowskyj, S Rosell, K Obrig, T Hicks, D Hinkle, P M Nahm, M Insel, R A Abboud, C N Blood-Cells-Mol-Dis. 2001 May-June; 27(3): 610-24 1079-9796

- **Hepatocyte growth factor (HGF) protects c-met-expressing Burkitt's lymphoma cell lines from apoptotic death induced by DNA damaging agents.**
  Author(s): Department of Clinical and Surgical Sciences, University of Edinburgh, Royal Infirmary, Edinburgh EH3 9YW, UK. g.skibinski@qub.ac.uk
  Source: Skibinski, G Skibinska, A James, K Eur-J-Cancer. 2001 August; 37(12): 1562-9 0959-8049

- **HiC-COM: a 2-month intensive chemotherapy regimen for children with stage III and IV Burkitt's lymphoma and B-cell acute lymphoblastic leukemia.**
  Author(s): Division of Pediatric Hematology/Oncology, Boston Floating Hospital, MA.
  Source: Schwenn, M R Blattner, S R Lynch, E Weinstein, H J J-Clin-Oncol. 1991 January; 9(1): 133-8 0732-183X

- **IL-4 induces LFA-1 and LFA-3 expression on Burkitt's lymphoma cell lines. Requirement of additional activation by phorbol myristate acetate for induction of homotypic cell adhesions.**
  Author(s): UNICET, Laboratory for Immunological Research, Dardilly, France.
  Source: Rousset, F Billaud, M Blanchard, D Figdor, C Lenoir, G M Spits, H De Vries, J E J-Immunol. 1989 September 1; 143(5): 1490-8 0022-1767

- **Induction of differentiation of African Burkitt's lymphoma cells by phorbol ester: possible relation with early B cells.**
  Source: Ho, Y S Subhendu, C Hsu, S M Cancer-Invest. 1987; 5(2): 101-7 0735-7907

- **Intensive, very short-term chemotherapy for advanced Burkitt's lymphoma in children.**
  Author(s): Department of Pediatric Oncology, Istituto Nazionale Tumori, Milan, Italy. f.spreafico@istitutotumori.mi.it
  Source: Spreafico, Filippo Massimino, Maura Luksch, Roberto Casanova, Michela Cefalo, Graziella S Collini, Paola Ferrari, Andrea Polastri, Daniela Terenziani, Monica

Gasparini, Marco Fossati Bellani, Franca J-Clin-Oncol. 2002 June 15; 20(12): 2783-8 0732-183X

- **Intraocular involvement of Burkitt's lymphoma in a Bedouin child.**
  Author(s): Department of Ophthalmology, Beilinson Medical Center, Petah Tiqva, Israel.
  Source: Wysenbeek, Y S Nissenkorn, I Cohen, S Ben Sira, I Stark, B Zaizov, R Pediatr-Hematol-Oncol. 1987; 4(4): 309-14 0888-0018

- **Loss of VLA-3 (CD49c/CD29) expression in two multidrug resistant Burkitt's lymphoma cell lines.**
  Author(s): Department of Pathology, Medizinische Hochschule Hannover, Germany.
  Source: Duensing, S Duensing, A Grosse, J Atzpodien, J Cancer-Biother-Radiopharm. 1998 October; 13(5): 369-73 1084-9785

- **Non-endemic Burkitt's lymphoma in a patient with Bloom's syndrome.**
  Author(s): Centre for Human Genetics, K. U. Leuven, Belgium.
  Source: Vandenberghe, E Van Hove, J Brock, P Schmidt, P Delabie, J Casteels Van Daele, M Cassiman, J J Vanderschueren Lodeweyckx, M Van den Berghe, H Leuk-Lymphoma. 1993 July; 10(4-5): 377-82 1042-8194

- **Non-Hodgkin's lymphoma protocols in the treatment of patients with Burkitt's lymphoma and lymphoblastic lymphoma: a report on 58 patients.**
  Author(s): Philipps-Universitat Marburg, Dept of Hematology/Oncology, Germany. kaiseru@mailer.uni-marburg.de
  Source: Kaiser, U Uebelacker, I Havemann, K Leuk-Lymphoma. 1999 December; 36(1-2): 101-8 1042-8194

- **Phorbol ester-induced inhibition of proliferation of Daudi Burkitt's lymphoma cells by impairment of cytokinesis.**
  Author(s): Department of Cellular and Molecular Sciences, St. George's Hospital Medical School, London, United Kingdom.
  Source: Menaya, J Clemens, M J Exp-Cell-Res. 1991 June; 194(2): 260-6 0014-4827

- **Plasma inter-alpha-trypsin inhibitor-related urinary glycoprotein EDC1 inhibits the growth of a Burkitt's lymphoma cell line.**
  Author(s): Department of Medicine, Emory University School of Medicine, Decatur, Georgia.
  Source: Chawla, R K Lawson, D H Travis, J J-Cell-Biochem. 1990 April; 42(4): 207-17 0730-2312

- **Potentiation of antiproliferative effects of monoclonal antibody Lym-1 and immunoconjugate Lym-1-gelonin on human Burkitt's lymphoma cells with gamma-interferon and tumor necrosis factor.**
  Author(s): Department of Oncology, Montefiore Medical Center, Bronx, NY 10467, USA.
  Source: O'Boyle, K P Colletti, D Mazurek, C Wang, Y Ray, S K Diamond, B Rosenblum, M G Epstein, A L Shochat, D Dutcher, J P et al. J-Immunother-Emphasis-Tumor-Immunol. 1995 November; 18(4): 221-30 1067-5582

- **Regulation of CD44-hyaluronan interactions in Burkitt's lymphoma and Epstein-Barr virus-transformed lymphoblastoid B cells by PMA and interleukin-4.**
  Author(s): Faculty of Medicine, University of Ottawa, Ottawa, Ontario, Canada.
  Source: Kryworuchko, M Gee, K Diaz Mitoma, F KuMarch, A Cell-Immunol. 1999 May 25; 194(1): 54-66 0008-8749

- **Regulatory elements in the immunoglobulin kappa locus induce c-myc activation and the promoter shift in Burkitt's lymphoma cells.**
  Author(s): Institut fur Klinische Molekularbiologie und Tumorgenetik GSF, Munchen, Germany.
  Source: Polack, A Feederle, R Klobeck, G Hortnagel, K EMBO-J. 1993 October; 12(10): 3913-20 0261-4189
- **Resistance to etoposide-induced apoptosis in a Burkitt's lymphoma cell line.**
  Author(s): Queensland Institute of Medical Research, PO Royal Brisbane Hospital, Herston, Australia.
  Source: Zhao, E G Song, Q Cross, S Misko, I Lees Miller, S P Lavin, M F Int-J-Cancer. 1998 August 31; 77(5): 755-62 0020-7136
- **Retinoic acid induces changes in c-fgr proto-oncogene mRNA levels in Burkitt's lymphoma cells.**
  Author(s): Department of Biochemistry and Molecular Biology, Bland Sutton Institute, University College and Middlesex School of Medicine, London, United Kingdom.
  Source: Faulkner, L Katz, D R Brickell, P M Immunobiology. 1993 August; 188(4-5): 460-8 0171-2985
- **The Epstein-Barr virus latent membrane protein 1 induces interleukin-10 in Burkitt's lymphoma cells but not in Hodgkin's cells involving the p38/SAPK2 pathway.**
  Author(s): Klinik fur Innere Medizin I, Zentrum fur Molekulare Medizin, Universitat zu Koln, D-50924 Cologne, Germany.
  Source: Vockerodt, M Haier, B Buttgereit, P Tesch, H Kube, D Virology. 2001 February 15; 280(2): 183-98 0042-6822
- **Truncation does not abrogate transcriptional downregulation of the c-myc gene by sodium butyrate in Burkitt's lymphoma cells.**
  Author(s): Abteilung fur Virologie, Universitat Freiburg, FRG.
  Source: Polack, A Eick, D Koch, E Bornkamm, G W EMBO-J. 1987 October; 6(10): 2959-64 0261-4189

Federal Resources on Nutrition

In addition to the IBIDS, the United States Department of Health and Human Services (HHS) and the United States Department of Agriculture (USDA) provide many sources of information on general nutrition and health. Recommended resources include:

- healthfinder®, HHS's gateway to health information, including diet and nutrition: **http://www.healthfinder.gov/scripts/SearchContext.asp?topic=238&page=0**
- The United States Department of Agriculture's Web site dedicated to nutrition information: **www.nutrition.gov**
- The Food and Drug Administration's Web site for federal food safety information: **www.foodsafety.gov**
- The National Action Plan on Overweight and Obesity sponsored by the United States Surgeon General: **http://www.surgeongeneral.gov/topics/obesity/**
- The Center for Food Safety and Applied Nutrition has an Internet site sponsored by the Food and Drug Administration and the Department of Health and Human Services: **http://vm.cfsan.fda.gov/**

- Center for Nutrition Policy and Promotion sponsored by the United States Department of Agriculture: **http://www.usda.gov/cnpp/**
- Food and Nutrition Information Center, National Agricultural Library sponsored by the United States Department of Agriculture: **http://www.nal.usda.gov/fnic/**
- Food and Nutrition Service sponsored by the United States Department of Agriculture: **http://www.fns.usda.gov/fns/**

Additional Web Resources

A number of additional Web sites offer encyclopedic information covering food and nutrition. The following is a representative sample:

- AOL: **http://search.aol.com/cat.adp?id=174&layer=&from=subcats**
- Family Village: **http://www.familyvillage.wisc.edu/med_nutrition.html**
- Google: **http://directory.google.com/Top/Health/Nutrition/**
- Healthnotes: **http://www.healthnotes.com/**
- Open Directory Project: **http://dmoz.org/Health/Nutrition/**
- Yahoo.com: **http://dir.yahoo.com/Health/Nutrition/**
- WebMD®Health: **http://my.webmd.com/nutrition**
- WholeHealthMD.com: **http://www.wholehealthmd.com/reflib/0,1529,00.html**

CHAPTER 3. ALTERNATIVE MEDICINE AND BURKITT'S LYMPHOMA

Overview

In this chapter, we will begin by introducing you to official information sources on complementary and alternative medicine (CAM) relating to Burkitt's lymphoma. At the conclusion of this chapter, we will provide additional sources.

National Center for Complementary and Alternative Medicine

The National Center for Complementary and Alternative Medicine (NCCAM) of the National Institutes of Health (**http://nccam.nih.gov/**) has created a link to the National Library of Medicine's databases to facilitate research for articles that specifically relate to Burkitt's lymphoma and complementary medicine. To search the database, go to the following Web site: **http://www.nlm.nih.gov/nccam/camonpubmed.html**. Select "CAM on PubMed." Enter "Burkitt's lymphoma" (or synonyms) into the search box. Click "Go." The following references provide information on particular aspects of complementary and alternative medicine that are related to Burkitt's lymphoma:

- **38.13: a monoclonal antibody directed against a Burkitt's lymphoma-associated antigen and its use as carrier for toxins.**
  Author(s): Wiels J, Balana A, Tursz T.
  Source: Iarc Sci Publ. 1985; (60): 457-64.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=4065950

- **Acute leukemias and Burkitt's lymphoma. Present status of therapy.**
  Author(s): Zubrod CG.
  Source: Cancer. 1968 April; 21(4): 553-7.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=4296158

- **Acute tumor lysis syndrome. A review of 37 patients with Burkitt's lymphoma.**
  Author(s): Cohen LF, Balow JE, Magrath IT, Poplack DG, Ziegler JL.

Source: The American Journal of Medicine. 1980 April; 68(4): 486-91.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=7369230

- **Advanced stage (III-IV) Burkitt's lymphoma and B-cell acute lymphoblastic leukaemia in children: kinetic and pharmacologic rationale for treatment and recent results (1979-1983).**
  Author(s): Murphy SB, Bowman WP, Hustu HO, Berard CW.
  Source: Iarc Sci Publ. 1985; (60): 405-18.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=3905592

- **African Burkitt's lymphoma and an Epstein-Barr virus-enhancing plant Euphorbia tirucalli.**
  Author(s): Osato T, Mizuno F, Imai S, Aya T, Koizumi S, Kinoshita T, Tokuda H, Ito Y, Hirai N, Hirota M, et al.
  Source: Lancet. 1987 May 30; 1(8544): 1257-8.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=2884382

- **African Burkitt's lymphoma: case report and light and electron microscopic findings.**
  Author(s): Yih WY, Myers SL, Meshul CK, Bartley MH.
  Source: Oral Surg Oral Med Oral Pathol. 1990 December; 70(6): 760-4.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=2263336

- **American Burkitt's lymphoma: a clinicopathologic study of 30 cases. I. Clinical factors relating to prolonged survival.**
  Author(s): Arseneau JC, Canellos GP, Banks PM, Berard CW, Gralnick HR, DeVita VT Jr.
  Source: The American Journal of Medicine. 1975 March; 58(3): 314-21.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=1115074

- **An international evaluation of CODOX-M and CODOX-M alternating with IVAC in adult Burkitt's lymphoma: results of United Kingdom Lymphoma Group LY06 study.**
  Author(s): Mead GM, Sydes MR, Walewski J, Grigg A, Hatton CS, Pescosta N, Guarnaccia C, Lewis MS, McKendrick J, Stenning SP, Wright D, Norbert P; UKLG LY06 collaborators.
  Source: Annals of Oncology : Official Journal of the European Society for Medical Oncology / Esmo. 2002 August; 13(8): 1264-74. Erratum In: Ann Oncol. 2002 December; 13(12): 1961. Norbert P [corrected to Pescosta N].
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=12181251

- **Antibodies to Epstein-Barr-virus antigens before and after the development of Burkitt's lymphoma in a patient treated for Hodgkin's disease.**
  Author(s): Magrath I, Henle W, Owor R, Olweny C.

Source: The New England Journal of Medicine. 1975 March 20; 292(12): 621-3.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=163432

- **Anticancer activities of curcumin on human Burkitt's lymphoma.**
  Author(s): Wu Y, Chen Y, Xu J, Lu L.
  Source: Zhonghua Zhong Liu Za Zhi. 2002 July; 24(4): 348-52.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=12408761

- **Are plant factors a missing link in the evolution of endemic Burkitt's lymphoma?**
  Author(s): van den Bosch C, Griffin BE, Kazembe P, Dziweni C, Kadzamira L.
  Source: British Journal of Cancer. 1993 December; 68(6): 1232-5.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=8260378

- **Autologous bone-marrow transplantation for Burkitt's lymphoma: marrow purging with anti-Y 29/55 monoclonal antibody and complement.**
  Author(s): Baumgartner C, Delaleu B, Imbach P, Luthy A, Odavic R, Brun del Re G, Bucher U, Forster HK, Hirt A, Wagner HP.
  Source: Iarc Sci Publ. 1985; (60): 435-40.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=3905594

- **Burkitt's lymphoma in a 45-year-old Nigerian woman.**
  Author(s): Lawal OO, Ojo OS, Durosinmi MA.
  Source: Trop Geogr Med. 1990 July; 42(3): 294-7.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=2293442

- **Burkitt's lymphoma in an American youth: usefulness of radionuclide imaging in diagnosis and measuring the effects of treatment.**
  Author(s): Kim E, Herman T.
  Source: Clinical Pediatrics. 1977 September; 16(9): 805-7.
  http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=pubmed&dopt=Abstract&list_uids=891082

- **Burkitt's lymphoma in Ghana: clinical features and response to chemotherapy.**
  Author(s): Nkrumah FK, Perkins IV.
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Additional Web Resources

A number of additional Web sites offer encyclopedic information covering CAM and related topics. The following is a representative sample:

- Alternative Medicine Foundation, Inc.: **http://www.herbmed.org/**
- AOL: **http://search.aol.com/cat.adp?id=169&layer=&from=subcats**
- Chinese Medicine: **http://www.newcenturynutrition.com/**
- drkoop.com®: **http://www.drkoop.com/InteractiveMedicine/IndexC.html**
- Family Village: **http://www.familyvillage.wisc.edu/med_altn.htm**
- Google: **http://directory.google.com/Top/Health/Alternative/**
- Healthnotes: **http://www.healthnotes.com/**
- MedWebPlus: **http://medwebplus.com/subject/Alternative_and_Complementary_Medicine**
- Open Directory Project: **http://dmoz.org/Health/Alternative/**
- HealthGate: **http://www.tnp.com/**
- WebMD®Health: **http://my.webmd.com/drugs_and_herbs**
- WholeHealthMD.com: **http://www.wholehealthmd.com/reflib/0,1529,00.html**
- Yahoo.com: **http://dir.yahoo.com/Health/Alternative_Medicine/**

General References

A good place to find general background information on CAM is the National Library of Medicine. It has prepared within the MEDLINEplus system an information topic page dedicated to complementary and alternative medicine. To access this page, go to the MEDLINEplus site at **http://www.nlm.nih.gov/medlineplus/alternativemedicine.html**. This Web site provides a general overview of various topics and can lead to a number of general sources.

CHAPTER 4. PERIODICALS AND NEWS ON BURKITT'S LYMPHOMA

Overview

In this chapter, we suggest a number of news sources and present various periodicals that cover Burkitt's lymphoma.

News Services and Press Releases

One of the simplest ways of tracking press releases on Burkitt's lymphoma is to search the news wires. In the following sample of sources, we will briefly describe how to access each service. These services only post recent news intended for public viewing.

PR Newswire

To access the PR Newswire archive, simply go to **http://www.prnewswire.com/**. Select your country. Type "Burkitt's lymphoma" (or synonyms) into the search box. You will automatically receive information on relevant news releases posted within the last 30 days. The search results are shown by order of relevance.

Reuters Health

The Reuters' Medical News and Health eLine databases can be very useful in exploring news archives relating to Burkitt's lymphoma. While some of the listed articles are free to view, others are available for purchase for a nominal fee. To access this archive, go to **http://www.reutershealth.com/en/index.html** and search by "Burkitt's lymphoma" (or synonyms). The following was recently listed in this archive for Burkitt's lymphoma:

- **Prozac kills Burkitt's lymphoma cells: scientists**
  Source: Reuters Health eLine
  Date: April 15, 2003

- **Decline in Burkitt's lymphoma reported in Nigeria**
  Source: Reuters Health eLine
  Date: July 29, 2002

The NIH

Within MEDLINEplus, the NIH has made an agreement with the New York Times Syndicate, the AP News Service, and Reuters to deliver news that can be browsed by the public. Search news releases at **http://www.nlm.nih.gov/medlineplus/alphanews_a.html**. MEDLINEplus allows you to browse across an alphabetical index. Or you can search by date at the following Web page: **http://www.nlm.nih.gov/medlineplus/newsbydate.html**. Often, news items are indexed by MEDLINEplus within its search engine.

Business Wire

Business Wire is similar to PR Newswire. To access this archive, simply go to **http://www.businesswire.com/**. You can scan the news by industry category or company name.

Market Wire

Market Wire is more focused on technology than the other wires. To browse the latest press releases by topic, such as alternative medicine, biotechnology, fitness, healthcare, legal, nutrition, and pharmaceuticals, access Market Wire's Medical/Health channel at **http://www.marketwire.com/mw/release_index?channel=MedicalHealth**. Or simply go to Market Wire's home page at **http://www.marketwire.com/mw/home**, type "Burkitt's lymphoma" (or synonyms) into the search box, and click on "Search News." As this service is technology oriented, you may wish to use it when searching for press releases covering diagnostic procedures or tests.

Search Engines

Medical news is also available in the news sections of commercial Internet search engines. See the health news page at Yahoo (**http://dir.yahoo.com/Health/News_and_Media/**), or you can use this Web site's general news search page at **http://news.yahoo.com/**. Type in "Burkitt's lymphoma" (or synonyms). If you know the name of a company that is relevant to Burkitt's lymphoma, you can go to any stock trading Web site (such as **http://www.etrade.com/**) and search for the company name there. News items across various news sources are reported on indicated hyperlinks. Google offers a similar service at **http://news.google.com/**.

BBC

Covering news from a more European perspective, the British Broadcasting Corporation (BBC) allows the public free access to their news archive located at **http://www.bbc.co.uk/**. Search by "Burkitt's lymphoma" (or synonyms).

Academic Periodicals covering Burkitt's Lymphoma

Numerous periodicals are currently indexed within the National Library of Medicine's PubMed database that are known to publish articles relating to Burkitt's lymphoma. In addition to these sources, you can search for articles covering Burkitt's lymphoma that have been published by any of the periodicals listed in previous chapters. To find the latest studies published, go to **http://www.ncbi.nlm.nih.gov/pubmed**, type the name of the periodical into the search box, and click "Go."

If you want complete details about the historical contents of a journal, you can also visit the following Web site: **http://www.ncbi.nlm.nih.gov/entrez/jrbrowser.cgi**. Here, type in the name of the journal or its abbreviation, and you will receive an index of published articles. At **http://locatorplus.gov/**, you can retrieve more indexing information on medical periodicals (e.g. the name of the publisher). Select the button "Search LOCATORplus." Then type in the name of the journal and select the advanced search option "Journal Title Search."

APPENDICES

APPENDIX A. PHYSICIAN RESOURCES

Overview

In this chapter, we focus on databases and Internet-based guidelines and information resources created or written for a professional audience.

NIH Guidelines

Commonly referred to as "clinical" or "professional" guidelines, the National Institutes of Health publish physician guidelines for the most common diseases. Publications are available at the following by relevant Institute[8]:

- Office of the Director (OD); guidelines consolidated across agencies available at **http://www.nih.gov/health/consumer/conkey.htm**
- National Institute of General Medical Sciences (NIGMS); fact sheets available at **http://www.nigms.nih.gov/news/facts/**
- National Library of Medicine (NLM); extensive encyclopedia (A.D.A.M., Inc.) with guidelines: **http://www.nlm.nih.gov/medlineplus/healthtopics.html**
- National Cancer Institute (NCI); guidelines available at **http://www.cancer.gov/cancerinfo/list.aspx?viewid=5f35036e-5497-4d86-8c2c-714a9f7c8d25**
- National Eye Institute (NEI); guidelines available at **http://www.nei.nih.gov/order/index.htm**
- National Heart, Lung, and Blood Institute (NHLBI); guidelines available at **http://www.nhlbi.nih.gov/guidelines/index.htm**
- National Human Genome Research Institute (NHGRI); research available at **http://www.genome.gov/page.cfm?pageID=10000375**
- National Institute on Aging (NIA); guidelines available at **http://www.nia.nih.gov/health/**

[8] These publications are typically written by one or more of the various NIH Institutes.

- National Institute on Alcohol Abuse and Alcoholism (NIAAA); guidelines available at **http://www.niaaa.nih.gov/publications/publications.htm**
- National Institute of Allergy and Infectious Diseases (NIAID); guidelines available at **http://www.niaid.nih.gov/publications/**
- National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS); fact sheets and guidelines available at **http://www.niams.nih.gov/hi/index.htm**
- National Institute of Child Health and Human Development (NICHD); guidelines available at **http://www.nichd.nih.gov/publications/pubskey.cfm**
- National Institute on Deafness and Other Communication Disorders (NIDCD); fact sheets and guidelines at **http://www.nidcd.nih.gov/health/**
- National Institute of Dental and Craniofacial Research (NIDCR); guidelines available at **http://www.nidr.nih.gov/health/**
- National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK); guidelines available at **http://www.niddk.nih.gov/health/health.htm**
- National Institute on Drug Abuse (NIDA); guidelines available at **http://www.nida.nih.gov/DrugAbuse.html**
- National Institute of Environmental Health Sciences (NIEHS); environmental health information available at **http://www.niehs.nih.gov/external/facts.htm**
- National Institute of Mental Health (NIMH); guidelines available at **http://www.nimh.nih.gov/practitioners/index.cfm**
- National Institute of Neurological Disorders and Stroke (NINDS); neurological disorder information pages available at **http://www.ninds.nih.gov/health_and_medical/disorder_index.htm**
- National Institute of Nursing Research (NINR); publications on selected illnesses at **http://www.nih.gov/ninr/news-info/publications.html**
- National Institute of Biomedical Imaging and Bioengineering; general information at **http://grants.nih.gov/grants/becon/becon_info.htm**
- Center for Information Technology (CIT); referrals to other agencies based on keyword searches available at **http://kb.nih.gov/www_query_main.asp**
- National Center for Complementary and Alternative Medicine (NCCAM); health information available at **http://nccam.nih.gov/health/**
- National Center for Research Resources (NCRR); various information directories available at **http://www.ncrr.nih.gov/publications.asp**
- Office of Rare Diseases; various fact sheets available at **http://rarediseases.info.nih.gov/html/resources/rep_pubs.html**
- Centers for Disease Control and Prevention; various fact sheets on infectious diseases available at **http://www.cdc.gov/publications.htm**

NIH Databases

In addition to the various Institutes of Health that publish professional guidelines, the NIH has designed a number of databases for professionals.[9] Physician-oriented resources provide a wide variety of information related to the biomedical and health sciences, both past and present. The format of these resources varies. Searchable databases, bibliographic citations, full-text articles (when available), archival collections, and images are all available. The following are referenced by the National Library of Medicine:[10]

- **Bioethics:** Access to published literature on the ethical, legal, and public policy issues surrounding healthcare and biomedical research. This information is provided in conjunction with the Kennedy Institute of Ethics located at Georgetown University, Washington, D.C.: **http://www.nlm.nih.gov/databases/databases_bioethics.html**
- **HIV/AIDS Resources:** Describes various links and databases dedicated to HIV/AIDS research: **http://www.nlm.nih.gov/pubs/factsheets/aidsinfs.html**
- **NLM Online Exhibitions:** Describes "Exhibitions in the History of Medicine": **http://www.nlm.nih.gov/exhibition/exhibition.html**. Additional resources for historical scholarship in medicine: **http://www.nlm.nih.gov/hmd/hmd.html**
- **Biotechnology Information:** Access to public databases. The National Center for Biotechnology Information conducts research in computational biology, develops software tools for analyzing genome data, and disseminates biomedical information for the better understanding of molecular processes affecting human health and disease: **http://www.ncbi.nlm.nih.gov/**
- **Population Information:** The National Library of Medicine provides access to worldwide coverage of population, family planning, and related health issues, including family planning technology and programs, fertility, and population law and policy: **http://www.nlm.nih.gov/databases/databases_population.html**
- **Cancer Information:** Access to cancer-oriented databases: **http://www.nlm.nih.gov/databases/databases_cancer.html**
- **Profiles in Science:** Offering the archival collections of prominent twentieth-century biomedical scientists to the public through modern digital technology: **http://www.profiles.nlm.nih.gov/**
- **Chemical Information:** Provides links to various chemical databases and references: **http://sis.nlm.nih.gov/Chem/ChemMain.html**
- **Clinical Alerts:** Reports the release of findings from the NIH-funded clinical trials where such release could significantly affect morbidity and mortality: **http://www.nlm.nih.gov/databases/alerts/clinical_alerts.html**
- **Space Life Sciences:** Provides links and information to space-based research (including NASA): **http://www.nlm.nih.gov/databases/databases_space.html**
- **MEDLINE:** Bibliographic database covering the fields of medicine, nursing, dentistry, veterinary medicine, the healthcare system, and the pre-clinical sciences: **http://www.nlm.nih.gov/databases/databases_medline.html**

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[9] Remember, for the general public, the National Library of Medicine recommends the databases referenced in MEDLINE*plus* (**http://medlineplus.gov/** or **http://www.nlm.nih.gov/medlineplus/databases.html**).

[10] See **http://www.nlm.nih.gov/databases/databases.html**.

- **Toxicology and Environmental Health Information (TOXNET):** Databases covering toxicology and environmental health: **http://sis.nlm.nih.gov/Tox/ToxMain.html**
- **Visible Human Interface:** Anatomically detailed, three-dimensional representations of normal male and female human bodies: **http://www.nlm.nih.gov/research/visible/visible_human.html**

The NLM Gateway[11]

The NLM (National Library of Medicine) Gateway is a Web-based system that lets users search simultaneously in multiple retrieval systems at the U.S. National Library of Medicine (NLM). It allows users of NLM services to initiate searches from one Web interface, providing one-stop searching for many of NLM's information resources or databases.[12] To use the NLM Gateway, simply go to the search site at **http://gateway.nlm.nih.gov/gw/Cmd**. Type "Burkitt's lymphoma" (or synonyms) into the search box and click "Search." The results will be presented in a tabular form, indicating the number of references in each database category.

Results Summary

Category	Items Found
Journal Articles	8058
Books / Periodicals / Audio Visual	53
Consumer Health	478
Meeting Abstracts	48
Other Collections	20
Total	8657

HSTAT[13]

HSTAT is a free, Web-based resource that provides access to full-text documents used in healthcare decision-making.[14] These documents include clinical practice guidelines, quick-reference guides for clinicians, consumer health brochures, evidence reports and technology assessments from the Agency for Healthcare Research and Quality (AHRQ), as well as AHRQ's Put Prevention Into Practice.[15] Simply search by "Burkitt's lymphoma" (or synonyms) at the following Web site: **http://text.nlm.nih.gov**.

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[11] Adapted from NLM: **http://gateway.nlm.nih.gov/gw/Cmd?Overview.x**.

[12] The NLM Gateway is currently being developed by the Lister Hill National Center for Biomedical Communications (LHNCBC) at the National Library of Medicine (NLM) of the National Institutes of Health (NIH).

[13] Adapted from HSTAT: **http://www.nlm.nih.gov/pubs/factsheets/hstat.html**.

[14] The HSTAT URL is **http://hstat.nlm.nih.gov/**.

[15] Other important documents in HSTAT include: the National Institutes of Health (NIH) Consensus Conference Reports and Technology Assessment Reports; the HIV/AIDS Treatment Information Service (ATIS) resource documents; the Substance Abuse and Mental Health Services Administration's Center for Substance Abuse Treatment (SAMHSA/CSAT) Treatment Improvement Protocols (TIP) and Center for Substance Abuse Prevention (SAMHSA/CSAP) Prevention Enhancement Protocols System (PEPS); the Public Health Service (PHS) Preventive Services Task Force's *Guide to Clinical Preventive Services*; the independent, nonfederal Task Force on Community Services' *Guide to Community Preventive Services*; and the Health Technology Advisory Committee (HTAC) of the Minnesota Health Care Commission (MHCC) health technology evaluations.

Coffee Break: Tutorials for Biologists[16]

Coffee Break is a general healthcare site that takes a scientific view of the news and covers recent breakthroughs in biology that may one day assist physicians in developing treatments. Here you will find a collection of short reports on recent biological discoveries. Each report incorporates interactive tutorials that demonstrate how bioinformatics tools are used as a part of the research process. Currently, all Coffee Breaks are written by NCBI staff.[17] Each report is about 400 words and is usually based on a discovery reported in one or more articles from recently published, peer-reviewed literature.[18] This site has new articles every few weeks, so it can be considered an online magazine of sorts. It is intended for general background information. You can access the Coffee Break Web site at the following hyperlink: **http://www.ncbi.nlm.nih.gov/Coffeebreak/**.

Other Commercial Databases

In addition to resources maintained by official agencies, other databases exist that are commercial ventures addressing medical professionals. Here are some examples that may interest you:

- **CliniWeb International:** Index and table of contents to selected clinical information on the Internet; see **http://www.ohsu.edu/cliniweb/**.
- **Medical World Search:** Searches full text from thousands of selected medical sites on the Internet; see **http://www.mwsearch.com/**.

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[16] Adapted from **http://www.ncbi.nlm.nih.gov/Coffeebreak/Archive/FAQ.html**.

[17] The figure that accompanies each article is frequently supplied by an expert external to NCBI, in which case the source of the figure is cited. The result is an interactive tutorial that tells a biological story.

[18] After a brief introduction that sets the work described into a broader context, the report focuses on how a molecular understanding can provide explanations of observed biology and lead to therapies for diseases. Each vignette is accompanied by a figure and hypertext links that lead to a series of pages that interactively show how NCBI tools and resources are used in the research process.

APPENDIX B. PATIENT RESOURCES

Overview

Official agencies, as well as federally funded institutions supported by national grants, frequently publish a variety of guidelines written with the patient in mind. These are typically called "Fact Sheets" or "Guidelines." They can take the form of a brochure, information kit, pamphlet, or flyer. Often they are only a few pages in length. Since new guidelines on Burkitt's lymphoma can appear at any moment and be published by a number of sources, the best approach to finding guidelines is to systematically scan the Internet-based services that post them.

Patient Guideline Sources

The remainder of this chapter directs you to sources which either publish or can help you find additional guidelines on topics related to Burkitt's lymphoma. Due to space limitations, these sources are listed in a concise manner. Do not hesitate to consult the following sources by either using the Internet hyperlink provided, or, in cases where the contact information is provided, contacting the publisher or author directly.

The National Institutes of Health

The NIH gateway to patients is located at **http://health.nih.gov/**. From this site, you can search across various sources and institutes, a number of which are summarized below.

Topic Pages: MEDLINEplus

The National Library of Medicine has created a vast and patient-oriented healthcare information portal called MEDLINEplus. Within this Internet-based system are "health topic pages" which list links to available materials relevant to Burkitt's lymphoma. To access this system, log on to **http://www.nlm.nih.gov/medlineplus/healthtopics.html**. From there you can either search using the alphabetical index or browse by broad topic areas. Recently, MEDLINEplus listed the following when searched for "Burkitt's lymphoma":

Hodgkin's Disease
http://www.nlm.nih.gov/medlineplus/hodgkinsdisease.html

Lymphoma
http://www.nlm.nih.gov/medlineplus/lymphoma.html

You may also choose to use the search utility provided by MEDLINEplus at the following Web address: **http://www.nlm.nih.gov/medlineplus/**. Simply type a keyword into the search box and click "Search." This utility is similar to the NIH search utility, with the exception that it only includes materials that are linked within the MEDLINEplus system (mostly patient-oriented information). It also has the disadvantage of generating unstructured results. We recommend, therefore, that you use this method only if you have a very targeted search.

The NIH Search Utility

The NIH search utility allows you to search for documents on over 100 selected Web sites that comprise the NIH-WEB-SPACE. Each of these servers is "crawled" and indexed on an ongoing basis. Your search will produce a list of various documents, all of which will relate in some way to Burkitt's lymphoma. The drawbacks of this approach are that the information is not organized by theme and that the references are often a mix of information for professionals and patients. Nevertheless, a large number of the listed Web sites provide useful background information. We can only recommend this route, therefore, for relatively rare or specific disorders, or when using highly targeted searches. To use the NIH search utility, visit the following Web page: **http://search.nih.gov/index.html**.

Additional Web Sources

A number of Web sites are available to the public that often link to government sites. These can also point you in the direction of essential information. The following is a representative sample:

- AOL: **http://search.aol.com/cat.adp?id=168&layer=&from=subcats**
- Family Village: **http://www.familyvillage.wisc.edu/specific.htm**
- Google: **http://directory.google.com/Top/Health/Conditions_and_Diseases/**
- Med Help International: **http://www.medhelp.org/HealthTopics/A.html**
- Open Directory Project: **http://dmoz.org/Health/Conditions_and_Diseases/**
- Yahoo.com: **http://dir.yahoo.com/Health/Diseases_and_Conditions/**
- WebMD®Health: **http://my.webmd.com/health_topics**

Finding Associations

There are several Internet directories that provide lists of medical associations with information on or resources relating to Burkitt's lymphoma. By consulting all of associations listed in this chapter, you will have nearly exhausted all sources for patient associations concerned with Burkitt's lymphoma.

The National Health Information Center (NHIC)

The National Health Information Center (NHIC) offers a free referral service to help people find organizations that provide information about Burkitt's lymphoma. For more information, see the NHIC's Web site at **http://www.health.gov/NHIC/** or contact an information specialist by calling 1-800-336-4797.

Directory of Health Organizations

The Directory of Health Organizations, provided by the National Library of Medicine Specialized Information Services, is a comprehensive source of information on associations. The Directory of Health Organizations database can be accessed via the Internet at **http://www.sis.nlm.nih.gov/Dir/DirMain.html**. It is composed of two parts: DIRLINE and Health Hotlines.

The DIRLINE database comprises some 10,000 records of organizations, research centers, and government institutes and associations that primarily focus on health and biomedicine. To access DIRLINE directly, go to the following Web site: **http://dirline.nlm.nih.gov/**. Simply type in "Burkitt's lymphoma" (or a synonym), and you will receive information on all relevant organizations listed in the database.

Health Hotlines directs you to toll-free numbers to over 300 organizations. You can access this database directly at **http://www.sis.nlm.nih.gov/hotlines/**. On this page, you are given the option to search by keyword or by browsing the subject list. When you have received your search results, click on the name of the organization for its description and contact information.

The Combined Health Information Database

Another comprehensive source of information on healthcare associations is the Combined Health Information Database. Using the "Detailed Search" option, you will need to limit your search to "Organizations" and "Burkitt's lymphoma". Type the following hyperlink into your Web browser: **http://chid.nih.gov/detail/detail.html**. To find associations, use the drop boxes at the bottom of the search page where "You may refine your search by." For publication date, select "All Years." Then, select your preferred language and the format option "Organization Resource Sheet." Type "Burkitt's lymphoma" (or synonyms) into the "For these words:" box. You should check back periodically with this database since it is updated every three months.

The National Organization for Rare Disorders, Inc.

The National Organization for Rare Disorders, Inc. has prepared a Web site that provides, at no charge, lists of associations organized by health topic. You can access this database at the following Web site: **http://www.rarediseases.org/search/orgsearch.html**. Type "Burkitt's lymphoma" (or a synonym) into the search box, and click "Submit Query."

APPENDIX C. FINDING MEDICAL LIBRARIES

Overview

In this Appendix, we show you how to quickly find a medical library in your area.

Preparation

Your local public library and medical libraries have interlibrary loan programs with the National Library of Medicine (NLM), one of the largest medical collections in the world. According to the NLM, most of the literature in the general and historical collections of the National Library of Medicine is available on interlibrary loan to any library. If you would like to access NLM medical literature, then visit a library in your area that can request the publications for you.[19]

Finding a Local Medical Library

The quickest method to locate medical libraries is to use the Internet-based directory published by the National Network of Libraries of Medicine (NN/LM). This network includes 4626 members and affiliates that provide many services to librarians, health professionals, and the public. To find a library in your area, simply visit **http://nnlm.gov/members/adv.html** or call 1-800-338-7657.

Medical Libraries in the U.S. and Canada

In addition to the NN/LM, the National Library of Medicine (NLM) lists a number of libraries with reference facilities that are open to the public. The following is the NLM's list and includes hyperlinks to each library's Web site. These Web pages can provide information on hours of operation and other restrictions. The list below is a small sample of

[19] Adapted from the NLM: **http://www.nlm.nih.gov/psd/cas/interlibrary.html**.

libraries recommended by the National Library of Medicine (sorted alphabetically by name of the U.S. state or Canadian province where the library is located)[20]:

- **Alabama:** Health InfoNet of Jefferson County (Jefferson County Library Cooperative, Lister Hill Library of the Health Sciences), **http://www.uab.edu/infonet/**
- **Alabama:** Richard M. Scrushy Library (American Sports Medicine Institute)
- **Arizona:** Samaritan Regional Medical Center: The Learning Center (Samaritan Health System, Phoenix, Arizona), **http://www.samaritan.edu/library/bannerlibs.htm**
- **California:** Kris Kelly Health Information Center (St. Joseph Health System, Humboldt), **http://www.humboldt1.com/~kkhic/index.html**
- **California:** Community Health Library of Los Gatos, **http://www.healthlib.org/orgresources.html**
- **California:** Consumer Health Program and Services (CHIPS) (County of Los Angeles Public Library, Los Angeles County Harbor-UCLA Medical Center Library) - Carson, CA, **http://www.colapublib.org/services/chips.html**
- **California:** Gateway Health Library (Sutter Gould Medical Foundation)
- **California:** Health Library (Stanford University Medical Center), **http://www-med.stanford.edu/healthlibrary/**
- **California:** Patient Education Resource Center - Health Information and Resources (University of California, San Francisco), **http://sfghdean.ucsf.edu/barnett/PERC/default.asp**
- **California:** Redwood Health Library (Petaluma Health Care District), **http://www.phcd.org/rdwdlib.html**
- **California:** Los Gatos PlaneTree Health Library, **http://planetreesanjose.org/**
- **California:** Sutter Resource Library (Sutter Hospitals Foundation, Sacramento), **http://suttermedicalcenter.org/library/**
- **California:** Health Sciences Libraries (University of California, Davis), **http://www.lib.ucdavis.edu/healthsci/**
- **California:** ValleyCare Health Library & Ryan Comer Cancer Resource Center (ValleyCare Health System, Pleasanton), **http://gaelnet.stmarys-ca.edu/other.libs/gbal/east/vchl.html**
- **California:** Washington Community Health Resource Library (Fremont), **http://www.healthlibrary.org/**
- **Colorado:** William V. Gervasini Memorial Library (Exempla Healthcare), **http://www.saintjosephdenver.org/yourhealth/libraries/**
- **Connecticut:** Hartford Hospital Health Science Libraries (Hartford Hospital), **http://www.harthosp.org/library/**
- **Connecticut:** Healthnet: Connecticut Consumer Health Information Center (University of Connecticut Health Center, Lyman Maynard Stowe Library), **http://library.uchc.edu/departm/hnet/**

[20] Abstracted from **http://www.nlm.nih.gov/medlineplus/libraries.html**.

- **Connecticut:** Waterbury Hospital Health Center Library (Waterbury Hospital, Waterbury), **http://www.waterburyhospital.com/library/consumer.shtml**
- **Delaware:** Consumer Health Library (Christiana Care Health System, Eugene du Pont Preventive Medicine & Rehabilitation Institute, Wilmington), **http://www.christianacare.org/health_guide/health_guide_pmri_health_info.cfm**
- **Delaware:** Lewis B. Flinn Library (Delaware Academy of Medicine, Wilmington), **http://www.delamed.org/chls.html**
- **Georgia:** Family Resource Library (Medical College of Georgia, Augusta), **http://cmc.mcg.edu/kids_families/fam_resources/fam_res_lib/frl.htm**
- **Georgia:** Health Resource Center (Medical Center of Central Georgia, Macon), **http://www.mccg.org/hrc/hrchome.asp**
- **Hawaii:** Hawaii Medical Library: Consumer Health Information Service (Hawaii Medical Library, Honolulu), **http://hml.org/CHIS/**
- **Idaho:** DeArmond Consumer Health Library (Kootenai Medical Center, Coeur d'Alene), **http://www.nicon.org/DeArmond/index.htm**
- **Illinois:** Health Learning Center of Northwestern Memorial Hospital (Chicago), **http://www.nmh.org/health_info/hlc.html**
- **Illinois:** Medical Library (OSF Saint Francis Medical Center, Peoria), **http://www.osfsaintfrancis.org/general/library/**
- **Kentucky:** Medical Library - Services for Patients, Families, Students & the Public (Central Baptist Hospital, Lexington), **http://www.centralbap.com/education/community/library.cfm**
- **Kentucky:** University of Kentucky - Health Information Library (Chandler Medical Center, Lexington), **http://www.mc.uky.edu/PatientEd/**
- **Louisiana:** Alton Ochsner Medical Foundation Library (Alton Ochsner Medical Foundation, New Orleans), **http://www.ochsner.org/library/**
- **Louisiana:** Louisiana State University Health Sciences Center Medical Library-Shreveport, **http://lib-sh.lsuhsc.edu/**
- **Maine:** Franklin Memorial Hospital Medical Library (Franklin Memorial Hospital, Farmington), **http://www.fchn.org/fmh/lib.htm**
- **Maine:** Gerrish-True Health Sciences Library (Central Maine Medical Center, Lewiston), **http://www.cmmc.org/library/library.html**
- **Maine:** Hadley Parrot Health Science Library (Eastern Maine Healthcare, Bangor), **http://www.emh.org/hll/hpl/guide.htm**
- **Maine:** Maine Medical Center Library (Maine Medical Center, Portland), **http://www.mmc.org/library/**
- **Maine:** Parkview Hospital (Brunswick), **http://www.parkviewhospital.org/**
- **Maine:** Southern Maine Medical Center Health Sciences Library (Southern Maine Medical Center, Biddeford), **http://www.smmc.org/services/service.php3?choice=10**
- **Maine:** Stephens Memorial Hospital's Health Information Library (Western Maine Health, Norway), **http://www.wmhcc.org/Library/**

- **Manitoba, Canada:** Consumer & Patient Health Information Service (University of Manitoba Libraries), **http://www.umanitoba.ca/libraries/units/health/reference/chis.html**
- **Manitoba, Canada:** J.W. Crane Memorial Library (Deer Lodge Centre, Winnipeg), **http://www.deerlodge.mb.ca/crane_library/about.asp**
- **Maryland:** Health Information Center at the Wheaton Regional Library (Montgomery County, Dept. of Public Libraries, Wheaton Regional Library), **http://www.mont.lib.md.us/healthinfo/hic.asp**
- **Massachusetts:** Baystate Medical Center Library (Baystate Health System), **http://www.baystatehealth.com/1024/**
- **Massachusetts:** Boston University Medical Center Alumni Medical Library (Boston University Medical Center), **http://med-libwww.bu.edu/library/lib.html**
- **Massachusetts:** Lowell General Hospital Health Sciences Library (Lowell General Hospital, Lowell), **http://www.lowellgeneral.org/library/HomePageLinks/WWW.htm**
- **Massachusetts:** Paul E. Woodard Health Sciences Library (New England Baptist Hospital, Boston), **http://www.nebh.org/health_lib.asp**
- **Massachusetts:** St. Luke's Hospital Health Sciences Library (St. Luke's Hospital, Southcoast Health System, New Bedford), **http://www.southcoast.org/library/**
- **Massachusetts:** Treadwell Library Consumer Health Reference Center (Massachusetts General Hospital), **http://www.mgh.harvard.edu/library/chrcindex.html**
- **Massachusetts:** UMass HealthNet (University of Massachusetts Medical School, Worchester), **http://healthnet.umassmed.edu/**
- **Michigan:** Botsford General Hospital Library - Consumer Health (Botsford General Hospital, Library & Internet Services), **http://www.botsfordlibrary.org/consumer.htm**
- **Michigan:** Helen DeRoy Medical Library (Providence Hospital and Medical Centers), **http://www.providence-hospital.org/library/**
- **Michigan:** Marquette General Hospital - Consumer Health Library (Marquette General Hospital, Health Information Center), **http://www.mgh.org/center.html**
- **Michigan:** Patient Education Resouce Center - University of Michigan Cancer Center (University of Michigan Comprehensive Cancer Center, Ann Arbor), **http://www.cancer.med.umich.edu/learn/leares.htm**
- **Michigan:** Sladen Library & Center for Health Information Resources - Consumer Health Information (Detroit), **http://www.henryford.com/body.cfm?id=39330**
- **Montana:** Center for Health Information (St. Patrick Hospital and Health Sciences Center, Missoula)
- **National:** Consumer Health Library Directory (Medical Library Association, Consumer and Patient Health Information Section), **http://caphis.mlanet.org/directory/index.html**
- **National:** National Network of Libraries of Medicine (National Library of Medicine) - provides library services for health professionals in the United States who do not have access to a medical library, **http://nnlm.gov/**
- **National:** NN/LM List of Libraries Serving the Public (National Network of Libraries of Medicine), **http://nnlm.gov/members/**

- **Nevada:** Health Science Library, West Charleston Library (Las Vegas-Clark County Library District, Las Vegas), **http://www.lvccld.org/special_collections/medical/index.htm**
- **New Hampshire:** Dartmouth Biomedical Libraries (Dartmouth College Library, Hanover), **http://www.dartmouth.edu/~biomed/resources.htmld/conshealth.htmld/**
- **New Jersey:** Consumer Health Library (Rahway Hospital, Rahway), **http://www.rahwayhospital.com/library.htm**
- **New Jersey:** Dr. Walter Phillips Health Sciences Library (Englewood Hospital and Medical Center, Englewood), **http://www.englewoodhospital.com/links/index.htm**
- **New Jersey:** Meland Foundation (Englewood Hospital and Medical Center, Englewood), **http://www.geocities.com/ResearchTriangle/9360/**
- **New York:** Choices in Health Information (New York Public Library) - NLM Consumer Pilot Project participant, **http://www.nypl.org/branch/health/links.html**
- **New York:** Health Information Center (Upstate Medical University, State University of New York, Syracuse), **http://www.upstate.edu/library/hic/**
- **New York:** Health Sciences Library (Long Island Jewish Medical Center, New Hyde Park), **http://www.lij.edu/library/library.html**
- **New York:** ViaHealth Medical Library (Rochester General Hospital), **http://www.nyam.org/library/**
- **Ohio:** Consumer Health Library (Akron General Medical Center, Medical & Consumer Health Library), **http://www.akrongeneral.org/hwlibrary.htm**
- **Oklahoma:** The Health Information Center at Saint Francis Hospital (Saint Francis Health System, Tulsa), **http://www.sfh-tulsa.com/services/healthinfo.asp**
- **Oregon:** Planetree Health Resource Center (Mid-Columbia Medical Center, The Dalles), **http://www.mcmc.net/phrc/**
- **Pennsylvania:** Community Health Information Library (Milton S. Hershey Medical Center, Hershey), **http://www.hmc.psu.edu/commhealth/**
- **Pennsylvania:** Community Health Resource Library (Geisinger Medical Center, Danville), **http://www.geisinger.edu/education/commlib.shtml**
- **Pennsylvania:** HealthInfo Library (Moses Taylor Hospital, Scranton), **http://www.mth.org/healthwellness.html**
- **Pennsylvania:** Hopwood Library (University of Pittsburgh, Health Sciences Library System, Pittsburgh), **http://www.hsls.pitt.edu/guides/chi/hopwood/index_html**
- **Pennsylvania:** Koop Community Health Information Center (College of Physicians of Philadelphia), **http://www.collphyphil.org/kooppg1.shtml**
- **Pennsylvania:** Learning Resources Center - Medical Library (Susquehanna Health System, Williamsport), **http://www.shscares.org/services/lrc/index.asp**
- **Pennsylvania:** Medical Library (UPMC Health System, Pittsburgh), **http://www.upmc.edu/passavant/library.htm**
- **Quebec, Canada:** Medical Library (Montreal General Hospital), **http://www.mghlib.mcgill.ca/**

- **South Dakota:** Rapid City Regional Hospital Medical Library (Rapid City Regional Hospital), **http://www.rcrh.org/Services/Library/Default.asp**
- **Texas:** Houston HealthWays (Houston Academy of Medicine-Texas Medical Center Library), **http://hhw.library.tmc.edu/**
- **Washington:** Community Health Library (Kittitas Valley Community Hospital), **http://www.kvch.com/**
- **Washington:** Southwest Washington Medical Center Library (Southwest Washington Medical Center, Vancouver), **http://www.swmedicalcenter.com/body.cfm?id=72**

ONLINE GLOSSARIES

The Internet provides access to a number of free-to-use medical dictionaries. The National Library of Medicine has compiled the following list of online dictionaries:

- ADAM Medical Encyclopedia (A.D.A.M., Inc.), comprehensive medical reference: **http://www.nlm.nih.gov/medlineplus/encyclopedia.html**
- MedicineNet.com Medical Dictionary (MedicineNet, Inc.): **http://www.medterms.com/Script/Main/hp.asp**
- Merriam-Webster Medical Dictionary (Inteli-Health, Inc.): **http://www.intelihealth.com/IH/**
- Multilingual Glossary of Technical and Popular Medical Terms in Eight European Languages (European Commission) - Danish, Dutch, English, French, German, Italian, Portuguese, and Spanish: **http://allserv.rug.ac.be/~rvdstich/eugloss/welcome.html**
- On-line Medical Dictionary (CancerWEB): **http://cancerweb.ncl.ac.uk/omd/**
- Rare Diseases Terms (Office of Rare Diseases): **http://ord.aspensys.com/asp/diseases/diseases.asp**
- Technology Glossary (National Library of Medicine) - Health Care Technology: **http://www.nlm.nih.gov/nichsr/ta101/ta10108.htm**

Beyond these, MEDLINEplus contains a very patient-friendly encyclopedia covering every aspect of medicine (licensed from A.D.A.M., Inc.). The ADAM Medical Encyclopedia can be accessed at **http://www.nlm.nih.gov/medlineplus/encyclopedia.html**. ADAM is also available on commercial Web sites such as drkoop.com (**http://www.drkoop.com/**) and Web MD (**http://my.webmd.com/adam/asset/adam_disease_articles/a_to_z/a**).

Online Dictionary Directories

The following are additional online directories compiled by the National Library of Medicine, including a number of specialized medical dictionaries:

- Medical Dictionaries: Medical & Biological (World Health Organization): **http://www.who.int/hlt/virtuallibrary/English/diction.htm#Medical**
- MEL-Michigan Electronic Library List of Online Health and Medical Dictionaries (Michigan Electronic Library): **http://mel.lib.mi.us/health/health-dictionaries.html**
- Patient Education: Glossaries (DMOZ Open Directory Project): **http://dmoz.org/Health/Education/Patient_Education/Glossaries/**
- Web of Online Dictionaries (Bucknell University): **http://www.yourdictionary.com/diction5.html#medicine**

BURKITT'S LYMPHOMA DICTIONARY

The definitions below are derived from official public sources, including the National Institutes of Health [NIH] and the European Union [EU].

Abdominal: Having to do with the abdomen, which is the part of the body between the chest and the hips that contains the pancreas, stomach, intestines, liver, gallbladder, and other organs. [NIH]

Abdominal Pain: Sensation of discomfort, distress, or agony in the abdominal region. [NIH]

Aberrant: Wandering or deviating from the usual or normal course. [EU]

Acceptor: A substance which, while normally not oxidized by oxygen or reduced by hydrogen, can be oxidized or reduced in presence of a substance which is itself undergoing oxidation or reduction. [NIH]

Acidosis: A pathologic condition resulting from accumulation of acid or depletion of the alkaline reserve (bicarbonate content) in the blood and body tissues, and characterized by an increase in hydrogen ion concentration. [EU]

Acquired Immunodeficiency Syndrome: An acquired defect of cellular immunity associated with infection by the human immunodeficiency virus (HIV), a CD4-positive T-lymphocyte count under 200 cells/microliter or less than 14% of total lymphocytes, and increased susceptibility to opportunistic infections and malignant neoplasms. Clinical manifestations also include emaciation (wasting) and dementia. These elements reflect criteria for AIDS as defined by the CDC in 1993. [NIH]

Acute leukemia: A rapidly progressing cancer of the blood-forming tissue (bone marrow). [NIH]

Acute lymphoblastic leukemia: ALL. A quickly progressing disease in which too many immature white blood cells called lymphoblasts are found in the blood and bone marrow. Also called acute lymphocytic leukemia. [NIH]

Acute lymphocytic leukemia: ALL. A quickly progressing disease in which too many immature white blood cells called lymphoblasts are found in the blood and bone marrow. Also called acute lymphoblastic leukemia. [NIH]

Acute renal: A condition in which the kidneys suddenly stop working. In most cases, kidneys can recover from almost complete loss of function. [NIH]

Adaptability: Ability to develop some form of tolerance to conditions extremely different from those under which a living organism evolved. [NIH]

Adenovirus: A group of viruses that cause respiratory tract and eye infections. Adenoviruses used in gene therapy are altered to carry a specific tumor-fighting gene. [NIH]

Adoptive Transfer: Form of passive immunization where previously sensitized immunologic agents (cells or serum) are transferred to non-immune recipients. When transfer of cells is used as a therapy for the treatment of neoplasms, it is called adoptive immunotherapy (immunotherapy, adoptive). [NIH]

Adverse Effect: An unwanted side effect of treatment. [NIH]

Affinity: 1. Inherent likeness or relationship. 2. A special attraction for a specific element, organ, or structure. 3. Chemical affinity; the force that binds atoms in molecules; the tendency of substances to combine by chemical reaction. 4. The strength of noncovalent chemical binding between two substances as measured by the dissociation constant of the

complex. 5. In immunology, a thermodynamic expression of the strength of interaction between a single antigen-binding site and a single antigenic determinant (and thus of the stereochemical compatibility between them), most accurately applied to interactions among simple, uniform antigenic determinants such as haptens. Expressed as the association constant (K litres mole -1), which, owing to the heterogeneity of affinities in a population of antibody molecules of a given specificity, actually represents an average value (mean intrinsic association constant). 6. The reciprocal of the dissociation constant. [EU]

Affinity Chromatography: In affinity chromatography, a ligand attached to a column binds specifically to the molecule to be purified. [NIH]

Agar: A complex sulfated polymer of galactose units, extracted from Gelidium cartilagineum, Gracilaria confervoides, and related red algae. It is used as a gel in the preparation of solid culture media for microorganisms, as a bulk laxative, in making emulsions, and as a supporting medium for immunodiffusion and immunoelectrophoresis. [NIH]

Age of Onset: The age or period of life at which a disease or the initial symptoms or manifestations of a disease appear in an individual. [NIH]

Airway: A device for securing unobstructed passage of air into and out of the lungs during general anesthesia. [NIH]

Airway Obstruction: Any hindrance to the passage of air into and out of the lungs. [NIH]

Alanine: A non-essential amino acid that occurs in high levels in its free state in plasma. It is produced from pyruvate by transamination. It is involved in sugar and acid metabolism, increases immunity, and provides energy for muscle tissue, brain, and the central nervous system. [NIH]

Algorithms: A procedure consisting of a sequence of algebraic formulas and/or logical steps to calculate or determine a given task. [NIH]

Alkaline: Having the reactions of an alkali. [EU]

Alleles: Mutually exclusive forms of the same gene, occupying the same locus on homologous chromosomes, and governing the same biochemical and developmental process. [NIH]

Allergen: An antigenic substance capable of producing immediate-type hypersensitivity (allergy). [EU]

Allium: A genus of liliaceous herbs containing onions (Allium cepa), garlic (Allium sativum), and others; many produce pungent, often bacteriostatic and physiologically active compounds and are used as food, condiment, and medicament, the latter in traditional medicine. [NIH]

Allogeneic: Taken from different individuals of the same species. [NIH]

Allograft: An organ or tissue transplant between two humans. [NIH]

Alopecia: Absence of hair from areas where it is normally present. [NIH]

Alpha Particles: Positively charged particles composed of two protons and two neutrons, i.e., helium nuclei, emitted during disintegration of very heavy isotopes; a beam of alpha particles or an alpha ray has very strong ionizing power, but weak penetrability. [NIH]

Alternative medicine: Practices not generally recognized by the medical community as standard or conventional medical approaches and used instead of standard treatments. Alternative medicine includes the taking of dietary supplements, megadose vitamins, and herbal preparations; the drinking of special teas; and practices such as massage therapy, magnet therapy, spiritual healing, and meditation. [NIH]

Alternative Splicing: A process whereby multiple protein isoforms are generated from a single gene. Alternative splicing involves the splicing together of nonconsecutive exons during the processing of some, but not all, transcripts of the gene. Thus a particular exon may be connected to any one of several alternative exons to form messenger RNA. The alternative forms produce proteins in which one part is common while the other part is different. [NIH]

Alveoli: Tiny air sacs at the end of the bronchioles in the lungs. [NIH]

Amino Acid Sequence: The order of amino acids as they occur in a polypeptide chain. This is referred to as the primary structure of proteins. It is of fundamental importance in determining protein conformation. [NIH]

Amino Acids: Organic compounds that generally contain an amino (-NH2) and a carboxyl (-COOH) group. Twenty alpha-amino acids are the subunits which are polymerized to form proteins. [NIH]

Amino Acids: Organic compounds that generally contain an amino (-NH2) and a carboxyl (-COOH) group. Twenty alpha-amino acids are the subunits which are polymerized to form proteins. [NIH]

Amino-terminal: The end of a protein or polypeptide chain that contains a free amino group (-NH2). [NIH]

Amplification: The production of additional copies of a chromosomal DNA sequence, found as either intrachromosomal or extrachromosomal DNA. [NIH]

Anaesthesia: Loss of feeling or sensation. Although the term is used for loss of tactile sensibility, or of any of the other senses, it is applied especially to loss of the sensation of pain, as it is induced to permit performance of surgery or other painful procedures. [EU]

Analog: In chemistry, a substance that is similar, but not identical, to another. [NIH]

Analogous: Resembling or similar in some respects, as in function or appearance, but not in origin or development;. [EU]

Anaphylatoxins: The family of peptides C3a, C4a, C5a, and C5a des-arginine produced in the serum during complement activation. They produce smooth muscle contraction, mast cell histamine release, affect platelet aggregation, and act as mediators of the local inflammatory process. The order of anaphylatoxin activity from strongest to weakest is C5a, C3a, C4a, and C5a des-arginine. The latter is the so-called "classical" anaphylatoxin but shows no spasmogenic activity though it contains some chemotactic ability. [NIH]

Anaplasia: Loss of structural differentiation and useful function of neoplastic cells. [NIH]

Anatomical: Pertaining to anatomy, or to the structure of the organism. [EU]

Anemia: A reduction in the number of circulating erythrocytes or in the quantity of hemoglobin. [NIH]

Anergy: Absence of immune response to particular substances. [NIH]

Anesthesia: A state characterized by loss of feeling or sensation. This depression of nerve function is usually the result of pharmacologic action and is induced to allow performance of surgery or other painful procedures. [NIH]

Animal model: An animal with a disease either the same as or like a disease in humans. Animal models are used to study the development and progression of diseases and to test new treatments before they are given to humans. Animals with transplanted human cancers or other tissues are called xenograft models. [NIH]

Annealing: The spontaneous alignment of two single DNA strands to form a double helix. [NIH]

Antibacterial: A substance that destroys bacteria or suppresses their growth or reproduction. [EU]

Antibiotic: A drug used to treat infections caused by bacteria and other microorganisms. [NIH]

Antibodies: Immunoglobulin molecules having a specific amino acid sequence by virtue of which they interact only with the antigen that induced their synthesis in cells of the lymphoid series (especially plasma cells), or with an antigen closely related to it. [NIH]

Antibody: A type of protein made by certain white blood cells in response to a foreign substance (antigen). Each antibody can bind to only a specific antigen. The purpose of this binding is to help destroy the antigen. Antibodies can work in several ways, depending on the nature of the antigen. Some antibodies destroy antigens directly. Others make it easier for white blood cells to destroy the antigen. [NIH]

Anticoagulant: A drug that helps prevent blood clots from forming. Also called a blood thinner. [NIH]

Antigen: Any substance which is capable, under appropriate conditions, of inducing a specific immune response and of reacting with the products of that response, that is, with specific antibody or specifically sensitized T-lymphocytes, or both. Antigens may be soluble substances, such as toxins and foreign proteins, or particulate, such as bacteria and tissue cells; however, only the portion of the protein or polysaccharide molecule known as the antigenic determinant (q.v.) combines with antibody or a specific receptor on a lymphocyte. Abbreviated Ag. [EU]

Antigen-Antibody Complex: The complex formed by the binding of antigen and antibody molecules. The deposition of large antigen-antibody complexes leading to tissue damage causes immune complex diseases. [NIH]

Antigen-presenting cell: APC. A cell that shows antigen on its surface to other cells of the immune system. This is an important part of an immune response. [NIH]

Anti-inflammatory: Having to do with reducing inflammation. [NIH]

Antimetabolite: A chemical that is very similar to one required in a normal biochemical reaction in cells. Antimetabolites can stop or slow down the reaction. [NIH]

Antimicrobial: Killing microorganisms, or suppressing their multiplication or growth. [EU]

Antineoplastic: Inhibiting or preventing the development of neoplasms, checking the maturation and proliferation of malignant cells. [EU]

Antiproliferative: Counteracting a process of proliferation. [EU]

Antiviral: Destroying viruses or suppressing their replication. [EU]

Apoptosis: One of the two mechanisms by which cell death occurs (the other being the pathological process of necrosis). Apoptosis is the mechanism responsible for the physiological deletion of cells and appears to be intrinsically programmed. It is characterized by distinctive morphologic changes in the nucleus and cytoplasm, chromatin cleavage at regularly spaced sites, and the endonucleolytic cleavage of genomic DNA (DNA fragmentation) at internucleosomal sites. This mode of cell death serves as a balance to mitosis in regulating the size of animal tissues and in mediating pathologic processes associated with tumor growth. [NIH]

Aqueous: Having to do with water. [NIH]

Arginine: An essential amino acid that is physiologically active in the L-form. [NIH]

Arginine butyrate: A substance that is being studied as a treatment for cancer. [NIH]

Arterial: Pertaining to an artery or to the arteries. [EU]

Arteries: The vessels carrying blood away from the heart. [NIH]

Artery: Vessel-carrying blood from the heart to various parts of the body. [NIH]

Articular: Of or pertaining to a joint. [EU]

Aspiration: The act of inhaling. [NIH]

Assay: Determination of the amount of a particular constituent of a mixture, or of the biological or pharmacological potency of a drug. [EU]

Asymptomatic: Having no signs or symptoms of disease. [NIH]

Atmospheric Pressure: The pressure at any point in an atmosphere due solely to the weight of the atmospheric gases above the point concerned. [NIH]

Atrial: Pertaining to an atrium. [EU]

Atypical: Irregular; not conformable to the type; in microbiology, applied specifically to strains of unusual type. [EU]

Autologous: Taken from an individual's own tissues, cells, or DNA. [NIH]

Autologous bone marrow transplantation: A procedure in which bone marrow is removed from a person, stored, and then given back to the person after intensive treatment. [NIH]

Bacteria: Unicellular prokaryotic microorganisms which generally possess rigid cell walls, multiply by cell division, and exhibit three principal forms: round or coccal, rodlike or bacillary, and spiral or spirochetal. [NIH]

Bacteriostatic: 1. Inhibiting the growth or multiplication of bacteria. 2. An agent that inhibits the growth or multiplication of bacteria. [EU]

Bacterium: Microscopic organism which may have a spherical, rod-like, or spiral unicellular or non-cellular body. Bacteria usually reproduce through asexual processes. [NIH]

Base: In chemistry, the nonacid part of a salt; a substance that combines with acids to form salts; a substance that dissociates to give hydroxide ions in aqueous solutions; a substance whose molecule or ion can combine with a proton (hydrogen ion); a substance capable of donating a pair of electrons (to an acid) for the formation of a coordinate covalent bond. [EU]

Basement Membrane: Ubiquitous supportive tissue adjacent to epithelium and around smooth and striated muscle cells. This tissue contains intrinsic macromolecular components such as collagen, laminin, and sulfated proteoglycans. As seen by light microscopy one of its subdivisions is the basal (basement) lamina. [NIH]

Benign: Not cancerous; does not invade nearby tissue or spread to other parts of the body. [NIH]

Bilateral: Affecting both the right and left side of body. [NIH]

Bile: An emulsifying agent produced in the liver and secreted into the duodenum. Its composition includes bile acids and salts, cholesterol, and electrolytes. It aids digestion of fats in the duodenum. [NIH]

Bile Pigments: Pigments that give a characteristic color to bile including: bilirubin, biliverdine, and bilicyanin. [NIH]

Binding Sites: The reactive parts of a macromolecule that directly participate in its specific combination with another molecule. [NIH]

Biochemical: Relating to biochemistry; characterized by, produced by, or involving chemical reactions in living organisms. [EU]

Biological response modifier: BRM. A substance that stimulates the body's response to infection and disease. [NIH]

Biopsy: Removal and pathologic examination of specimens in the form of small pieces of tissue from the living body. [NIH]

Biopsy specimen: Tissue removed from the body and examined under a microscope to determine whether disease is present. [NIH]

Biosynthesis: The building up of a chemical compound in the physiologic processes of a living organism. [EU]

Biotechnology: Body of knowledge related to the use of organisms, cells or cell-derived constituents for the purpose of developing products which are technically, scientifically and clinically useful. Alteration of biologic function at the molecular level (i.e., genetic engineering) is a central focus; laboratory methods used include transfection and cloning technologies, sequence and structure analysis algorithms, computer databases, and gene and protein structure function analysis and prediction. [NIH]

Bladder: The organ that stores urine. [NIH]

Bleomycin: A complex of related glycopeptide antibiotics from Streptomyces verticillus consisting of bleomycin A2 and B2. It inhibits DNA metabolism and is used as an antineoplastic, especially for solid tumors. [NIH]

Blood pressure: The pressure of blood against the walls of a blood vessel or heart chamber. Unless there is reference to another location, such as the pulmonary artery or one of the heart chambers, it refers to the pressure in the systemic arteries, as measured, for example, in the forearm. [NIH]

Blood vessel: A tube in the body through which blood circulates. Blood vessels include a network of arteries, arterioles, capillaries, venules, and veins. [NIH]

Body Fluids: Liquid components of living organisms. [NIH]

Body Regions: Anatomical areas of the body. [NIH]

Bone Marrow: The soft tissue filling the cavities of bones. Bone marrow exists in two types, yellow and red. Yellow marrow is found in the large cavities of large bones and consists mostly of fat cells and a few primitive blood cells. Red marrow is a hematopoietic tissue and is the site of production of erythrocytes and granular leukocytes. Bone marrow is made up of a framework of connective tissue containing branching fibers with the frame being filled with marrow cells. [NIH]

Bone Marrow Purging: Techniques for the removal of subpopulations of cells (usually residual tumor cells) from the bone marrow ex vivo before it is infused. The purging is achieved by a variety of agents including pharmacologic agents, biophysical agents (laser photoirradiation or radioisotopes) and immunologic agents. Bone marrow purging is used in both autologous and allogeneic bone marrow transplantation. [NIH]

Bone Marrow Transplantation: The transference of bone marrow from one human or animal to another. [NIH]

Boron: A trace element with the atomic symbol B, atomic number 5, and atomic weight 10.81. Boron-10, an isotope of boron, is used as a neutron absorber in boron neutron capture therapy. [NIH]

Bowel: The long tube-shaped organ in the abdomen that completes the process of digestion. There is both a small and a large bowel. Also called the intestine. [NIH]

Brachytherapy: A collective term for interstitial, intracavity, and surface radiotherapy. It uses small sealed or partly-sealed sources that may be placed on or near the body surface or within a natural body cavity or implanted directly into the tissues. [NIH]

Brain Diseases: Pathologic conditions affecting the brain, which is composed of the

intracranial components of the central nervous system. This includes (but is not limited to) the cerebral cortex; intracranial white matter; basal ganglia; thalamus; hypothalamus; brain stem; and cerebellum. [NIH]

Calcium: A basic element found in nearly all organized tissues. It is a member of the alkaline earth family of metals with the atomic symbol Ca, atomic number 20, and atomic weight 40. Calcium is the most abundant mineral in the body and combines with phosphorus to form calcium phosphate in the bones and teeth. It is essential for the normal functioning of nerves and muscles and plays a role in blood coagulation (as factor IV) and in many enzymatic processes. [NIH]

Carbohydrate: An aldehyde or ketone derivative of a polyhydric alcohol, particularly of the pentahydric and hexahydric alcohols. They are so named because the hydrogen and oxygen are usually in the proportion to form water, (CH2O)n. The most important carbohydrates are the starches, sugars, celluloses, and gums. They are classified into mono-, di-, tri-, poly- and heterosaccharides. [EU]

Carboxy: Cannabinoid. [NIH]

Carcinogenic: Producing carcinoma. [EU]

Carcinogens: Substances that increase the risk of neoplasms in humans or animals. Both genotoxic chemicals, which affect DNA directly, and nongenotoxic chemicals, which induce neoplasms by other mechanism, are included. [NIH]

Carcinoma: Cancer that begins in the skin or in tissues that line or cover internal organs. [NIH]

Cardiac: Having to do with the heart. [NIH]

Cardiomyopathy: A general diagnostic term designating primary myocardial disease, often of obscure or unknown etiology. [EU]

Carrier State: The condition of harboring an infective organism without manifesting symptoms of infection. The organism must be readily transmissable to another susceptible host. [NIH]

Case report: A detailed report of the diagnosis, treatment, and follow-up of an individual patient. Case reports also contain some demographic information about the patient (for example, age, gender, ethnic origin). [NIH]

Case series: A group or series of case reports involving patients who were given similar treatment. Reports of case series usually contain detailed information about the individual patients. This includes demographic information (for example, age, gender, ethnic origin) and information on diagnosis, treatment, response to treatment, and follow-up after treatment. [NIH]

Caspase: Enzyme released by the cell at a crucial stage in apoptosis in order to shred all cellular proteins. [NIH]

Catabolism: Any destructive metabolic process by which organisms convert substances into excreted compounds. [EU]

Cavernous Sinus: An irregularly shaped venous space in the dura mater at either side of the sphenoid bone. [NIH]

CDC2: It is crucial for entry into mitosis of eukaryotic cells. [NIH]

Cell: The individual unit that makes up all of the tissues of the body. All living things are made up of one or more cells. [NIH]

Cell Adhesion: Adherence of cells to surfaces or to other cells. [NIH]

Cell Cycle: The complex series of phenomena, occurring between the end of one cell

division and the end of the next, by which cellular material is divided between daughter cells. [NIH]

Cell Death: The termination of the cell's ability to carry out vital functions such as metabolism, growth, reproduction, responsiveness, and adaptability. [NIH]

Cell Differentiation: Progressive restriction of the developmental potential and increasing specialization of function which takes place during the development of the embryo and leads to the formation of specialized cells, tissues, and organs. [NIH]

Cell Division: The fission of a cell. [NIH]

Cell Lineage: The developmental history of cells as traced from the first division of the original cell or cells in the embryo. [NIH]

Cell membrane: Cell membrane = plasma membrane. The structure enveloping a cell, enclosing the cytoplasm, and forming a selective permeability barrier; it consists of lipids, proteins, and some carbohydrates, the lipids thought to form a bilayer in which integral proteins are embedded to varying degrees. [EU]

Cell proliferation: An increase in the number of cells as a result of cell growth and cell division. [NIH]

Cell Size: The physical dimensions of a cell. It refers mainly to changes in dimensions correlated with physiological or pathological changes in cells. [NIH]

Central Nervous System: The main information-processing organs of the nervous system, consisting of the brain, spinal cord, and meninges. [NIH]

Ceramide: A type of fat produced in the body. It may cause some types of cells to die, and is being studied in cancer treatment. [NIH]

Cerebral: Of or pertaining of the cerebrum or the brain. [EU]

Character: In current usage, approximately equivalent to personality. The sum of the relatively fixed personality traits and habitual modes of response of an individual. [NIH]

Chemokines: Class of pro-inflammatory cytokines that have the ability to attract and activate leukocytes. They can be divided into at least three structural branches: C (chemokines, C), CC (chemokines, CC), and CXC (chemokines, CXC), according to variations in a shared cysteine motif. [NIH]

Chemopreventive: Natural or synthetic compound used to intervene in the early precancerous stages of carcinogenesis. [NIH]

Chemotactic Factors: Chemical substances that attract or repel cells or organisms. The concept denotes especially those factors released as a result of tissue injury, invasion, or immunologic activity, that attract leukocytes, macrophages, or other cells to the site of infection or insult. [NIH]

Chemotherapy: Treatment with anticancer drugs. [NIH]

Chin: The anatomical frontal portion of the mandible, also known as the mentum, that contains the line of fusion of the two separate halves of the mandible (symphysis menti). This line of fusion divides inferiorly to enclose a triangular area called the mental protuberance. On each side, inferior to the second premolar tooth, is the mental foramen for the passage of blood vessels and a nerve. [NIH]

Cholera: An acute diarrheal disease endemic in India and Southeast Asia whose causative agent is vibrio cholerae. This condition can lead to severe dehydration in a matter of hours unless quickly treated. [NIH]

Cholesterol: The principal sterol of all higher animals, distributed in body tissues, especially the brain and spinal cord, and in animal fats and oils. [NIH]

Chromatin: The material of chromosomes. It is a complex of DNA, histones, and nonhistone proteins (chromosomal proteins, non-histone) found within the nucleus of a cell. [NIH]

Chromosomal: Pertaining to chromosomes. [EU]

Chromosome: Part of a cell that contains genetic information. Except for sperm and eggs, all human cells contain 46 chromosomes. [NIH]

Chronic: A disease or condition that persists or progresses over a long period of time. [NIH]

Chronic Disease: Disease or ailment of long duration. [NIH]

Chronic myelogenous leukemia: CML. A slowly progressing disease in which too many white blood cells are made in the bone marrow. Also called chronic myeloid leukemia or chronic granulocytic leukemia. [NIH]

CIS: Cancer Information Service. The CIS is the National Cancer Institute's link to the public, interpreting and explaining research findings in a clear and understandable manner, and providing personalized responses to specific questions about cancer. Access the CIS by calling 1-800-4-CANCER, or by using the Web site at http://cis.nci.nih.gov. [NIH]

C-kit receptor: A protein on the surface of some cells that binds to stem cell factor (a substance that causes certain types of cells to grow). Altered forms of this receptor may be associated with some types of cancer. [NIH]

Clinical study: A research study in which patients receive treatment in a clinic or other medical facility. Reports of clinical studies can contain results for single patients (case reports) or many patients (case series or clinical trials). [NIH]

Clinical trial: A research study that tests how well new medical treatments or other interventions work in people. Each study is designed to test new methods of screening, prevention, diagnosis, or treatment of a disease. [NIH]

Cloning: The production of a number of genetically identical individuals; in genetic engineering, a process for the efficient replication of a great number of identical DNA molecules. [NIH]

Coagulation: 1. The process of clot formation. 2. In colloid chemistry, the solidification of a sol into a gelatinous mass; an alteration of a disperse phase or of a dissolved solid which causes the separation of the system into a liquid phase and an insoluble mass called the clot or curd. Coagulation is usually irreversible. 3. In surgery, the disruption of tissue by physical means to form an amorphous residuum, as in electrocoagulation and photocoagulation. [EU]

Codon: A set of three nucleotides in a protein coding sequence that specifies individual amino acids or a termination signal (codon, terminator). Most codons are universal, but some organisms do not produce the transfer RNAs (RNA, transfer) complementary to all codons. These codons are referred to as unassigned codons (codons, nonsense). [NIH]

Coenzyme: An organic nonprotein molecule, frequently a phosphorylated derivative of a water-soluble vitamin, that binds with the protein molecule (apoenzyme) to form the active enzyme (holoenzyme). [EU]

Cofactor: A substance, microorganism or environmental factor that activates or enhances the action of another entity such as a disease-causing agent. [NIH]

Colchicine: A major alkaloid from Colchicum autumnale L. and found also in other Colchicum species. Its primary therapeutic use is in the treatment of gout, but it has been used also in the therapy of familial Mediterranean fever (periodic disease). [NIH]

Colic: Paroxysms of pain. This condition usually occurs in the abdominal region but may occur in other body regions as well. [NIH]

Colitis: Inflammation of the colon. [NIH]

Collagen: A polypeptide substance comprising about one third of the total protein in mammalian organisms. It is the main constituent of skin, connective tissue, and the organic substance of bones and teeth. Different forms of collagen are produced in the body but all consist of three alpha-polypeptide chains arranged in a triple helix. Collagen is differentiated from other fibrous proteins, such as elastin, by the content of proline, hydroxyproline, and hydroxylysine; by the absence of tryptophan; and particularly by the high content of polar groups which are responsible for its swelling properties. [NIH]

Colon: The long, coiled, tubelike organ that removes water from digested food. The remaining material, solid waste called stool, moves through the colon to the rectum and leaves the body through the anus. [NIH]

Combination chemotherapy: Treatment using more than one anticancer drug. [NIH]

Combination Therapy: Association of 3 drugs to treat AIDS (AZT + DDC or DDI + protease inhibitor). [NIH]

Combinatorial: A cut-and-paste process that churns out thousands of potentially valuable compounds at once. [NIH]

Complement: A term originally used to refer to the heat-labile factor in serum that causes immune cytolysis, the lysis of antibody-coated cells, and now referring to the entire functionally related system comprising at least 20 distinct serum proteins that is the effector not only of immune cytolysis but also of other biologic functions. Complement activation occurs by two different sequences, the classic and alternative pathways. The proteins of the classic pathway are termed 'components of complement' and are designated by the symbols C1 through C9. C1 is a calcium-dependent complex of three distinct proteins C1q, C1r and C1s. The proteins of the alternative pathway (collectively referred to as the properdin system) and complement regulatory proteins are known by semisystematic or trivial names. Fragments resulting from proteolytic cleavage of complement proteins are designated with lower-case letter suffixes, e.g., C3a. Inactivated fragments may be designated with the suffix 'i', e.g. C3bi. Activated components or complexes with biological activity are designated by a bar over the symbol e.g. C1 or C4b,2a. The classic pathway is activated by the binding of C1 to classic pathway activators, primarily antigen-antibody complexes containing IgM, IgG1, IgG3; C1q binds to a single IgM molecule or two adjacent IgG molecules. The alternative pathway can be activated by IgA immune complexes and also by nonimmunologic materials including bacterial endotoxins, microbial polysaccharides, and cell walls. Activation of the classic pathway triggers an enzymatic cascade involving C1, C4, C2 and C3; activation of the alternative pathway triggers a cascade involving C3 and factors B, D and P. Both result in the cleavage of C5 and the formation of the membrane attack complex. Complement activation also results in the formation of many biologically active complement fragments that act as anaphylatoxins, opsonins, or chemotactic factors. [EU]

Complementarity Determining Regions: Three regions (CDR1, CDR2 and CDR3) of amino acid sequence in theimmunoglobulin variable region that are highly divergent. Together the CDRs from the light and heavy immunoglobulin chains form a surface that is complementary to the antigen. These regions are also present in other members of the immunoglobulin superfamily, for example, T-cell receptors (receptors, antigen, T-cell). [NIH]

Complementary and alternative medicine: CAM. Forms of treatment that are used in addition to (complementary) or instead of (alternative) standard treatments. These practices are not considered standard medical approaches. CAM includes dietary supplements, megadose vitamins, herbal preparations, special teas, massage therapy, magnet therapy, spiritual healing, and meditation. [NIH]

Complementary medicine: Practices not generally recognized by the medical community as

standard or conventional medical approaches and used to enhance or complement the standard treatments. Complementary medicine includes the taking of dietary supplements, megadose vitamins, and herbal preparations; the drinking of special teas; and practices such as massage therapy, magnet therapy, spiritual healing, and meditation. [NIH]

Complete remission: The disappearance of all signs of cancer. Also called a complete response. [NIH]

Complete response: The disappearance of all signs of cancer in response to treatment. This does not always mean the cancer has been cured. [NIH]

Computational Biology: A field of biology concerned with the development of techniques for the collection and manipulation of biological data, and the use of such data to make biological discoveries or predictions. This field encompasses all computational methods and theories applicable to molecular biology and areas of computer-based techniques for solving biological problems including manipulation of models and datasets. [NIH]

Connective Tissue: Tissue that supports and binds other tissues. It consists of connective tissue cells embedded in a large amount of extracellular matrix. [NIH]

Connective Tissue: Tissue that supports and binds other tissues. It consists of connective tissue cells embedded in a large amount of extracellular matrix. [NIH]

Consciousness: Sense of awareness of self and of the environment. [NIH]

Constipation: Infrequent or difficult evacuation of feces. [NIH]

Contraceptive: An agent that diminishes the likelihood of or prevents conception. [EU]

Contraindications: Any factor or sign that it is unwise to pursue a certain kind of action or treatment, e. g. giving a general anesthetic to a person with pneumonia. [NIH]

Contralateral: Having to do with the opposite side of the body. [NIH]

Cooperative group: A group of physicians, hospitals, or both formed to treat a large number of persons in the same way so that new treatment can be evaluated quickly. Clinical trials of new cancer treatments often require many more people than a single physician or hospital can care for. [NIH]

Cornea: The transparent part of the eye that covers the iris and the pupil and allows light to enter the inside. [NIH]

Corticosteroids: Hormones that have antitumor activity in lymphomas and lymphoid leukemias; in addition, corticosteroids (steroids) may be used for hormone replacement and for the management of some of the complications of cancer and its treatment. [NIH]

Cranial: Pertaining to the cranium, or to the anterior (in animals) or superior (in humans) end of the body. [EU]

Crossing-over: The exchange of corresponding segments between chromatids of homologous chromosomes during meiosia, forming a chiasma. [NIH]

Culture Media: Any liquid or solid preparation made specifically for the growth, storage, or transport of microorganisms or other types of cells. The variety of media that exist allow for the culturing of specific microorganisms and cell types, such as differential media, selective media, test media, and defined media. Solid media consist of liquid media that have been solidified with an agent such as agar or gelatin. [NIH]

Curative: Tending to overcome disease and promote recovery. [EU]

Curcumin: A dye obtained from tumeric, the powdered root of Curcuma longa Linn. It is used in the preparation of curcuma paper and the detection of boron. Curcumin appears to possess a spectrum of pharmacological properties, due primarily to its inhibitory effects on metabolic enzymes. [NIH]

Cyclin: Molecule that regulates the cell cycle. [NIH]

Cycloheximide: Antibiotic substance isolated from streptomycin-producing strains of Streptomyces griseus. It acts by inhibiting elongation during protein synthesis. [NIH]

Cyclophosphamide: Precursor of an alkylating nitrogen mustard antineoplastic and immunosuppressive agent that must be activated in the liver to form the active aldophosphamide. It is used in the treatment of lymphomas, leukemias, etc. Its side effect, alopecia, has been made use of in defleecing sheep. Cyclophosphamide may also cause sterility, birth defects, mutations, and cancer. [NIH]

Cysteine: A thiol-containing non-essential amino acid that is oxidized to form cystine. [NIH]

Cytochrome: Any electron transfer hemoprotein having a mode of action in which the transfer of a single electron is effected by a reversible valence change of the central iron atom of the heme prosthetic group between the +2 and +3 oxidation states; classified as cytochromes a in which the heme contains a formyl side chain, cytochromes b, which contain protoheme or a closely similar heme that is not covalently bound to the protein, cytochromes c in which protoheme or other heme is covalently bound to the protein, and cytochromes d in which the iron-tetrapyrrole has fewer conjugated double bonds than the hemes have. Well-known cytochromes have been numbered consecutively within groups and are designated by subscripts (beginning with no subscript), e.g. cytochromes c, c1, C2, . New cytochromes are named according to the wavelength in nanometres of the absorption maximum of the a-band of the iron (II) form in pyridine, e.g., c-555. [EU]

Cytogenetics: A branch of genetics which deals with the cytological and molecular behavior of genes and chromosomes during cell division. [NIH]

Cytokine: Small but highly potent protein that modulates the activity of many cell types, including T and B cells. [NIH]

Cytokinesis: Division of the rest of cell. [NIH]

Cytomegalovirus: A genus of the family Herpesviridae, subfamily Betaherpesvirinae, infecting the salivary glands, liver, spleen, lungs, eyes, and other organs, in which they produce characteristically enlarged cells with intranuclear inclusions. Infection with Cytomegalovirus is also seen as an opportunistic infection in AIDS. [NIH]

Cytomegalovirus Infections: Infection with Cytomegalovirus, characterized by enlarged cells bearing intranuclear inclusions. Infection may be in almost any organ, but the salivary glands are the most common site in children, as are the lungs in adults. [NIH]

Cytoplasm: The protoplasm of a cell exclusive of that of the nucleus; it consists of a continuous aqueous solution (cytosol) and the organelles and inclusions suspended in it (phaneroplasm), and is the site of most of the chemical activities of the cell. [EU]

Cytotoxic: Cell-killing. [NIH]

Cytotoxicity: Quality of being capable of producing a specific toxic action upon cells of special organs. [NIH]

De novo: In cancer, the first occurrence of cancer in the body. [NIH]

Decarboxylation: The removal of a carboxyl group, usually in the form of carbon dioxide, from a chemical compound. [NIH]

Degenerative: Undergoing degeneration : tending to degenerate; having the character of or involving degeneration; causing or tending to cause degeneration. [EU]

Deletion: A genetic rearrangement through loss of segments of DNA (chromosomes), bringing sequences, which are normally separated, into close proximity. [NIH]

Dementia: An acquired organic mental disorder with loss of intellectual abilities of

sufficient severity to interfere with social or occupational functioning. The dysfunction is multifaceted and involves memory, behavior, personality, judgment, attention, spatial relations, language, abstract thought, and other executive functions. The intellectual decline is usually progressive, and initially spares the level of consciousness. [NIH]

Denaturation: Rupture of the hydrogen bonds by heating a DNA solution and then cooling it rapidly causes the two complementary strands to separate. [NIH]

Dendrites: Extensions of the nerve cell body. They are short and branched and receive stimuli from other neurons. [NIH]

Dendritic: 1. Branched like a tree. 2. Pertaining to or possessing dendrites. [EU]

Dendritic cell: A special type of antigen-presenting cell (APC) that activates T lymphocytes. [NIH]

Dental Caries: Localized destruction of the tooth surface initiated by decalcification of the enamel followed by enzymatic lysis of organic structures and leading to cavity formation. If left unchecked, the cavity may penetrate the enamel and dentin and reach the pulp. The three most prominent theories used to explain the etiology of the disase are that acids produced by bacteria lead to decalcification; that micro-organisms destroy the enamel protein; or that keratolytic micro-organisms produce chelates that lead to decalcification. [NIH]

Dentition: The teeth in the dental arch; ordinarily used to designate the natural teeth in position in their alveoli. [EU]

Depolarization: The process or act of neutralizing polarity. In neurophysiology, the reversal of the resting potential in excitable cell membranes when stimulated, i.e., the tendency of the cell membrane potential to become positive with respect to the potential outside the cell. [EU]

Desensitization: The prevention or reduction of immediate hypersensitivity reactions by administration of graded doses of allergen; called also hyposensitization and immunotherapy. [EU]

Diagnostic procedure: A method used to identify a disease. [NIH]

Diffusion: The tendency of a gas or solute to pass from a point of higher pressure or concentration to a point of lower pressure or concentration and to distribute itself throughout the available space; a major mechanism of biological transport. [NIH]

Digestion: The process of breakdown of food for metabolism and use by the body. [NIH]

Digestive tract: The organs through which food passes when food is eaten. These organs are the mouth, esophagus, stomach, small and large intestines, and rectum. [NIH]

Dimerization: The process by which two molecules of the same chemical composition form a condensation product or polymer. [NIH]

Direct: 1. Straight; in a straight line. 2. Performed immediately and without the intervention of subsidiary means. [EU]

Discrete: Made up of separate parts or characterized by lesions which do not become blended; not running together; separate. [NIH]

Disease Progression: The worsening of a disease over time. This concept is most often used for chronic and incurable diseases where the stage of the disease is an important determinant of therapy and prognosis. [NIH]

Dissociation: 1. The act of separating or state of being separated. 2. The separation of a molecule into two or more fragments (atoms, molecules, ions, or free radicals) produced by the absorption of light or thermal energy or by solvation. 3. In psychology, a defense mechanism in which a group of mental processes are segregated from the rest of a person's mental activity in order to avoid emotional distress, as in the dissociative disorders (q.v.), or

in which an idea or object is segregated from its emotional significance; in the first sense it is roughly equivalent to splitting, in the second, to isolation. 4. A defect of mental integration in which one or more groups of mental processes become separated off from normal consciousness and, thus separated, function as a unitary whole. [EU]

Distal: Remote; farther from any point of reference; opposed to proximal. In dentistry, used to designate a position on the dental arch farther from the median line of the jaw. [EU]

Doxycycline: A synthetic tetracycline derivative with a range of antimicrobial activity and mode of action similar to that of tetracycline, but more effective against many species. Animal studies suggest that it may cause less tooth staining than other tetracyclines. [NIH]

Drive: A state of internal activity of an organism that is a necessary condition before a given stimulus will elicit a class of responses; e.g., a certain level of hunger (drive) must be present before food will elicit an eating response. [NIH]

Drug Interactions: The action of a drug that may affect the activity, metabolism, or toxicity of another drug. [NIH]

Duodenum: The first part of the small intestine. [NIH]

Dura mater: The outermost, toughest, and most fibrous of the three membranes (meninges) covering the brain and spinal cord; called also pachymeninx. [EU]

Ectopic: Pertaining to or characterized by ectopia. [EU]

Effector: It is often an enzyme that converts an inactive precursor molecule into an active second messenger. [NIH]

Efficacy: The extent to which a specific intervention, procedure, regimen, or service produces a beneficial result under ideal conditions. Ideally, the determination of efficacy is based on the results of a randomized control trial. [NIH]

Elective: Subject to the choice or decision of the patient or physician; applied to procedures that are advantageous to the patient but not urgent. [EU]

Electrocoagulation: Electrosurgical procedures used to treat hemorrhage (e.g., bleeding ulcers) and to ablate tumors, mucosal lesions, and refractory arrhythmias. [NIH]

Electrolyte: A substance that dissociates into ions when fused or in solution, and thus becomes capable of conducting electricity; an ionic solute. [EU]

Electrons: Stable elementary particles having the smallest known negative charge, present in all elements; also called negatrons. Positively charged electrons are called positrons. The numbers, energies and arrangement of electrons around atomic nuclei determine the chemical identities of elements. Beams of electrons are called cathode rays or beta rays, the latter being a high-energy biproduct of nuclear decay. [NIH]

Electroporation: A technique in which electric pulses of intensity in kilovolts per centimeter and of microsecond-to-millisecond duration cause a temporary loss of the semipermeability of cell membranes, thus leading to ion leakage, escape of metabolites, and increased uptake by cells of drugs, molecular probes, and DNA. Some applications of electroporation include introduction of plasmids or foreign DNA into living cells for transfection, fusion of cells to prepare hybridomas, and insertion of proteins into cell membranes. [NIH]

Emaciation: Clinical manifestation of excessive leanness usually caused by disease or a lack of nutrition. [NIH]

Embryo: The prenatal stage of mammalian development characterized by rapid morphological changes and the differentiation of basic structures. [NIH]

Embryogenesis: The process of embryo or embryoid formation, whether by sexual (zygotic) or asexual means. In asexual embryogenesis embryoids arise directly from the explant or on

intermediary callus tissue. In some cases they arise from individual cells (somatic cell embryoge). [NIH]

Emulsions: Colloids of two immiscible liquids where either phase may be either fatty or aqueous; lipid-in-water emulsions are usually liquid, like milk or lotion and water-in-lipid emulsions tend to be creams. [NIH]

Enamel: A very hard whitish substance which covers the dentine of the anatomical crown of a tooth. [NIH]

Encephalitis: Inflammation of the brain due to infection, autoimmune processes, toxins, and other conditions. Viral infections (see encephalitis, viral) are a relatively frequent cause of this condition. [NIH]

Encephalitis, Viral: Inflammation of brain parenchymal tissue as a result of viral infection. Encephalitis may occur as primary or secondary manifestation of Togaviridae infections; Herpesviridae infections; Adenoviridae infections; Flaviviridae infections; Bunyaviridae infections; Picornaviridae infections; Paramyxoviridae infections; Orthomyxoviridae infections; Retroviridae infections; and Arenaviridae infections. [NIH]

Endemic: Present or usually prevalent in a population or geographical area at all times; said of a disease or agent. Called also endemial. [EU]

Endogenous: Produced inside an organism or cell. The opposite is external (exogenous) production. [NIH]

Endotoxin: Toxin from cell walls of bacteria. [NIH]

Enhancer: Transcriptional element in the virus genome. [NIH]

Enteropeptidase: A specialized proteolytic enzyme secreted by intestinal cells. It converts trypsinogen into its active form trypsin by removing the N-terminal peptide. EC 3.4.21.9. [NIH]

Environmental Exposure: The exposure to potentially harmful chemical, physical, or biological agents in the environment or to environmental factors that may include ionizing radiation, pathogenic organisms, or toxic chemicals. [NIH]

Environmental Health: The science of controlling or modifying those conditions, influences, or forces surrounding man which relate to promoting, establishing, and maintaining health. [NIH]

Enzymatic: Phase where enzyme cuts the precursor protein. [NIH]

Enzyme: A protein that speeds up chemical reactions in the body. [NIH]

Epidemic: Occurring suddenly in numbers clearly in excess of normal expectancy; said especially of infectious diseases but applied also to any disease, injury, or other health-related event occurring in such outbreaks. [EU]

Epidemiological: Relating to, or involving epidemiology. [EU]

Epidural: The space between the wall of the spinal canal and the covering of the spinal cord. An epidural injection is given into this space. [NIH]

Epithelial: Refers to the cells that line the internal and external surfaces of the body. [NIH]

Epithelial Cells: Cells that line the inner and outer surfaces of the body. [NIH]

Epithelium: One or more layers of epithelial cells, supported by the basal lamina, which covers the inner or outer surfaces of the body. [NIH]

Erythrocytes: Red blood cells. Mature erythrocytes are non-nucleated, biconcave disks containing hemoglobin whose function is to transport oxygen. [NIH]

Esophagus: The muscular tube through which food passes from the throat to the stomach.

[NIH]

Etoposide: A semisynthetic derivative of podophyllotoxin that exhibits antitumor activity. Etoposide inhibits DNA synthesis by forming a complex with topoisomerase II and DNA. This complex induces breaks in double stranded DNA and prevents repair by topoisomerase II binding. Accumulated breaks in DNA prevent entry into the mitotic phase of cell division, and lead to cell death. Etoposide acts primarily in the G2 and S phases of the cell cycle. [NIH]

Eukaryotic Cells: Cells of the higher organisms, containing a true nucleus bounded by a nuclear membrane. [NIH]

Evacuation: An emptying, as of the bowels. [EU]

Excitation: An act of irritation or stimulation or of responding to a stimulus; the addition of energy, as the excitation of a molecule by absorption of photons. [EU]

Exhaustion: The feeling of weariness of mind and body. [NIH]

Exogenous: Developed or originating outside the organism, as exogenous disease. [EU]

Exon: The part of the DNA that encodes the information for the actual amino acid sequence of the protein. In many eucaryotic genes, the coding sequences consist of a series of exons alternating with intron sequences. [NIH]

Exophthalmos: Abnormal protrusion of both eyes; may be caused by endocrine gland malfunction, malignancy, injury, or paralysis of the extrinsic muscles of the eye. [NIH]

External-beam radiation: Radiation therapy that uses a machine to aim high-energy rays at the cancer. Also called external radiation. [NIH]

Extracellular: Outside a cell or cells. [EU]

Extracellular Matrix: A meshwork-like substance found within the extracellular space and in association with the basement membrane of the cell surface. It promotes cellular proliferation and provides a supporting structure to which cells or cell lysates in culture dishes adhere. [NIH]

Extracellular Space: Interstitial space between cells, occupied by fluid as well as amorphous and fibrous substances. [NIH]

Eye Infections: Infection, moderate to severe, caused by bacteria, fungi, or viruses, which occurs either on the external surface of the eye or intraocularly with probable inflammation, visual impairment, or blindness. [NIH]

Family Planning: Programs or services designed to assist the family in controlling reproduction by either improving or diminishing fertility. [NIH]

Fat: Total lipids including phospholipids. [NIH]

Fatigue: The state of weariness following a period of exertion, mental or physical, characterized by a decreased capacity for work and reduced efficiency to respond to stimuli. [NIH]

Feces: The excrement discharged from the intestines, consisting of bacteria, cells exfoliated from the intestines, secretions, chiefly of the liver, and a small amount of food residue. [EU]

Fenretinide: A synthetic retinoid that is used orally as a chemopreventive against prostate cancer and in women at risk of developing contralateral breast cancer. It is also effective as an antineoplastic agent. [NIH]

Fetal Hemoglobin: The major component of hemoglobin in the fetus. This hemoglobin has two alpha and two gamma polypeptide subunits in comparison to normal adult hemoglobin, which has two alpha and two beta polypeptide subunits. Fetal hemoglobin concentrations can be elevated (usually above 0.5%) in children and adults affected by

leukemia and several types of anemia. [NIH]

Fetus: The developing offspring from 7 to 8 weeks after conception until birth. [NIH]

Fibroblasts: Connective tissue cells which secrete an extracellular matrix rich in collagen and other macromolecules. [NIH]

Fistula: Abnormal communication most commonly seen between two internal organs, or between an internal organ and the surface of the body. [NIH]

Fixation: 1. The act or operation of holding, suturing, or fastening in a fixed position. 2. The condition of being held in a fixed position. 3. In psychiatry, a term with two related but distinct meanings : (1) arrest of development at a particular stage, which like regression (return to an earlier stage), if temporary is a normal reaction to setbacks and difficulties but if protracted or frequent is a cause of developmental failures and emotional problems, and (2) a close and suffocating attachment to another person, especially a childhood figure, such as one's mother or father. Both meanings are derived from psychoanalytic theory and refer to 'fixation' of libidinal energy either in a specific erogenous zone, hence fixation at the oral, anal, or phallic stage, or in a specific object, hence mother or father fixation. 4. The use of a fixative (q.v.) to preserve histological or cytological specimens. 5. In chemistry, the process whereby a substance is removed from the gaseous or solution phase and localized, as in carbon dioxide fixation or nitrogen fixation. 6. In ophthalmology, direction of the gaze so that the visual image of the object falls on the fovea centralis. 7. In film processing, the chemical removal of all undeveloped salts of the film emulsion, leaving only the developed silver to form a permanent image. [EU]

Flow Cytometry: Technique using an instrument system for making, processing, and displaying one or more measurements on individual cells obtained from a cell suspension. Cells are usually stained with one or more fluorescent dyes specific to cell components of interest, e.g., DNA, and fluorescence of each cell is measured as it rapidly transverses the excitation beam (laser or mercury arc lamp). Fluorescence provides a quantitative measure of various biochemical and biophysical properties of the cell, as well as a basis for cell sorting. Other measurable optical parameters include light absorption and light scattering, the latter being applicable to the measurement of cell size, shape, density, granularity, and stain uptake. [NIH]

Fluorescence: The property of emitting radiation while being irradiated. The radiation emitted is usually of longer wavelength than that incident or absorbed, e.g., a substance can be irradiated with invisible radiation and emit visible light. X-ray fluorescence is used in diagnosis. [NIH]

Fluorescent Dyes: Dyes that emit light when exposed to light. The wave length of the emitted light is usually longer than that of the incident light. Fluorochromes are substances that cause fluorescence in other substances, i.e., dyes used to mark or label other compounds with fluorescent tags. They are used as markers in biochemistry and immunology. [NIH]

Fold: A plication or doubling of various parts of the body. [NIH]

Follicles: Shafts through which hair grows. [NIH]

Fractionation: Dividing the total dose of radiation therapy into several smaller, equal doses delivered over a period of several days. [NIH]

Gallbladder: The pear-shaped organ that sits below the liver. Bile is concentrated and stored in the gallbladder. [NIH]

Gamma Rays: Very powerful and penetrating, high-energy electromagnetic radiation of shorter wavelength than that of x-rays. They are emitted by a decaying nucleus, usually between 0.01 and 10 MeV. They are also called nuclear x-rays. [NIH]

Gamma-interferon: Interferon produced by T-lymphocytes in response to various mitogens and antigens. Gamma interferon appears to have potent antineoplastic, immunoregulatory and antiviral activity. [NIH]

Ganciclovir: Acyclovir analog that is a potent inhibitor of the Herpesvirus family including cytomegalovirus. Ganciclovir is used to treat complications from AIDS-associated cytomegalovirus infections. [NIH]

Ganglia: Clusters of multipolar neurons surrounded by a capsule of loosely organized connective tissue located outside the central nervous system. [NIH]

Ganglion: 1. A knot, or knotlike mass. 2. A general term for a group of nerve cell bodies located outside the central nervous system; occasionally applied to certain nuclear groups within the brain or spinal cord, e.g. basal ganglia. 3. A benign cystic tumour occurring on a aponeurosis or tendon, as in the wrist or dorsum of the foot; it consists of a thin fibrous capsule enclosing a clear mucinous fluid. [EU]

Gangrenous: A circumscribed, deep-seated, suppurative inflammation of the subcutaneous tissue of the eyelid discharging pus from several points. [NIH]

Gas: Air that comes from normal breakdown of food. The gases are passed out of the body through the rectum (flatus) or the mouth (burp). [NIH]

Gastric: Having to do with the stomach. [NIH]

Gastrointestinal: Refers to the stomach and intestines. [NIH]

Gastrointestinal stromal tumor: GIST. A type of tumor that usually begins in cells in the wall of the gastrointestinal tract. It can be benign or malignant. [NIH]

Gastrointestinal tract: The stomach and intestines. [NIH]

Gels: Colloids with a solid continuous phase and liquid as the dispersed phase; gels may be unstable when, due to temperature or other cause, the solid phase liquifies; the resulting colloid is called a sol. [NIH]

Gene: The functional and physical unit of heredity passed from parent to offspring. Genes are pieces of DNA, and most genes contain the information for making a specific protein. [NIH]

Gene Expression: The phenotypic manifestation of a gene or genes by the processes of gene action. [NIH]

Gene Rearrangement: The ordered rearrangement of gene regions by DNA recombination such as that which occurs normally during development. [NIH]

Gene Silencing: Interruption or suppression of the expression of a gene at transcriptional or translational levels. [NIH]

Gene Targeting: The integration of exogenous DNA into the genome of an organism at sites where its expression can be suitably controlled. This integration occurs as a result of homologous recombination. [NIH]

Gene Therapy: The introduction of new genes into cells for the purpose of treating disease by restoring or adding gene expression. Techniques include insertion of retroviral vectors, transfection, homologous recombination, and injection of new genes into the nuclei of single cell embryos. The entire gene therapy process may consist of multiple steps. The new genes may be introduced into proliferating cells in vivo (e.g., bone marrow) or in vitro (e.g., fibroblast cultures) and the modified cells transferred to the site where the gene expression is required. Gene therapy may be particularly useful for treating enzyme deficiency diseases, hemoglobinopathies, and leukemias and may also prove useful in restoring drug sensitivity, particularly for leukemia. [NIH]

Genetic Engineering: Directed modification of the gene complement of a living organism by such techniques as altering the DNA, substituting genetic material by means of a virus, transplanting whole nuclei, transplanting cell hybrids, etc. [NIH]

Genetic testing: Analyzing DNA to look for a genetic alteration that may indicate an increased risk for developing a specific disease or disorder. [NIH]

Genetics: The biological science that deals with the phenomena and mechanisms of heredity. [NIH]

Genital: Pertaining to the genitalia. [EU]

Genotype: The genetic constitution of the individual; the characterization of the genes. [NIH]

Germinal Center: The activated center of a lymphoid follicle in secondary lymphoid tissue where B-lymphocytes are stimulated by antigens and helper T cells (T-lymphocytes, helper-inducer) are stimulated to generate memory cells. [NIH]

Gland: An organ that produces and releases one or more substances for use in the body. Some glands produce fluids that affect tissues or organs. Others produce hormones or participate in blood production. [NIH]

Glomeruli: Plural of glomerulus. [NIH]

Glomerulus: A tiny set of looping blood vessels in the nephron where blood is filtered in the kidney. [NIH]

Glucocorticoid: A compound that belongs to the family of compounds called corticosteroids (steroids). Glucocorticoids affect metabolism and have anti-inflammatory and immunosuppressive effects. They may be naturally produced (hormones) or synthetic (drugs). [NIH]

Glucuronic Acid: Derivatives of uronic acid found throughout the plant and animal kingdoms. They detoxify drugs and toxins by conjugating with them to form glucuronides in the liver which are more water-soluble metabolites that can be easily eliminated from the body. [NIH]

Glycine: A non-essential amino acid. It is found primarily in gelatin and silk fibroin and used therapeutically as a nutrient. It is also a fast inhibitory neurotransmitter. [NIH]

Glycoprotein: A protein that has sugar molecules attached to it. [NIH]

Glycosylation: The chemical or biochemical addition of carbohydrate or glycosyl groups to other chemicals, especially peptides or proteins. Glycosyl transferases are used in this biochemical reaction. [NIH]

Governing Board: The group in which legal authority is vested for the control of health-related institutions and organizations. [NIH]

Gp120: 120-kD HIV envelope glycoprotein which is involved in the binding of the virus to its membrane receptor, the CD4 molecule, found on the surface of certain cells in the body. [NIH]

Grade: The grade of a tumor depends on how abnormal the cancer cells look under a microscope and how quickly the tumor is likely to grow and spread. Grading systems are different for each type of cancer. [NIH]

Graft: Healthy skin, bone, or other tissue taken from one part of the body and used to replace diseased or injured tissue removed from another part of the body. [NIH]

Graft Rejection: An immune response with both cellular and humoral components, directed against an allogeneic transplant, whose tissue antigens are not compatible with those of the recipient. [NIH]

Granulocytes: Leukocytes with abundant granules in the cytoplasm. They are divided into

three groups: neutrophils, eosinophils, and basophils. [NIH]

Groin: The external junctural region between the lower part of the abdomen and the thigh. [NIH]

Habitual: Of the nature of a habit; according to habit; established by or repeated by force of habit, customary. [EU]

Haptens: Small antigenic determinants capable of eliciting an immune response only when coupled to a carrier. Haptens bind to antibodies but by themselves cannot elicit an antibody response. [NIH]

Hematopoiesis: The development and formation of various types of blood cells. [NIH]

Hemoglobin: One of the fractions of glycosylated hemoglobin A1c. Glycosylated hemoglobin is formed when linkages of glucose and related monosaccharides bind to hemoglobin A and its concentration represents the average blood glucose level over the previous several weeks. HbA1c levels are used as a measure of long-term control of plasma glucose (normal, 4 to 6 percent). In controlled diabetes mellitus, the concentration of glycosylated hemoglobin A is within the normal range, but in uncontrolled cases the level may be 3 to 4 times the normal conentration. Generally, complications are substantially lower among patients with Hb levels of 7 percent or less than in patients with HbA1c levels of 9 percent or more. [NIH]

Hemoglobin H: An abnormal hemoglobin composed of four beta chains. It is caused by the reduced synthesis of the alpha chain. This abnormality results in alpha-thalassemia. [NIH]

Hemoglobinopathies: A group of inherited disorders characterized by structural alterations within the hemoglobin molecule. [NIH]

Hemolytic: A disease that affects the blood and blood vessels. It destroys red blood cells, cells that cause the blood to clot, and the lining of blood vessels. HUS is often caused by the Escherichia coli bacterium in contaminated food. People with HUS may develop acute renal failure. [NIH]

Heparin: Heparinic acid. A highly acidic mucopolysaccharide formed of equal parts of sulfated D-glucosamine and D-glucuronic acid with sulfaminic bridges. The molecular weight ranges from six to twenty thousand. Heparin occurs in and is obtained from liver, lung, mast cells, etc., of vertebrates. Its function is unknown, but it is used to prevent blood clotting in vivo and vitro, in the form of many different salts. [NIH]

Hepatic: Refers to the liver. [NIH]

Hepatitis: Inflammation of the liver and liver disease involving degenerative or necrotic alterations of hepatocytes. [NIH]

Hepatocytes: The main structural component of the liver. They are specialized epithelial cells that are organized into interconnected plates called lobules. [NIH]

Hereditary: Of, relating to, or denoting factors that can be transmitted genetically from one generation to another. [NIH]

Heredity: 1. The genetic transmission of a particular quality or trait from parent to offspring. 2. The genetic constitution of an individual. [EU]

Herpes: Any inflammatory skin disease caused by a herpesvirus and characterized by the formation of clusters of small vesicles. When used alone, the term may refer to herpes simplex or to herpes zoster. [EU]

Herpes virus: A member of the herpes family of viruses. [NIH]

Herpes Zoster: Acute vesicular inflammation. [NIH]

Heterogeneity: The property of one or more samples or populations which implies that they

are not identical in respect of some or all of their parameters, e. g. heterogeneity of variance. [NIH]

Heterozygotes: Having unlike alleles at one or more corresponding loci on homologous chromosomes. [NIH]

Histocompatibility: The degree of antigenic similarity between the tissues of different individuals, which determines the acceptance or rejection of allografts. [NIH]

Histones: Small chromosomal proteins (approx 12-20 kD) possessing an open, unfolded structure and attached to the DNA in cell nuclei by ionic linkages. Classification into the various types (designated histone I, histone II, etc.) is based on the relative amounts of arginine and lysine in each. [NIH]

Homologous: Corresponding in structure, position, origin, etc., as (a) the feathers of a bird and the scales of a fish, (b) antigen and its specific antibody, (c) allelic chromosomes. [EU]

Homotypic: Adhesion between neutrophils. [NIH]

Hormone: A substance in the body that regulates certain organs. Hormones such as gastrin help in breaking down food. Some hormones come from cells in the stomach and small intestine. [NIH]

Hybrid: Cross fertilization between two varieties or, more usually, two species of vines, see also crossing. [NIH]

Hybridization: The genetic process of crossbreeding to produce a hybrid. Hybrid nucleic acids can be formed by nucleic acid hybridization of DNA and RNA molecules. Protein hybridization allows for hybrid proteins to be formed from polypeptide chains. [NIH]

Hybridomas: Cells artificially created by fusion of activated lymphocytes with neoplastic cells. The resulting hybrid cells are cloned and produce pure or "monoclonal" antibodies or T-cell products, identical to those produced by the immunologically competent parent, and continually grow and divide as the neoplastic parent. [NIH]

Hydrogen: The first chemical element in the periodic table. It has the atomic symbol H, atomic number 1, and atomic weight 1. It exists, under normal conditions, as a colorless, odorless, tasteless, diatomic gas. Hydrogen ions are protons. Besides the common H1 isotope, hydrogen exists as the stable isotope deuterium and the unstable, radioactive isotope tritium. [NIH]

Hydrolysis: The process of cleaving a chemical compound by the addition of a molecule of water. [NIH]

Hydroxyurea: An antineoplastic agent that inhibits DNA synthesis through the inhibition of ribonucleoside diphosphate reductase. [NIH]

Hyperbaric: Characterized by greater than normal pressure or weight; applied to gases under greater than atmospheric pressure, as hyperbaric oxygen, or to a solution of greater specific gravity than another taken as a standard of reference. [EU]

Hyperbaric oxygen: Oxygen that is at an atmospheric pressure higher than the pressure at sea level. Breathing hyperbaric oxygen to enhance the effectiveness of radiation therapy is being studied. [NIH]

Hyperbilirubinemia: Pathologic process consisting of an abnormal increase in the amount of bilirubin in the circulating blood, which may result in jaundice. [NIH]

Hyperplasia: An increase in the number of cells in a tissue or organ, not due to tumor formation. It differs from hypertrophy, which is an increase in bulk without an increase in the number of cells. [NIH]

Hypersensitivity: Altered reactivity to an antigen, which can result in pathologic reactions

upon subsequent exposure to that particular antigen. [NIH]

Hypertrophic cardiomyopathy: Heart muscle disease that leads to thickening of the heart walls, interfering with the heart's ability to fill with and pump blood. [NIH]

Hypertrophy: General increase in bulk of a part or organ, not due to tumor formation, nor to an increase in the number of cells. [NIH]

Hyperuricemia: A buildup of uric acid (a byproduct of metabolism) in the blood; a side effect of some anticancer drugs. [NIH]

Hypothalamus: Ventral part of the diencephalon extending from the region of the optic chiasm to the caudal border of the mammillary bodies and forming the inferior and lateral walls of the third ventricle. [NIH]

Immune response: The activity of the immune system against foreign substances (antigens). [NIH]

Immune system: The organs, cells, and molecules responsible for the recognition and disposal of foreign ("non-self") material which enters the body. [NIH]

Immunity: Nonsusceptibility to the invasive or pathogenic effects of foreign microorganisms or to the toxic effect of antigenic substances. [NIH]

Immunization: Deliberate stimulation of the host's immune response. Active immunization involves administration of antigens or immunologic adjuvants. Passive immunization involves administration of immune sera or lymphocytes or their extracts (e.g., transfer factor, immune RNA) or transplantation of immunocompetent cell producing tissue (thymus or bone marrow). [NIH]

Immunocompromised: Having a weakened immune system caused by certain diseases or treatments. [NIH]

Immunodeficiency: The decreased ability of the body to fight infection and disease. [NIH]

Immunodiffusion: Technique involving the diffusion of antigen or antibody through a semisolid medium, usually agar or agarose gel, with the result being a precipitin reaction. [NIH]

Immunoelectrophoresis: A technique that combines protein electrophoresis and double immunodiffusion. In this procedure proteins are first separated by gel electrophoresis (usually agarose), then made visible by immunodiffusion of specific antibodies. A distinct elliptical precipitin arc results for each protein detectable by the antisera. [NIH]

Immunoglobulin Variable Region: That region of the immunoglobulin (antibody) molecule that varies in its amino acid sequence and composition, confers the antigenic specificity, and is thought to comprise the binding site for the antigen. It is located at the N-terminus of the Fab fragment of the immunoglobulin. It includes hypervariable regions (complementarity determining regions) and framework regions. [NIH]

Immunoglobulins: Glycoproteins present in the blood (antibodies) and in other tissue. They are classified by structure and activity into five classes (IgA, IgD, IgE, IgG, IgM). [NIH]

Immunologic: The ability of the antibody-forming system to recall a previous experience with an antigen and to respond to a second exposure with the prompt production of large amounts of antibody. [NIH]

Immunology: The study of the body's immune system. [NIH]

Immunosuppressant: An agent capable of suppressing immune responses. [EU]

Immunosuppression: Deliberate prevention or diminution of the host's immune response. It may be nonspecific as in the administration of immunosuppressive agents (drugs or radiation) or by lymphocyte depletion or may be specific as in desensitization or the

simultaneous administration of antigen and immunosuppressive drugs. [NIH]

Immunosuppressive: Describes the ability to lower immune system responses. [NIH]

Immunosuppressive Agents: Agents that suppress immune function by one of several mechanisms of action. Classical cytotoxic immunosuppressants act by inhibiting DNA synthesis. Others may act through activation of suppressor T-cell populations or by inhibiting the activation of helper cells. While immunosuppression has been brought about in the past primarily to prevent rejection of transplanted organs, new applications involving mediation of the effects of interleukins and other cytokines are emerging. [NIH]

Immunotherapy: Manipulation of the host's immune system in treatment of disease. It includes both active and passive immunization as well as immunosuppressive therapy to prevent graft rejection. [NIH]

Immunotoxins: Semisynthetic conjugates of various toxic molecules, including radioactive isotopes and bacterial or plant toxins, with specific immune substances such as immunoglobulins, monoclonal antibodies, and antigens. The antitumor or antiviral immune substance carries the toxin to the tumor or infected cell where the toxin exerts its poisonous effect. [NIH]

Impairment: In the context of health experience, an impairment is any loss or abnormality of psychological, physiological, or anatomical structure or function. [NIH]

Implant radiation: A procedure in which radioactive material sealed in needles, seeds, wires, or catheters is placed directly into or near the tumor. Also called [NIH]

In situ: In the natural or normal place; confined to the site of origin without invasion of neighbouring tissues. [EU]

In Situ Hybridization: A technique that localizes specific nucleic acid sequences within intact chromosomes, eukaryotic cells, or bacterial cells through the use of specific nucleic acid-labeled probes. [NIH]

In vitro: In the laboratory (outside the body). The opposite of in vivo (in the body). [NIH]

In vivo: In the body. The opposite of in vitro (outside the body or in the laboratory). [NIH]

Incision: A cut made in the body during surgery. [NIH]

Incisional: The removal of a sample of tissue for examination under a microscope. [NIH]

Incisional biopsy: A surgical procedure in which a portion of a lump or suspicious area is removed for diagnosis. The tissue is then examined under a microscope. [NIH]

Incubation: The development of an infectious disease from the entrance of the pathogen to the appearance of clinical symptoms. [EU]

Indolent: A type of cancer that grows slowly. [NIH]

Induction: The act or process of inducing or causing to occur, especially the production of a specific morphogenetic effect in the developing embryo through the influence of evocators or organizers, or the production of anaesthesia or unconsciousness by use of appropriate agents. [EU]

Infection: 1. Invasion and multiplication of microorganisms in body tissues, which may be clinically unapparent or result in local cellular injury due to competitive metabolism, toxins, intracellular replication, or antigen-antibody response. The infection may remain localized, subclinical, and temporary if the body's defensive mechanisms are effective. A local infection may persist and spread by extension to become an acute, subacute, or chronic clinical infection or disease state. A local infection may also become systemic when the microorganisms gain access to the lymphatic or vascular system. 2. An infectious disease. [EU]

Infectious Mononucleosis: A common, acute infection usually caused by the Epstein-Barr virus (Human herpesvirus 4). There is an increase in mononuclear white blood cells and other atypical lymphocytes, generalized lymphadenopathy, splenomegaly, and occasionally hepatomegaly with hepatitis. [NIH]

Infiltration: The diffusion or accumulation in a tissue or cells of substances not normal to it or in amounts of the normal. Also, the material so accumulated. [EU]

Inflammation: A pathological process characterized by injury or destruction of tissues caused by a variety of cytologic and chemical reactions. It is usually manifested by typical signs of pain, heat, redness, swelling, and loss of function. [NIH]

Inguinal: Pertaining to the inguen, or groin. [EU]

Initiation: Mutation induced by a chemical reactive substance causing cell changes; being a step in a carcinogenic process. [NIH]

Innervation: 1. The distribution or supply of nerves to a part. 2. The supply of nervous energy or of nerve stimulus sent to a part. [EU]

Insight: The capacity to understand one's own motives, to be aware of one's own psychodynamics, to appreciate the meaning of symbolic behavior. [NIH]

Insulin: A protein hormone secreted by beta cells of the pancreas. Insulin plays a major role in the regulation of glucose metabolism, generally promoting the cellular utilization of glucose. It is also an important regulator of protein and lipid metabolism. Insulin is used as a drug to control insulin-dependent diabetes mellitus. [NIH]

Interferon: A biological response modifier (a substance that can improve the body's natural response to disease). Interferons interfere with the division of cancer cells and can slow tumor growth. There are several types of interferons, including interferon-alpha, -beta, and -gamma. These substances are normally produced by the body. They are also made in the laboratory for use in treating cancer and other diseases. [NIH]

Interferon-alpha: One of the type I interferons produced by peripheral blood leukocytes or lymphoblastoid cells when exposed to live or inactivated virus, double-stranded RNA, or bacterial products. It is the major interferon produced by virus-induced leukocyte cultures and, in addition to its pronounced antiviral activity, it causes activation of NK cells. [NIH]

Interleukin-1: A soluble factor produced by monocytes, macrophages, and other cells which activates T-lymphocytes and potentiates their response to mitogens or antigens. IL-1 consists of two distinct forms, IL-1 alpha and IL-1 beta which perform the same functions but are distinct proteins. The biological effects of IL-1 include the ability to replace macrophage requirements for T-cell activation. The factor is distinct from interleukin-2. [NIH]

Interleukin-10: Factor that is a coregulator of mast cell growth. It is produced by T-cells and B-cells and shows extensive homology with the Epstein-Barr virus BCRFI gene. [NIH]

Interleukin-2: Chemical mediator produced by activated T lymphocytes and which regulates the proliferation of T cells, as well as playing a role in the regulation of NK cell activity. [NIH]

Interleukins: Soluble factors which stimulate growth-related activities of leukocytes as well as other cell types. They enhance cell proliferation and differentiation, DNA synthesis, secretion of other biologically active molecules and responses to immune and inflammatory stimuli. [NIH]

Internal radiation: A procedure in which radioactive material sealed in needles, seeds, wires, or catheters is placed directly into or near the tumor. Also called brachytherapy, implant radiation, or interstitial radiation therapy. [NIH]

Interstitial: Pertaining to or situated between parts or in the interspaces of a tissue. [EU]

Intestinal: Having to do with the intestines. [NIH]

Intestine: A long, tube-shaped organ in the abdomen that completes the process of digestion. There is both a large intestine and a small intestine. Also called the bowel. [NIH]

Intracellular: Inside a cell. [NIH]

Intracellular Membranes: Membranes of subcellular structures. [NIH]

Intravascular: Within a vessel or vessels. [EU]

Intravenous: IV. Into a vein. [NIH]

Intrinsic: Situated entirely within or pertaining exclusively to a part. [EU]

Invasive: 1. Having the quality of invasiveness. 2. Involving puncture or incision of the skin or insertion of an instrument or foreign material into the body; said of diagnostic techniques. [EU]

Ions: An atom or group of atoms that have a positive or negative electric charge due to a gain (negative charge) or loss (positive charge) of one or more electrons. Atoms with a positive charge are known as cations; those with a negative charge are anions. [NIH]

Irradiation: The use of high-energy radiation from x-rays, neutrons, and other sources to kill cancer cells and shrink tumors. Radiation may come from a machine outside the body (external-beam radiation therapy) or from materials called radioisotopes. Radioisotopes produce radiation and can be placed in or near the tumor or in the area near cancer cells. This type of radiation treatment is called internal radiation therapy, implant radiation, interstitial radiation, or brachytherapy. Systemic radiation therapy uses a radioactive substance, such as a radiolabeled monoclonal antibody, that circulates throughout the body. Irradiation is also called radiation therapy, radiotherapy, and x-ray therapy. [NIH]

Jaundice: A clinical manifestation of hyperbilirubinemia, consisting of deposition of bile pigments in the skin, resulting in a yellowish staining of the skin and mucous membranes. [NIH]

Kb: A measure of the length of DNA fragments, 1 Kb = 1000 base pairs. The largest DNA fragments are up to 50 kilobases long. [NIH]

Keratolytic: An agent that promotes keratolysis. [EU]

Kidney Transplantation: The transference of a kidney from one human or animal to another. [NIH]

Kinetic: Pertaining to or producing motion. [EU]

Labile: 1. Gliding; moving from point to point over the surface; unstable; fluctuating. 2. Chemically unstable. [EU]

Lactate Dehydrogenase: A tetrameric enzyme that, along with the coenzyme NAD+, catalyzes the interconversion of lactate and pyruvate. In vertebrates, genes for three different subunits (LDH-A, LDH-B and LDH-C) exist. [NIH]

Large Intestine: The part of the intestine that goes from the cecum to the rectum. The large intestine absorbs water from stool and changes it from a liquid to a solid form. The large intestine is 5 feet long and includes the appendix, cecum, colon, and rectum. Also called colon. [NIH]

Latency: The period of apparent inactivity between the time when a stimulus is presented and the moment a response occurs. [NIH]

Latent: Phoria which occurs at one distance or another and which usually has no troublesome effect. [NIH]

Laxative: An agent that acts to promote evacuation of the bowel; a cathartic or purgative. [EU]

Lectin: A complex molecule that has both protein and sugars. Lectins are able to bind to the outside of a cell and cause biochemical changes in it. Lectins are made by both animals and plants. [NIH]

Lesion: An area of abnormal tissue change. [NIH]

Leucocyte: All the white cells of the blood and their precursors (myeloid cell series, lymphoid cell series) but commonly used to indicate granulocytes exclusive of lymphocytes. [NIH]

Leukaemia: An acute or chronic disease of unknown cause in man and other warm-blooded animals that involves the blood-forming organs, is characterized by an abnormal increase in the number of leucocytes in the tissues of the body with or without a corresponding increase of those in the circulating blood, and is classified according of the type leucocyte most prominently involved. [EU]

Leukemia: Cancer of blood-forming tissue. [NIH]

Leukoplakia: A white patch that may develop on mucous membranes such as the cheek, gums, or tongue and may become cancerous. [NIH]

Ligands: A RNA simulation method developed by the MIT. [NIH]

Ligation: Application of a ligature to tie a vessel or strangulate a part. [NIH]

Lip: Either of the two fleshy, full-blooded margins of the mouth. [NIH]

Lipoprotein: Any of the lipid-protein complexes in which lipids are transported in the blood; lipoprotein particles consist of a spherical hydrophobic core of triglycerides or cholesterol esters surrounded by an amphipathic monolayer of phospholipids, cholesterol, and apolipoproteins; the four principal classes are high-density, low-density, and very-low-density lipoproteins and chylomicrons. [EU]

Liver: A large, glandular organ located in the upper abdomen. The liver cleanses the blood and aids in digestion by secreting bile. [NIH]

Localization: The process of determining or marking the location or site of a lesion or disease. May also refer to the process of keeping a lesion or disease in a specific location or site. [NIH]

Localized: Cancer which has not metastasized yet. [NIH]

Locus Control Region: A regulatory region first identified in the human beta-globin locus but subsequently found in other loci. The region is believed to regulate transcription by opening and remodeling chromatin structure. It may also have enhancer activity. [NIH]

Low-density lipoprotein: Lipoprotein that contains most of the cholesterol in the blood. LDL carries cholesterol to the tissues of the body, including the arteries. A high level of LDL increases the risk of heart disease. LDL typically contains 60 to 70 percent of the total serum cholesterol and both are directly correlated with CHD risk. [NIH]

Lymph: The almost colorless fluid that travels through the lymphatic system and carries cells that help fight infection and disease. [NIH]

Lymph node: A rounded mass of lymphatic tissue that is surrounded by a capsule of connective tissue. Also known as a lymph gland. Lymph nodes are spread out along lymphatic vessels and contain many lymphocytes, which filter the lymphatic fluid (lymph). [NIH]

Lymphadenopathy: Disease or swelling of the lymph nodes. [NIH]

Lymphatic: The tissues and organs, including the bone marrow, spleen, thymus, and lymph nodes, that produce and store cells that fight infection and disease. [NIH]

Lymphatic system: The tissues and organs that produce, store, and carry white blood cells

that fight infection and other diseases. This system includes the bone marrow, spleen, thymus, lymph nodes and a network of thin tubes that carry lymph and white blood cells. These tubes branch, like blood vessels, into all the tissues of the body. [NIH]

Lymphoblastic: One of the most aggressive types of non-Hodgkin lymphoma. [NIH]

Lymphoblasts: Interferon produced predominantly by leucocyte cells. [NIH]

Lymphocyte Count: A count of the number of lymphocytes in the blood. [NIH]

Lymphocyte Depletion: Immunosuppression by reduction of circulating lymphocytes or by T-cell depletion of bone marrow. The former may be accomplished in vivo by thoracic duct drainage or administration of antilymphocyte serum. The latter is performed ex vivo on bone marrow before its transplantation. [NIH]

Lymphocyte Subsets: A classification of lymphocytes based on structurally or functionally different populations of cells. [NIH]

Lymphocyte Transformation: Morphologic alteration of small lymphocytes in culture into large blast-like cells able to synthesize DNA and RNA and to divide mitotically. It is induced by interleukins, mitogens such as phytohemagglutinins, and by specific antigens. It may also occur in vivo, as in graft rejection and chronic myelogenous leukemia. [NIH]

Lymphocytes: White blood cells formed in the body's lymphoid tissue. The nucleus is round or ovoid with coarse, irregularly clumped chromatin while the cytoplasm is typically pale blue with azurophilic (if any) granules. Most lymphocytes can be classified as either T or B (with subpopulations of each); those with characteristics of neither major class are called null cells. [NIH]

Lymphoid: Referring to lymphocytes, a type of white blood cell. Also refers to tissue in which lymphocytes develop. [NIH]

Lymphoma: A general term for various neoplastic diseases of the lymphoid tissue. [NIH]

Lymphoma, Follicular: Malignant lymphoma in which the lymphomatous cells are clustered into identifiable nodules within the lymph nodes. The nodules resemble to some extent the germinal centers of lymph node follicles and most likely represent neoplastic proliferation of lymph node-derived follicular center B-lymphocytes. This class of lymphoma usually occurs in older persons, is commonly multinodal, and possibly extranodal. Patients whose lymphomas present a follicular or nodular pattern generally have a more indolent course than those presenting with a diffuse pattern. [NIH]

Lymphoproliferative: Disorders characterized by proliferation of lymphoid tissue, general or unspecified. [NIH]

Lymphoproliferative Disorders: Disorders characterized by proliferation of lymphoid tissue, general or unspecified. [NIH]

Lymphosarcoma: An obsolete term for a malignant tumor of lymphatic tissue. [NIH]

Lysine: An essential amino acid. It is often added to animal feed. [NIH]

Lytic: 1. Pertaining to lysis or to a lysin. 2. Producing lysis. [EU]

Macrophage: A type of white blood cell that surrounds and kills microorganisms, removes dead cells, and stimulates the action of other immune system cells. [NIH]

Macrophage Inflammatory Protein-1: A chemokine that is chemotactic for neutrophils and monocytes, stimulates macrophages, and may play a role in regulating hematopoiesis. Its two variants, MIP-1alpha and MIP-1beta, are 60% homologous to each other. [NIH]

Malaria: A protozoan disease caused in humans by four species of the genus Plasmodium (P. falciparum (malaria, falciparum), P. vivax (malaria, vivax), P. ovale, and P. malariae) and transmitted by the bite of an infected female mosquito of the genus Anopheles. Malaria is

endemic in parts of Asia, Africa, Central and South America, Oceania, and certain Caribbean islands. It is characterized by extreme exhaustion associated with paroxysms of high fever, sweating, shaking chills, and anemia. Malaria in animals is caused by other species of plasmodia. [NIH]

Malaria, Falciparum: Malaria caused by Plasmodium falciparum. This is the severest form of malaria and is associated with the highest levels of parasites in the blood. This disease is characterized by irregularly recurring febrile paroxysms that in extreme cases occur with acute cerebral, renal, or gastrointestinal manifestations. [NIH]

Malaria, Vivax: Malaria caused by Plasmodium vivax. This form of malaria is less severe than malaria, falciparum, but there is a higher probability for relapses to occur. Febrile paroxysms often occur every other day. [NIH]

Malignancy: A cancerous tumor that can invade and destroy nearby tissue and spread to other parts of the body. [NIH]

Malignant: Cancerous; a growth with a tendency to invade and destroy nearby tissue and spread to other parts of the body. [NIH]

Malignant tumor: A tumor capable of metastasizing. [NIH]

Mandible: The largest and strongest bone of the face constituting the lower jaw. It supports the lower teeth. [NIH]

Mandibular Condyle: The posterior process on the ramus of the mandible composed of two parts: a superior part, the articular portion, and an inferior part, the condylar neck. [NIH]

Mastitis: Inflammatory disease of the breast, or mammary gland. [NIH]

Maxillofacial Development: The process of growth and differentiation of the jaws and face. [NIH]

Measles Virus: The type species of morbillivirus and the cause of the highly infectious human disease measles, which affects mostly children. [NIH]

Medial: Lying near the midsaggital plane of the body; opposed to lateral. [NIH]

Mediate: Indirect; accomplished by the aid of an intervening medium. [EU]

Medicament: A medicinal substance or agent. [EU]

MEDLINE: An online database of MEDLARS, the computerized bibliographic Medical Literature Analysis and Retrieval System of the National Library of Medicine. [NIH]

Medroxyprogesterone Acetate: An injectable contraceptive, generally marketed under the name Depo-Provera. [NIH]

Meiosis: A special method of cell division, occurring in maturation of the germ cells, by means of which each daughter nucleus receives half the number of chromosomes characteristic of the somatic cells of the species. [NIH]

Melanocytes: Epidermal dendritic pigment cells which control long-term morphological color changes by alteration in their number or in the amount of pigment they produce and store in the pigment containing organelles called melanosomes. Melanophores are larger cells which do not exist in mammals. [NIH]

Melanoma: A form of skin cancer that arises in melanocytes, the cells that produce pigment. Melanoma usually begins in a mole. [NIH]

Melanosomes: Melanin-containing organelles found in melanocytes and melanophores. [NIH]

Membrane: A very thin layer of tissue that covers a surface. [NIH]

Membrane Proteins: Proteins which are found in membranes including cellular and

intracellular membranes. They consist of two types, peripheral and integral proteins. They include most membrane-associated enzymes, antigenic proteins, transport proteins, and drug, hormone, and lectin receptors. [NIH]

Memory: Complex mental function having four distinct phases: (1) memorizing or learning, (2) retention, (3) recall, and (4) recognition. Clinically, it is usually subdivided into immediate, recent, and remote memory. [NIH]

Meninges: The three membranes that cover and protect the brain and spinal cord. [NIH]

Meningitis: Inflammation of the meninges. When it affects the dura mater, the disease is termed pachymeningitis; when the arachnoid and pia mater are involved, it is called leptomeningitis, or meningitis proper. [EU]

Mental: Pertaining to the mind; psychic. 2. (L. mentum chin) pertaining to the chin. [EU]

Mental Retardation: Refers to sub-average general intellectual functioning which originated during the developmental period and is associated with impairment in adaptive behavior. [NIH]

Mercury: A silver metallic element that exists as a liquid at room temperature. It has the atomic symbol Hg (from hydrargyrum, liquid silver), atomic number 80, and atomic weight 200.59. Mercury is used in many industrial applications and its salts have been employed therapeutically as purgatives, antisyphilitics, disinfectants, and astringents. It can be absorbed through the skin and mucous membranes which leads to mercury poisoning. Because of its toxicity, the clinical use of mercury and mercurials is diminishing. [NIH]

Metabolite: Any substance produced by metabolism or by a metabolic process. [EU]

Metaphase: The second phase of cell division, in which the chromosomes line up across the equatorial plane of the spindle prior to separation. [NIH]

Metastasis: The spread of cancer from one part of the body to another. Tumors formed from cells that have spread are called "secondary tumors" and contain cells that are like those in the original (primary) tumor. The plural is metastases. [NIH]

Methotrexate: An antineoplastic antimetabolite with immunosuppressant properties. It is an inhibitor of dihydrofolate reductase and prevents the formation of tetrahydrofolate, necessary for synthesis of thymidylate, an essential component of DNA. [NIH]

Microbe: An organism which cannot be observed with the naked eye; e. g. unicellular animals, lower algae, lower fungi, bacteria. [NIH]

Microorganism: An organism that can be seen only through a microscope. Microorganisms include bacteria, protozoa, algae, and fungi. Although viruses are not considered living organisms, they are sometimes classified as microorganisms. [NIH]

Micro-organism: An organism which cannot be observed with the naked eye; e. g. unicellular animals, lower algae, lower fungi, bacteria. [NIH]

Migration: The systematic movement of genes between populations of the same species, geographic race, or variety. [NIH]

Mitochondrial Swelling: Increase in volume of mitochondria due to an influx of fluid; it occurs in hypotonic solutions due to osmotic pressure and in isotonic solutions as a result of altered permeability of the membranes of respiring mitochondria. [NIH]

Mitomycin: An antineoplastic antibiotic produced by Streptomyces caespitosus. It acts as a bi- or trifunctional alkylating agent causing cross-linking of DNA and inhibition of DNA synthesis. [NIH]

Mitosis: A method of indirect cell division by means of which the two daughter nuclei normally receive identical complements of the number of chromosomes of the somatic cells

of the species. [NIH]

Mitotic: Cell resulting from mitosis. [NIH]

Mobility: Capability of movement, of being moved, or of flowing freely. [EU]

Modeling: A treatment procedure whereby the therapist presents the target behavior which the learner is to imitate and make part of his repertoire. [NIH]

Modification: A change in an organism, or in a process in an organism, that is acquired from its own activity or environment. [NIH]

Molecular: Of, pertaining to, or composed of molecules : a very small mass of matter. [EU]

Molecular Probes: A group of atoms or molecules attached to other molecules or cellular structures and used in studying the properties of these molecules and structures. Radioactive DNA or RNA sequences are used in molecular genetics to detect the presence of a complementary sequence by molecular hybridization. [NIH]

Molecule: A chemical made up of two or more atoms. The atoms in a molecule can be the same (an oxygen molecule has two oxygen atoms) or different (a water molecule has two hydrogen atoms and one oxygen atom). Biological molecules, such as proteins and DNA, can be made up of many thousands of atoms. [NIH]

Monitor: An apparatus which automatically records such physiological signs as respiration, pulse, and blood pressure in an anesthetized patient or one undergoing surgical or other procedures. [NIH]

Monoclonal: An antibody produced by culturing a single type of cell. It therefore consists of a single species of immunoglobulin molecules. [NIH]

Monoclonal antibodies: Laboratory-produced substances that can locate and bind to cancer cells wherever they are in the body. Many monoclonal antibodies are used in cancer detection or therapy; each one recognizes a different protein on certain cancer cells. Monoclonal antibodies can be used alone, or they can be used to deliver drugs, toxins, or radioactive material directly to a tumor. [NIH]

Monocytes: Large, phagocytic mononuclear leukocytes produced in the vertebrate bone marrow and released into the blood; contain a large, oval or somewhat indented nucleus surrounded by voluminous cytoplasm and numerous organelles. [NIH]

Mononuclear: A cell with one nucleus. [NIH]

Monophosphate: So called second messenger for neurotransmitters and hormones. [NIH]

Morbillivirus: A genus of the family Paramyxoviridae (subfamily Paramyxovirinae) where all the virions have hemagglutinin but not neuraminidase activity. All members produce both cytoplasmic and intranuclear inclusion bodies. MEASLES VIRUS is the type species. [NIH]

Morphological: Relating to the configuration or the structure of live organs. [NIH]

Mucosa: A mucous membrane, or tunica mucosa. [EU]

Mucositis: A complication of some cancer therapies in which the lining of the digestive system becomes inflamed. Often seen as sores in the mouth. [NIH]

Muscular Diseases: Acquired, familial, and congenital disorders of skeletal muscle and smooth muscle. [NIH]

Myristate: Pharmacological activator of protein kinase C. [NIH]

Nasopharynx: The nasal part of the pharynx, lying above the level of the soft palate. [NIH]

NCI: National Cancer Institute. NCI, part of the National Institutes of Health of the United States Department of Health and Human Services, is the federal government's principal

agency for cancer research. NCI conducts, coordinates, and funds cancer research, training, health information dissemination, and other programs with respect to the cause, diagnosis, prevention, and treatment of cancer. Access the NCI Web site at http://cancer.gov. [NIH]

Necrosis: A pathological process caused by the progressive degradative action of enzymes that is generally associated with severe cellular trauma. It is characterized by mitochondrial swelling, nuclear flocculation, uncontrolled cell lysis, and ultimately cell death. [NIH]

Neoplasia: Abnormal and uncontrolled cell growth. [NIH]

Neoplasm: A new growth of benign or malignant tissue. [NIH]

Nervous System: The entire nerve apparatus composed of the brain, spinal cord, nerves and ganglia. [NIH]

Neuralgia: Intense or aching pain that occurs along the course or distribution of a peripheral or cranial nerve. [NIH]

Neuroblastoma: Cancer that arises in immature nerve cells and affects mostly infants and children. [NIH]

Neuromuscular: Pertaining to muscles and nerves. [EU]

Neuromuscular Junction: The synapse between a neuron and a muscle. [NIH]

Neurotransmitter: Any of a group of substances that are released on excitation from the axon terminal of a presynaptic neuron of the central or peripheral nervous system and travel across the synaptic cleft to either excite or inhibit the target cell. Among the many substances that have the properties of a neurotransmitter are acetylcholine, norepinephrine, epinephrine, dopamine, glycine, y-aminobutyrate, glutamic acid, substance P, enkephalins, endorphins, and serotonin. [EU]

Neutrons: Electrically neutral elementary particles found in all atomic nuclei except light hydrogen; the mass is equal to that of the proton and electron combined and they are unstable when isolated from the nucleus, undergoing beta decay. Slow, thermal, epithermal, and fast neutrons refer to the energy levels with which the neutrons are ejected from heavier nuclei during their decay. [NIH]

Neutrophils: Granular leukocytes having a nucleus with three to five lobes connected by slender threads of chromatin, and cytoplasm containing fine inconspicuous granules and stainable by neutral dyes. [NIH]

Nitrogen: An element with the atomic symbol N, atomic number 7, and atomic weight 14. Nitrogen exists as a diatomic gas and makes up about 78% of the earth's atmosphere by volume. It is a constituent of proteins and nucleic acids and found in all living cells. [NIH]

Nuclear: A test of the structure, blood flow, and function of the kidneys. The doctor injects a mildly radioactive solution into an arm vein and uses x-rays to monitor its progress through the kidneys. [NIH]

Nuclear Matrix: The fibrogranular network of residual structural elements within which are immersed both chromatin and ribonucleoproteins. It extends throughout the nuclear interior from the nucleolus to the nuclear pore complexes along the nuclear periphery. [NIH]

Nuclear Pore: An opening through the nuclear envelope formed by the nuclear pore complex which transports nuclear proteins or RNA into or out of the cell nucleus and which, under some conditions, acts as an ion channel. [NIH]

Nuclei: A body of specialized protoplasm found in nearly all cells and containing the chromosomes. [NIH]

Nucleic acid: Either of two types of macromolecule (DNA or RNA) formed by polymerization of nucleotides. Nucleic acids are found in all living cells and contain the

information (genetic code) for the transfer of genetic information from one generation to the next. [NIH]

Nucleic Acid Hybridization: The process whereby two single-stranded polynucleotides form a double-stranded molecule, with hydrogen bonding between the complementary bases in the two strains. [NIH]

Nucleus: A body of specialized protoplasm found in nearly all cells and containing the chromosomes. [NIH]

Ocular: 1. Of, pertaining to, or affecting the eye. 2. Eyepiece. [EU]

Oncogene: A gene that normally directs cell growth. If altered, an oncogene can promote or allow the uncontrolled growth of cancer. Alterations can be inherited or caused by an environmental exposure to carcinogens. [NIH]

Oncogenic: Chemical, viral, radioactive or other agent that causes cancer; carcinogenic. [NIH]

Oncology: The study of cancer. [NIH]

Oncolysis: The destruction of or disposal by absorption of any neoplastic cells. [NIH]

Oncolytic: Pertaining to, characterized by, or causing oncolysis (= the lysis or destruction of tumour cells). [EU]

Operon: The genetic unit consisting of a feedback system under the control of an operator gene, in which a structural gene transcribes its message in the form of mRNA upon blockade of a repressor produced by a regulator gene. Included here is the attenuator site of bacterial operons where transcription termination is regulated. [NIH]

Ophthalmoplegia: Paralysis of one or more of the ocular muscles due to disorders of the eye muscles, neuromuscular junction, supporting soft tissue, tendons, or innervation to the muscles. [NIH]

Opportunistic Infections: An infection caused by an organism which becomes pathogenic under certain conditions, e.g., during immunosuppression. [NIH]

Oral Health: The optimal state of the mouth and normal functioning of the organs of the mouth without evidence of disease. [NIH]

Oral Hygiene: The practice of personal hygiene of the mouth. It includes the maintenance of oral cleanliness, tissue tone, and general preservation of oral health. [NIH]

Orbit: One of the two cavities in the skull which contains an eyeball. Each eye is located in a bony socket or orbit. [NIH]

Orbital: Pertaining to the orbit (= the bony cavity that contains the eyeball). [EU]

Organ Culture: The growth in aseptic culture of plant organs such as roots or shoots, beginning with organ primordia or segments and maintaining the characteristics of the organ. [NIH]

Organelles: Specific particles of membrane-bound organized living substances present in eukaryotic cells, such as the mitochondria; the golgi apparatus; endoplasmic reticulum; lysomomes; plastids; and vacuoles. [NIH]

Ornithine: An amino acid produced in the urea cycle by the splitting off of urea from arginine. [NIH]

Ornithine Decarboxylase: A pyridoxal-phosphate protein, believed to be the rate-limiting compound in the biosynthesis of polyamines. It catalyzes the decarboxylation of ornithine to form putrescine, which is then linked to a propylamine moiety of decarboxylated S-adenosylmethionine to form spermidine. EC 4.1.1.17. [NIH]

Ovary: Either of the paired glands in the female that produce the female germ cells and

secrete some of the female sex hormones. [NIH]

Oxidative Phosphorylation: Electron transfer through the cytochrome system liberating free energy which is transformed into high-energy phosphate bonds. [NIH]

P53 gene: A tumor suppressor gene that normally inhibits the growth of tumors. This gene is altered in many types of cancer. [NIH]

Pachymeningitis: Inflammation of the dura mater of the brain, the spinal cord or the optic nerve. [NIH]

Palate: The structure that forms the roof of the mouth. It consists of the anterior hard palate and the posterior soft palate. [NIH]

Palliative: 1. Affording relief, but not cure. 2. An alleviating medicine. [EU]

Palsy: Disease of the peripheral nervous system occurring usually after many years of increased lead absorption. [NIH]

Pancreas: A mixed exocrine and endocrine gland situated transversely across the posterior abdominal wall in the epigastric and hypochondriac regions. The endocrine portion is comprised of the Islets of Langerhans, while the exocrine portion is a compound acinar gland that secretes digestive enzymes. [NIH]

Pancreatic: Having to do with the pancreas. [NIH]

Paraplegia: Severe or complete loss of motor function in the lower extremities and lower portions of the trunk. This condition is most often associated with spinal cord diseases, although brain diseases; peripheral nervous system diseases; neuromuscular diseases; and muscular diseases may also cause bilateral leg weakness. [NIH]

Partial remission: The shrinking, but not complete disappearance, of a tumor in response to therapy. Also called partial response. [NIH]

Particle: A tiny mass of material. [EU]

Patch: A piece of material used to cover or protect a wound, an injured part, etc.: a patch over the eye. [NIH]

Pathogen: Any disease-producing microorganism. [EU]

Pathologic: 1. Indicative of or caused by a morbid condition. 2. Pertaining to pathology (= branch of medicine that treats the essential nature of the disease, especially the structural and functional changes in tissues and organs of the body caused by the disease). [EU]

Pathologic Processes: The abnormal mechanisms and forms involved in the dysfunctions of tissues and organs. [NIH]

Peptide: Any compound consisting of two or more amino acids, the building blocks of proteins. Peptides are combined to make proteins. [NIH]

Peptide T: N-(N-(N(2)-(N-(N-(N-(N-D-Alanyl L-seryl)-L-threonyl)-L-threonyl) L-threonyl)-L-asparaginyl)-L-tyrosyl) L-threonine. Octapeptide sharing sequence homology with HIV envelope protein gp120. It is potentially useful as antiviral agent in AIDS therapy. The core pentapeptide sequence, TTNYT, consisting of amino acids 4-8 in peptide T, is the HIV envelope sequence required for attachment to the CD4 receptor. [NIH]

Peripheral blood: Blood circulating throughout the body. [NIH]

Peripheral Nervous System: The nervous system outside of the brain and spinal cord. The peripheral nervous system has autonomic and somatic divisions. The autonomic nervous system includes the enteric, parasympathetic, and sympathetic subdivisions. The somatic nervous system includes the cranial and spinal nerves and their ganglia and the peripheral sensory receptors. [NIH]

Peripheral Nervous System Diseases: Diseases of the peripheral nerves external to the brain and spinal cord, which includes diseases of the nerve roots, ganglia, plexi, autonomic nerves, sensory nerves, and motor nerves. [NIH]

Pharmacologic: Pertaining to pharmacology or to the properties and reactions of drugs. [EU]

Pharynx: The hollow tube about 5 inches long that starts behind the nose and ends at the top of the trachea (windpipe) and esophagus (the tube that goes to the stomach). [NIH]

Phenotype: The outward appearance of the individual. It is the product of interactions between genes and between the genotype and the environment. This includes the killer phenotype, characteristic of yeasts. [NIH]

Phlebotomy: The letting of blood from a vein. Although it is one of the techniques used in drawing blood to be used in diagnostic procedures, in modern medicine, it is used commonly in the treatment of erythrocytosis, hemochromocytosis, polycythemia vera, and porphyria cutanea tarda. Its historical counterpart is bloodletting. (From Cecil Textbook of Medicine, 19th ed & Wintrobe's Clinical Hematology, 9th ed) Venipuncture is not only for the letting of blood from a vein but also for the injecting of a drug into the vein for diagnostic analysis. [NIH]

Phorbol: Class of chemicals that promotes the development of tumors. [NIH]

Phospholipases: A class of enzymes that catalyze the hydrolysis of phosphoglycerides or glycerophosphatidates. EC 3.1.-. [NIH]

Phosphorus: A non-metallic element that is found in the blood, muscles, nevers, bones, and teeth, and is a component of adenosine triphosphate (ATP; the primary energy source for the body's cells.) [NIH]

Phosphorylated: Attached to a phosphate group. [NIH]

Phosphorylation: The introduction of a phosphoryl group into a compound through the formation of an ester bond between the compound and a phosphorus moiety. [NIH]

Photocoagulation: Using a special strong beam of light (laser) to seal off bleeding blood vessels such as in the eye. The laser can also burn away blood vessels that should not have grown in the eye. This is the main treatment for diabetic retinopathy. [NIH]

Physiologic: Having to do with the functions of the body. When used in the phrase "physiologic age," it refers to an age assigned by general health, as opposed to calendar age. [NIH]

Physiology: The science that deals with the life processes and functions of organismus, their cells, tissues, and organs. [NIH]

Phytohemagglutinins: Mucoproteins isolated from the kidney bean (Phaseolus vulgaris); some of them are mitogenic to lymphocytes, others agglutinate all or certain types of erythrocytes or lymphocytes. They are used mainly in the study of immune mechanisms and in cell culture. [NIH]

Pigment: A substance that gives color to tissue. Pigments are responsible for the color of skin, eyes, and hair. [NIH]

Pilot study: The initial study examining a new method or treatment. [NIH]

Pituitary Gland: A small, unpaired gland situated in the sella turcica tissue. It is connected to the hypothalamus by a short stalk. [NIH]

Plants: Multicellular, eukaryotic life forms of the kingdom Plantae. They are characterized by a mainly photosynthetic mode of nutrition; essentially unlimited growth at localized regions of cell divisions (meristems); cellulose within cells providing rigidity; the absence of organs of locomotion; absense of nervous and sensory systems; and an alteration of haploid

and diploid generations. [NIH]

Plasma: The clear, yellowish, fluid part of the blood that carries the blood cells. The proteins that form blood clots are in plasma. [NIH]

Plasma cells: A type of white blood cell that produces antibodies. [NIH]

Plasmacytoma: Any discrete, presumably solitary, mass of neoplastic plasma cells either in bone marrow or various extramedullary sites. [NIH]

Plasmid: An autonomously replicating, extra-chromosomal DNA molecule found in many bacteria. Plasmids are widely used as carriers of cloned genes. [NIH]

Platelet Activation: A series of progressive, overlapping events triggered by exposure of the platelets to subendothelial tissue. These events include shape change, adhesiveness, aggregation, and release reactions. When carried through to completion, these events lead to the formation of a stable hemostatic plug. [NIH]

Pneumonia: Inflammation of the lungs. [NIH]

Podophyllotoxin: The main active constituent of the resin from the roots of may apple or mandrake (Podophyllum peltatum and P. emodi). It is a potent spindle poison, toxic if taken internally, and has been used as a cathartic. It is very irritating to skin and mucous membranes, has keratolytic actions, has been used to treat warts and keratoses, and may have antineoplastic properties, as do some of its congeners and derivatives. [NIH]

Polymerase: An enzyme which catalyses the synthesis of DNA using a single DNA strand as a template. The polymerase copies the template in the 5'-3'direction provided that sufficient quantities of free nucleotides, dATP and dTTP are present. [NIH]

Polymerase Chain Reaction: In vitro method for producing large amounts of specific DNA or RNA fragments of defined length and sequence from small amounts of short oligonucleotide flanking sequences (primers). The essential steps include thermal denaturation of the double-stranded target molecules, annealing of the primers to their complementary sequences, and extension of the annealed primers by enzymatic synthesis with DNA polymerase. The reaction is efficient, specific, and extremely sensitive. Uses for the reaction include disease diagnosis, detection of difficult-to-isolate pathogens, mutation analysis, genetic testing, DNA sequencing, and analyzing evolutionary relationships. [NIH]

Polymorphism: The occurrence together of two or more distinct forms in the same population. [NIH]

Polypeptide: A peptide which on hydrolysis yields more than two amino acids; called tripeptides, tetrapeptides, etc. according to the number of amino acids contained. [EU]

Polysaccharide: A type of carbohydrate. It contains sugar molecules that are linked together chemically. [NIH]

Porphyria: A group of disorders characterized by the excessive production of porphyrins or their precursors that arises from abnormalities in the regulation of the porphyrin-heme pathway. The porphyrias are usually divided into three broad groups, erythropoietic, hepatic, and erythrohepatic, according to the major sites of abnormal porphyrin synthesis. [NIH]

Porphyria Cutanea Tarda: A form of hepatic porphyria (porphyria, hepatic) characterized by photosensitivity resulting in bullae that rupture easily to form shallow ulcers. This condition occurs in two forms: a sporadic, nonfamilial form that begins in middle age and has normal amounts of uroporphyrinogen decarboxylase with diminished activity in the liver; and a familial form in which there is an autosomal dominant inherited deficiency of uroporphyrinogen decarboxylase in the liver and red blood cells. [NIH]

Posterior: Situated in back of, or in the back part of, or affecting the back or dorsal surface of

the body. In lower animals, it refers to the caudal end of the body. [EU]

Postsynaptic: Nerve potential generated by an inhibitory hyperpolarizing stimulation. [NIH]

Potentiates: A degree of synergism which causes the exposure of the organism to a harmful substance to worsen a disease already contracted. [NIH]

Potentiation: An overall effect of two drugs taken together which is greater than the sum of the effects of each drug taken alone. [NIH]

Practice Guidelines: Directions or principles presenting current or future rules of policy for the health care practitioner to assist him in patient care decisions regarding diagnosis, therapy, or related clinical circumstances. The guidelines may be developed by government agencies at any level, institutions, professional societies, governing boards, or by the convening of expert panels. The guidelines form a basis for the evaluation of all aspects of health care and delivery. [NIH]

Precursor: Something that precedes. In biological processes, a substance from which another, usually more active or mature substance is formed. In clinical medicine, a sign or symptom that heralds another. [EU]

Predisposition: A latent susceptibility to disease which may be activated under certain conditions, as by stress. [EU]

Prevalence: The total number of cases of a given disease in a specified population at a designated time. It is differentiated from incidence, which refers to the number of new cases in the population at a given time. [NIH]

Primary central nervous system lymphoma: Cancer that arises in the lymphoid tissue found in the central nervous system (CNS). The CNS includes the brain and spinal cord. [NIH]

Primary tumor: The original tumor. [NIH]

Prodrug: A substance that gives rise to a pharmacologically active metabolite, although not itself active (i. e. an inactive precursor). [NIH]

Progeny: The offspring produced in any generation. [NIH]

Prognostic factor: A situation or condition, or a characteristic of a patient, that can be used to estimate the chance of recovery from a disease, or the chance of the disease recurring (coming back). [NIH]

Progression: Increase in the size of a tumor or spread of cancer in the body. [NIH]

Progressive: Advancing; going forward; going from bad to worse; increasing in scope or severity. [EU]

Projection: A defense mechanism, operating unconsciously, whereby that which is emotionally unacceptable in the self is rejected and attributed (projected) to others. [NIH]

Promoter: A chemical substance that increases the activity of a carcinogenic process. [NIH]

Prophase: The first phase of cell division, in which the chromosomes become visible, the nucleus starts to lose its identity, the spindle appears, and the centrioles migrate toward opposite poles. [NIH]

Prophylaxis: An attempt to prevent disease. [NIH]

Proptosis: Forward projection or displacement especially of the eyeball : exophthalmos. [EU]

Prospective study: An epidemiologic study in which a group of individuals (a cohort), all free of a particular disease and varying in their exposure to a possible risk factor, is followed over a specific amount of time to determine the incidence rates of the disease in the exposed and unexposed groups. [NIH]

Prostate: A gland in males that surrounds the neck of the bladder and the urethra. It secretes a substance that liquifies coagulated semen. It is situated in the pelvic cavity behind the lower part of the pubic symphysis, above the deep layer of the triangular ligament, and rests upon the rectum. [NIH]

Protease: Proteinase (= any enzyme that catalyses the splitting of interior peptide bonds in a protein). [EU]

Protein Binding: The process in which substances, either endogenous or exogenous, bind to proteins, peptides, enzymes, protein precursors, or allied compounds. Specific protein-binding measures are often used as assays in diagnostic assessments. [NIH]

Protein C: A vitamin-K dependent zymogen present in the blood, which, upon activation by thrombin and thrombomodulin exerts anticoagulant properties by inactivating factors Va and VIIIa at the rate-limiting steps of thrombin formation. [NIH]

Protein Isoforms: Different forms of a protein that may be produced from different genes, or from the same gene by alternative splicing. [NIH]

Protein S: The vitamin K-dependent cofactor of activated protein C. Together with protein C, it inhibits the action of factors VIIIa and Va. A deficiency in protein S can lead to recurrent venous and arterial thrombosis. [NIH]

Proteins: Polymers of amino acids linked by peptide bonds. The specific sequence of amino acids determines the shape and function of the protein. [NIH]

Proteolytic: 1. Pertaining to, characterized by, or promoting proteolysis. 2. An enzyme that promotes proteolysis (= the splitting of proteins by hydrolysis of the peptide bonds with formation of smaller polypeptides). [EU]

Protocol: The detailed plan for a clinical trial that states the trial's rationale, purpose, drug or vaccine dosages, length of study, routes of administration, who may participate, and other aspects of trial design. [NIH]

Protons: Stable elementary particles having the smallest known positive charge, found in the nuclei of all elements. The proton mass is less than that of a neutron. A proton is the nucleus of the light hydrogen atom, i.e., the hydrogen ion. [NIH]

Protozoa: A subkingdom consisting of unicellular organisms that are the simplest in the animal kingdom. Most are free living. They range in size from submicroscopic to macroscopic. Protozoa are divided into seven phyla: Sarcomastigophora, Labyrinthomorpha, Apicomplexa, Microspora, Ascetospora, Myxozoa, and Ciliophora. [NIH]

Protozoal: Having to do with the simplest organisms in the animal kingdom. Protozoa are single-cell organisms, such as ameba, and are different from bacteria, which are not members of the animal kingdom. Some protozoa can be seen without a microscope. [NIH]

Protozoan: 1. Any individual of the protozoa; protozoon. 2. Of or pertaining to the protozoa; protozoal. [EU]

Proximal: Nearest; closer to any point of reference; opposed to distal. [EU]

Pseudorabies: A highly contagious herpesvirus infection affecting the central nervous system of swine, cattle, dogs, cats, rats, and other animals. [NIH]

Psychic: Pertaining to the psyche or to the mind; mental. [EU]

Puberty: The period during which the secondary sex characteristics begin to develop and the capability of sexual reproduction is attained. [EU]

Public Policy: A course or method of action selected, usually by a government, from among alternatives to guide and determine present and future decisions. [NIH]

Publishing: "The business or profession of the commercial production and issuance of

literature" (Webster's 3d). It includes the publisher, publication processes, editing and editors. Production may be by conventional printing methods or by electronic publishing. [NIH]

Pulse: The rhythmical expansion and contraction of an artery produced by waves of pressure caused by the ejection of blood from the left ventricle of the heart as it contracts. [NIH]

Putrescine: A toxic diamine formed by putrefaction from the decarboxylation of arginine and ornithine. [NIH]

Pyridoxal: 3-Hydroxy-5-(hydroxymethyl)-2-methyl-4- pyridinecarboxaldehyde. [NIH]

Quality of Health Care: The levels of excellence which characterize the health service or health care provided based on accepted standards of quality. [NIH]

Quiescent: Marked by a state of inactivity or repose. [EU]

Race: A population within a species which exhibits general similarities within itself, but is both discontinuous and distinct from other populations of that species, though not sufficiently so as to achieve the status of a taxon. [NIH]

Radiation: Emission or propagation of electromagnetic energy (waves/rays), or the waves/rays themselves; a stream of electromagnetic particles (electrons, neutrons, protons, alpha particles) or a mixture of these. The most common source is the sun. [NIH]

Radiation therapy: The use of high-energy radiation from x-rays, gamma rays, neutrons, and other sources to kill cancer cells and shrink tumors. Radiation may come from a machine outside the body (external-beam radiation therapy), or it may come from radioactive material placed in the body in the area near cancer cells (internal radiation therapy, implant radiation, or brachytherapy). Systemic radiation therapy uses a radioactive substance, such as a radiolabeled monoclonal antibody, that circulates throughout the body. Also called radiotherapy. [NIH]

Radioactive: Giving off radiation. [NIH]

Radiolabeled: Any compound that has been joined with a radioactive substance. [NIH]

Radionuclide Imaging: Process whereby a radionuclide is injected or measured (through tissue) from an external source, and a display is obtained from any one of several rectilinear scanner or gamma camera systems. The image obtained from a moving detector is called a scan, while the image obtained from a stationary camera device is called a scintiphotograph. [NIH]

Radiotherapy: The use of ionizing radiation to treat malignant neoplasms and other benign conditions. The most common forms of ionizing radiation used as therapy are x-rays, gamma rays, and electrons. A special form of radiotherapy, targeted radiotherapy, links a cytotoxic radionuclide to a molecule that targets the tumor. When this molecule is an antibody or other immunologic molecule, the technique is called radioimmunotherapy. [NIH]

Ramus: Most commonly used for branches of nerves, but applied also to other structures. [NIH]

Randomized: Describes an experiment or clinical trial in which animal or human subjects are assigned by chance to separate groups that compare different treatments. [NIH]

Randomized clinical trial: A study in which the participants are assigned by chance to separate groups that compare different treatments; neither the researchers nor the participants can choose which group. Using chance to assign people to groups means that the groups will be similar and that the treatments they receive can be compared objectively. At the time of the trial, it is not known which treatment is best. It is the patient's choice to be in a randomized trial. [NIH]

Reactivation: The restoration of activity to something that has been inactivated. [EU]

Reading Frames: The sequence of codons by which translation may occur. A segment of mRNA 5'AUCCGA3' could be translated in three reading frames, 5'AUC. or 5'UCC. or 5'CCG., depending on the location of the start codon. [NIH]

Receptor: A molecule inside or on the surface of a cell that binds to a specific substance and causes a specific physiologic effect in the cell. [NIH]

Recombinant: A cell or an individual with a new combination of genes not found together in either parent; usually applied to linked genes. [EU]

Recombination: The formation of new combinations of genes as a result of segregation in crosses between genetically different parents; also the rearrangement of linked genes due to crossing-over. [NIH]

Red blood cells: RBCs. Cells that carry oxygen to all parts of the body. Also called erythrocytes. [NIH]

Reductase: Enzyme converting testosterone to dihydrotestosterone. [NIH]

Refer: To send or direct for treatment, aid, information, de decision. [NIH]

Refraction: A test to determine the best eyeglasses or contact lenses to correct a refractive error (myopia, hyperopia, or astigmatism). [NIH]

Refractory: Not readily yielding to treatment. [EU]

Regimen: A treatment plan that specifies the dosage, the schedule, and the duration of treatment. [NIH]

Relapse: The return of signs and symptoms of cancer after a period of improvement. [NIH]

Remission: A decrease in or disappearance of signs and symptoms of cancer. In partial remission, some, but not all, signs and symptoms of cancer have disappeared. In complete remission, all signs and symptoms of cancer have disappeared, although there still may be cancer in the body. [NIH]

Repressor: Any of the specific allosteric protein molecules, products of regulator genes, which bind to the operator of operons and prevent RNA polymerase from proceeding into the operon to transcribe messenger RNA. [NIH]

Resection: Removal of tissue or part or all of an organ by surgery. [NIH]

Retinoblastoma: An eye cancer that most often occurs in children younger than 5 years. It occurs in hereditary and nonhereditary (sporadic) forms. [NIH]

Retrospective: Looking back at events that have already taken place. [NIH]

Retroviral vector: RNA from a virus that is used to insert genetic material into cells. [NIH]

Retrovirus: A member of a group of RNA viruses, the RNA of which is copied during viral replication into DNA by reverse transcriptase. The viral DNA is then able to be integrated into the host chromosomal DNA. [NIH]

Rheumatoid: Resembling rheumatism. [EU]

Rheumatoid arthritis: A form of arthritis, the cause of which is unknown, although infection, hypersensitivity, hormone imbalance and psychologic stress have been suggested as possible causes. [NIH]

Rhinitis: Inflammation of the mucous membrane of the nose. [NIH]

Ribonucleoproteins: Proteins conjugated with ribonucleic acids (RNA) or specific RNA. Many viruses are ribonucleoproteins. [NIH]

Ribonucleoside Diphosphate Reductase: An enzyme of the oxidoreductase class that

catalyzes the formation of 2'-deoxyribonucleotides from the corresponding ribonucleotides using NADPH as the ultimate electron donor. The deoxyribonucleoside diphosphates are used in DNA synthesis. (From Dorland, 27th ed) EC 1.17.4.1. [NIH]

Ribose: A pentose active in biological systems usually in its D-form. [NIH]

Ribosome: A granule of protein and RNA, synthesized in the nucleolus and found in the cytoplasm of cells. Ribosomes are the main sites of protein synthesis. Messenger RNA attaches to them and there receives molecules of transfer RNA bearing amino acids. [NIH]

Risk factor: A habit, trait, condition, or genetic alteration that increases a person's chance of developing a disease. [NIH]

Rituximab: A type of monoclonal antibody used in cancer detection or therapy. Monoclonal antibodies are laboratory-produced substances that can locate and bind to cancer cells. [NIH]

Sagittal: The line of direction passing through the body from back to front, or any vertical plane parallel to the medial plane of the body and inclusive of that plane; often restricted to the medial plane, the plane of the sagittal suture. [NIH]

Salivary: The duct that convey saliva to the mouth. [NIH]

Salivary glands: Glands in the mouth that produce saliva. [NIH]

Sarcoma: A connective tissue neoplasm formed by proliferation of mesodermal cells; it is usually highly malignant. [NIH]

Screening: Checking for disease when there are no symptoms. [NIH]

Sedimentation: The act of causing the deposit of sediment, especially by the use of a centrifugal machine. [EU]

Segregation: The separation in meiotic cell division of homologous chromosome pairs and their contained allelomorphic gene pairs. [NIH]

Sella: A deep depression in the shape of a Turkish saddle in the upper surface of the body of the sphenoid bone in the deepest part of which is lodged the hypophysis cerebri. [NIH]

Sella Turcica: A bony prominence situated on the upper surface of the body of the sphenoid bone. It houses the pituitary gland. [NIH]

Semisynthetic: Produced by chemical manipulation of naturally occurring substances. [EU]

Sensitization: 1. Administration of antigen to induce a primary immune response; priming; immunization. 2. Exposure to allergen that results in the development of hypersensitivity. 3. The coating of erythrocytes with antibody so that they are subject to lysis by complement in the presence of homologous antigen, the first stage of a complement fixation test. [EU]

Septicaemia: A term originally used to denote a putrefactive process in the body, but now usually referring to infection with pyogenic micro-organisms; a genus of Diptera; the severe type of infection in which the blood stream is invaded by large numbers of the causal. [NIH]

Sequence Analysis: A multistage process that includes the determination of a sequence (protein, carbohydrate, etc.), its fragmentation and analysis, and the interpretation of the resulting sequence information. [NIH]

Sequence Homology: The degree of similarity between sequences. Studies of amino acid and nucleotide sequences provide useful information about the genetic relatedness of certain species. [NIH]

Sequencing: The determination of the order of nucleotides in a DNA or RNA chain. [NIH]

Serine: A non-essential amino acid occurring in natural form as the L-isomer. It is synthesized from glycine or threonine. It is involved in the biosynthesis of purines, pyrimidines, and other amino acids. [NIH]

Serotypes: A cause of haemorrhagic septicaemia (in cattle, sheep and pigs), fowl cholera of birds, pasteurellosis of rabbits, and gangrenous mastitis of ewes. It is also commonly found in atrophic rhinitis of pigs. [NIH]

Serum: The clear liquid part of the blood that remains after blood cells and clotting proteins have been removed. [NIH]

Sex Characteristics: Those characteristics that distinguish one sex from the other. The primary sex characteristics are the ovaries and testes and their related hormones. Secondary sex characteristics are those which are masculine or feminine but not directly related to reproduction. [NIH]

Shock: The general bodily disturbance following a severe injury; an emotional or moral upset occasioned by some disturbing or unexpected experience; disruption of the circulation, which can upset all body functions: sometimes referred to as circulatory shock. [NIH]

Side effect: A consequence other than the one(s) for which an agent or measure is used, as the adverse effects produced by a drug, especially on a tissue or organ system other than the one sought to be benefited by its administration. [EU]

Signal Transduction: The intercellular or intracellular transfer of information (biological activation/inhibition) through a signal pathway. In each signal transduction system, an activation/inhibition signal from a biologically active molecule (hormone, neurotransmitter) is mediated via the coupling of a receptor/enzyme to a second messenger system or to an ion channel. Signal transduction plays an important role in activating cellular functions, cell differentiation, and cell proliferation. Examples of signal transduction systems are the GABA-postsynaptic receptor-calcium ion channel system, the receptor-mediated T-cell activation pathway, and the receptor-mediated activation of phospholipases. Those coupled to membrane depolarization or intracellular release of calcium include the receptor-mediated activation of cytotoxic functions in granulocytes and the synaptic potentiation of protein kinase activation. Some signal transduction pathways may be part of larger signal transduction pathways; for example, protein kinase activation is part of the platelet activation signal pathway. [NIH]

Signs and Symptoms: Clinical manifestations that can be either objective when observed by a physician, or subjective when perceived by the patient. [NIH]

Single-agent: The use of a single drug or other therapy. [NIH]

Skull: The skeleton of the head including the bones of the face and the bones enclosing the brain. [NIH]

Small intestine: The part of the digestive tract that is located between the stomach and the large intestine. [NIH]

Sodium: An element that is a member of the alkali group of metals. It has the atomic symbol Na, atomic number 11, and atomic weight 23. With a valence of 1, it has a strong affinity for oxygen and other nonmetallic elements. Sodium provides the chief cation of the extracellular body fluids. Its salts are the most widely used in medicine. (From Dorland, 27th ed) Physiologically the sodium ion plays a major role in blood pressure regulation, maintenance of fluid volume, and electrolyte balance. [NIH]

Soft tissue: Refers to muscle, fat, fibrous tissue, blood vessels, or other supporting tissue of the body. [NIH]

Solid tumor: Cancer of body tissues other than blood, bone marrow, or the lymphatic system. [NIH]

Soma: The body as distinct from the mind; all the body tissue except the germ cells; all the axial body. [NIH]

Somatic: 1. Pertaining to or characteristic of the soma or body. 2. Pertaining to the body wall in contrast to the viscera. [EU]

Somatic cells: All the body cells except the reproductive (germ) cells. [NIH]

Specialist: In medicine, one who concentrates on 1 special branch of medical science. [NIH]

Species: A taxonomic category subordinate to a genus (or subgenus) and superior to a subspecies or variety, composed of individuals possessing common characters distinguishing them from other categories of individuals of the same taxonomic level. In taxonomic nomenclature, species are designated by the genus name followed by a Latin or Latinized adjective or noun. [EU]

Specificity: Degree of selectivity shown by an antibody with respect to the number and types of antigens with which the antibody combines, as well as with respect to the rates and the extents of these reactions. [NIH]

Spectrum: A charted band of wavelengths of electromagnetic vibrations obtained by refraction and diffraction. By extension, a measurable range of activity, such as the range of bacteria affected by an antibiotic (antibacterial s.) or the complete range of manifestations of a disease. [EU]

Sperm: The fecundating fluid of the male. [NIH]

Spermidine: A polyamine formed from putrescine. It is found in almost all tissues in association with nucleic acids. It is found as a cation at all pH values, and is thought to help stabilize some membranes and nucleic acid structures. It is a precursor of spermine. [NIH]

Spermine: A biogenic polyamine formed from spermidine. It is found in a wide variety of organisms and tissues and is an essential growth factor in some bacteria. It is found as a polycation at all pH values. Spermine is associated with nucleic acids, particularly in viruses, and is thought to stabilize the helical structure. [NIH]

Sphenoid: An unpaired cranial bone with a body containing the sphenoid sinus and forming the posterior part of the medial walls of the orbits. [NIH]

Spinal cord: The main trunk or bundle of nerves running down the spine through holes in the spinal bone (the vertebrae) from the brain to the level of the lower back. [NIH]

Spinal Cord Diseases: Pathologic conditions which feature spinal cord damage or dysfunction, including disorders involving the meninges and perimeningeal spaces surrounding the spinal cord. Traumatic injuries, vascular diseases, infections, and inflammatory/autoimmune processes may affect the spinal cord. [NIH]

Spleen: An organ that is part of the lymphatic system. The spleen produces lymphocytes, filters the blood, stores blood cells, and destroys old blood cells. It is located on the left side of the abdomen near the stomach. [NIH]

Splenomegaly: Enlargement of the spleen. [NIH]

Sporadic: Neither endemic nor epidemic; occurring occasionally in a random or isolated manner. [EU]

Stabilization: The creation of a stable state. [EU]

Stem Cell Factor: Hematopoietic growth factor and the ligand of the c-kit receptor CD117 (proto-oncogene protein C-kit). It is expressed during embryogenesis and provides a key signal in multiple aspects of mast-cell differentiation and function. [NIH]

Sterility: 1. The inability to produce offspring, i.e., the inability to conceive (female s.) or to induce conception (male s.). 2. The state of being aseptic, or free from microorganisms. [EU]

Steroids: Drugs used to relieve swelling and inflammation. [NIH]

Stimulus: That which can elicit or evoke action (response) in a muscle, nerve, gland or other

excitable issue, or cause an augmenting action upon any function or metabolic process. [NIH]

Stomach: An organ of digestion situated in the left upper quadrant of the abdomen between the termination of the esophagus and the beginning of the duodenum. [NIH]

Strand: DNA normally exists in the bacterial nucleus in a helix, in which two strands are coiled together. [NIH]

Streptomycin: O-2-Deoxy-2-(methylamino)-alpha-L-glucopyranosyl-(1-2)-O-5- deoxy-3-C-formyl-alpha-L-lyxofuranosyl-(1-4)-N,N'-bis- (aminoiminomethyl)-D-streptamine. Antibiotic substance produced by the soil actinomycete Streptomyces griseus. It acts by inhibiting the initiation and elongation processes during protein synthesis. [NIH]

Stress: Forcibly exerted influence; pressure. Any condition or situation that causes strain or tension. Stress may be either physical or psychologic, or both. [NIH]

Stromal: Large, veil-like cell in the bone marrow. [NIH]

Stromal Cells: Connective tissue cells of an organ found in the loose connective tissue. These are most often associated with the uterine mucosa and the ovary as well as the hematopoietic system and elsewhere. [NIH]

Subacute: Somewhat acute; between acute and chronic. [EU]

Subclinical: Without clinical manifestations; said of the early stage(s) of an infection or other disease or abnormality before symptoms and signs become apparent or detectable by clinical examination or laboratory tests, or of a very mild form of an infection or other disease or abnormality. [EU]

Substrate: A substance upon which an enzyme acts. [EU]

Substrate Specificity: A characteristic feature of enzyme activity in relation to the kind of substrate on which the enzyme or catalytic molecule reacts. [NIH]

Superinfection: A frequent complication of drug therapy for microbial infection. It may result from opportunistic colonization following immunosuppression by the primary pathogen and can be influenced by the time interval between infections, microbial physiology, or host resistance. Experimental challenge and in vitro models are sometimes used in virulence and infectivity studies. [NIH]

Suppression: A conscious exclusion of disapproved desire contrary with repression, in which the process of exclusion is not conscious. [NIH]

Suppressive: Tending to suppress : effecting suppression; specifically : serving to suppress activity, function, symptoms. [EU]

Synaptic: Pertaining to or affecting a synapse (= site of functional apposition between neurons, at which an impulse is transmitted from one neuron to another by electrical or chemical means); pertaining to synapsis (= pairing off in point-for-point association of homologous chromosomes from the male and female pronuclei during the early prophase of meiosis). [EU]

Synchrony: The normal physiologic sequencing of atrial and ventricular activation and contraction. [NIH]

Syncope: A temporary suspension of consciousness due to generalized cerebral schemia, a faint or swoon. [EU]

Synergistic: Acting together; enhancing the effect of another force or agent. [EU]

Synthetic retinoid: A substance related to vitamin A that is produced in a laboratory. [NIH]

Systemic: Affecting the entire body. [NIH]

Telomere: A terminal section of a chromosome which has a specialized structure and which

is involved in chromosomal replication and stability. Its length is believed to be a few hundred base pairs. [NIH]

Teniposide: A semisynthetic derivative of podophyllotoxin that exhibits antitumor activity. Teniposide inhibits DNA synthesis by forming a complex with topoisomerase II and DNA. This complex induces breaks in double stranded DNA and prevents repair by topoisomerase II binding. Accumulated breaks in DNA prevent cells from entering into the mitotic phase of the cell cycle, and lead to cell death. Teniposide acts primarily in the G2 and S phases of the cycle. [NIH]

Testicular: Pertaining to a testis. [EU]

Testis: Either of the paired male reproductive glands that produce the male germ cells and the male hormones. [NIH]

Tetracycline: An antibiotic originally produced by Streptomyces viridifaciens, but used mostly in synthetic form. It is an inhibitor of aminoacyl-tRNA binding during protein synthesis. [NIH]

Tetravalent: Pertaining to a group of 4 homologous or partly homologous chromosomes during the zygotene stage of prophase to the first metaphase in meiosis. [NIH]

Thalassemia: A group of hereditary hemolytic anemias in which there is decreased synthesis of one or more hemoglobin polypeptide chains. There are several genetic types with clinical pictures ranging from barely detectable hematologic abnormality to severe and fatal anemia. [NIH]

Therapeutics: The branch of medicine which is concerned with the treatment of diseases, palliative or curative. [NIH]

Thermal: Pertaining to or characterized by heat. [EU]

Threonine: An essential amino acid occurring naturally in the L-form, which is the active form. It is found in eggs, milk, gelatin, and other proteins. [NIH]

Thrombin: An enzyme formed from prothrombin that converts fibrinogen to fibrin. (Dorland, 27th ed) EC 3.4.21.5. [NIH]

Thrombomodulin: A cell surface glycoprotein of endothelial cells that binds thrombin and serves as a cofactor in the activation of protein C and its regulation of blood coagulation. [NIH]

Thrombosis: The formation or presence of a blood clot inside a blood vessel. [NIH]

Thymidine: A chemical compound found in DNA. Also used as treatment for mucositis. [NIH]

Thymidine Kinase: An enzyme that catalyzes the conversion of ATP and thymidine to ADP and thymidine 5'-phosphate. Deoxyuridine can also act as an acceptor and dGTP as a donor. (From Enzyme Nomenclature, 1992) EC 2.7.1.21. [NIH]

Thymus: An organ that is part of the lymphatic system, in which T lymphocytes grow and multiply. The thymus is in the chest behind the breastbone. [NIH]

Tissue: A group or layer of cells that are alike in type and work together to perform a specific function. [NIH]

Tissue Culture: Maintaining or growing of tissue, organ primordia, or the whole or part of an organ in vitro so as to preserve its architecture and/or function (Dorland, 28th ed). Tissue culture includes both organ culture and cell culture. [NIH]

Tone: 1. The normal degree of vigour and tension; in muscle, the resistance to passive elongation or stretch; tonus. 2. A particular quality of sound or of voice. 3. To make permanent, or to change, the colour of silver stain by chemical treatment, usually with a

heavy metal. [EU]

Tonsil: A round-to-oval mass of lymphoid tissue embedded in the lateral wall of the pharynx situated on each side of the fauces, between the anterior and posterior pillars of the soft palate. [NIH]

Toxic: Having to do with poison or something harmful to the body. Toxic substances usually cause unwanted side effects. [NIH]

Toxicity: The quality of being poisonous, especially the degree of virulence of a toxic microbe or of a poison. [EU]

Toxicology: The science concerned with the detection, chemical composition, and pharmacologic action of toxic substances or poisons and the treatment and prevention of toxic manifestations. [NIH]

Toxin: A poison; frequently used to refer specifically to a protein produced by some higher plants, certain animals, and pathogenic bacteria, which is highly toxic for other living organisms. Such substances are differentiated from the simple chemical poisons and the vegetable alkaloids by their high molecular weight and antigenicity. [EU]

Toxoplasmosis: The acquired form of infection by Toxoplasma gondii in animals and man. [NIH]

Transcriptase: An enzyme which catalyses the synthesis of a complementary mRNA molecule from a DNA template in the presence of a mixture of the four ribonucleotides (ATP, UTP, GTP and CTP). [NIH]

Transcription Factors: Endogenous substances, usually proteins, which are effective in the initiation, stimulation, or termination of the genetic transcription process. [NIH]

Transduction: The transfer of genes from one cell to another by means of a viral (in the case of bacteria, a bacteriophage) vector or a vector which is similar to a virus particle (pseudovirion). [NIH]

Transfection: The uptake of naked or purified DNA into cells, usually eukaryotic. It is analogous to bacterial transformation. [NIH]

Transferases: Transferases are enzymes transferring a group, for example, the methyl group or a glycosyl group, from one compound (generally regarded as donor) to another compound (generally regarded as acceptor). The classification is based on the scheme "donor:acceptor group transferase". (Enzyme Nomenclature, 1992) EC 2. [NIH]

Transgenes: Genes that are introduced into an organism using gene transfer techniques. [NIH]

Translation: The process whereby the genetic information present in the linear sequence of ribonucleotides in mRNA is converted into a corresponding sequence of amino acids in a protein. It occurs on the ribosome and is unidirectional. [NIH]

Translational: The cleavage of signal sequence that directs the passage of the protein through a cell or organelle membrane. [NIH]

Translocation: The movement of material in solution inside the body of the plant. [NIH]

Transplantation: Transference of a tissue or organ, alive or dead, within an individual, between individuals of the same species, or between individuals of different species. [NIH]

Trauma: Any injury, wound, or shock, must frequently physical or structural shock, producing a disturbance. [NIH]

Treatment Failure: A measure of the quality of health care by assessment of unsuccessful results of management and procedures used in combating disease, in individual cases or series. [NIH]

Tropism: Directed movements and orientations found in plants, such as the turning of the sunflower to face the sun. [NIH]

Trypsin: A serine endopeptidase that is formed from trypsinogen in the pancreas. It is converted into its active form by enteropeptidase in the small intestine. It catalyzes hydrolysis of the carboxyl group of either arginine or lysine. EC 3.4.21.4. [NIH]

Tubulin: A microtubule subunit protein found in large quantities in mammalian brain. It has also been isolated from sperm flagella, cilia, and other sources. Structurally, the protein is a dimer with a molecular weight of approximately 120,000 and a sedimentation coefficient of 5.8S. It binds to colchicine, vincristine, and vinblastine. [NIH]

Tumor infiltrating lymphocytes: White blood cells that have left the bloodstream and migrated into a tumor. [NIH]

Tumor Lysis Syndrome: A syndrome resulting from cytotoxic therapy, occurring generally in aggressive, rapidly proliferating lymphoproliferative disorders. It is characterized by combinations of hyperuricemia, lactic acidosis, hyperkalemia, hyperphosphatemia and hypocalcemia. [NIH]

Tumor Necrosis Factor: Serum glycoprotein produced by activated macrophages and other mammalian mononuclear leukocytes which has necrotizing activity against tumor cell lines and increases ability to reject tumor transplants. It mimics the action of endotoxin but differs from it. It has a molecular weight of less than 70,000 kDa. [NIH]

Tumor suppressor gene: Genes in the body that can suppress or block the development of cancer. [NIH]

Tumorigenic: Chemical, viral, radioactive or other agent that causes cancer; carcinogenic. [NIH]

Tumour: 1. Swelling, one of the cardinal signs of inflammations; morbid enlargement. 2. A new growth of tissue in which the multiplication of cells is uncontrolled and progressive; called also neoplasm. [EU]

Tunica: A rather vague term to denote the lining coat of hollow organs, tubes, or cavities. [NIH]

Type 2 diabetes: Usually characterized by a gradual onset with minimal or no symptoms of metabolic disturbance and no requirement for exogenous insulin. The peak age of onset is 50 to 60 years. Obesity and possibly a genetic factor are usually present. [NIH]

Ubiquitin: A highly conserved 76 amino acid-protein found in all eukaryotic cells. [NIH]

Urate Oxidase: An enzyme that catalyzes the conversion of urate and unidentified products. It is a copper protein. The initial products decompose to form allantoin. EC 1.7.3.3. [NIH]

Urea: A compound (CO(NH2)2), formed in the liver from ammonia produced by the deamination of amino acids. It is the principal end product of protein catabolism and constitutes about one half of the total urinary solids. [NIH]

Urinary: Having to do with urine or the organs of the body that produce and get rid of urine. [NIH]

Urine: Fluid containing water and waste products. Urine is made by the kidneys, stored in the bladder, and leaves the body through the urethra. [NIH]

Vaccination: Administration of vaccines to stimulate the host's immune response. This includes any preparation intended for active immunological prophylaxis. [NIH]

Vaccine: A substance or group of substances meant to cause the immune system to respond to a tumor or to microorganisms, such as bacteria or viruses. [NIH]

Varicella: Chicken pox. [EU]

Vascular: Pertaining to blood vessels or indicative of a copious blood supply. [EU]

VE: The total volume of gas either inspired or expired in one minute. [NIH]

Vector: Plasmid or other self-replicating DNA molecule that transfers DNA between cells in nature or in recombinant DNA technology. [NIH]

Vein: Vessel-carrying blood from various parts of the body to the heart. [NIH]

Venous: Of or pertaining to the veins. [EU]

Ventricle: One of the two pumping chambers of the heart. The right ventricle receives oxygen-poor blood from the right atrium and pumps it to the lungs through the pulmonary artery. The left ventricle receives oxygen-rich blood from the left atrium and pumps it to the body through the aorta. [NIH]

Ventricular: Pertaining to a ventricle. [EU]

Vesicular: 1. Composed of or relating to small, saclike bodies. 2. Pertaining to or made up of vesicles on the skin. [EU]

Veterinary Medicine: The medical science concerned with the prevention, diagnosis, and treatment of diseases in animals. [NIH]

Vimentin: An intermediate filament protein found in most differentiating cells, in cells grown in tissue culture, and in certain fully differentiated cells. Its insolubility suggests that it serves a structural function in the cytoplasm. MW 52,000. [NIH]

Vinblastine: An anticancer drug that belongs to the family of plant drugs called vinca alkaloids. It is a mitotic inhibitor. [NIH]

Vinca Alkaloids: A class of alkaloids from the genus of apocyanaceous woody herbs including periwinkles. They are some of the most useful antineoplastic agents. [NIH]

Vincristine: An anticancer drug that belongs to the family of plant drugs called vinca alkaloids. [NIH]

Viral: Pertaining to, caused by, or of the nature of virus. [EU]

Viral Load: The quantity of measurable virus in the blood. Change in viral load, measured in plasma, is used as a surrogate marker in HIV disease progression. [NIH]

Viral Proteins: Proteins found in any species of virus. [NIH]

Viral vector: A type of virus used in cancer therapy. The virus is changed in the laboratory and cannot cause disease. Viral vectors produce tumor antigens (proteins found on a tumor cell) and can stimulate an antitumor immune response in the body. Viral vectors may also be used to carry genes that can change cancer cells back to normal cells. [NIH]

Virulence: The degree of pathogenicity within a group or species of microorganisms or viruses as indicated by case fatality rates and/or the ability of the organism to invade the tissues of the host. [NIH]

Virus: Submicroscopic organism that causes infectious disease. In cancer therapy, some viruses may be made into vaccines that help the body build an immune response to, and kill, tumor cells. [NIH]

Virus Replication: The process of intracellular viral multiplication, consisting of the synthesis of proteins, nucleic acids, and sometimes lipids, and their assembly into a new infectious particle. [NIH]

Viscera: Any of the large interior organs in any one of the three great cavities of the body, especially in the abdomen. [NIH]

Vitro: Descriptive of an event or enzyme reaction under experimental investigation occurring outside a living organism. Parts of an organism or microorganism are used

together with artificial substrates and/or conditions. [NIH]

Vivo: Outside of or removed from the body of a living organism. [NIH]

White blood cell: A type of cell in the immune system that helps the body fight infection and disease. White blood cells include lymphocytes, granulocytes, macrophages, and others. [NIH]

Xenograft: The cells of one species transplanted to another species. [NIH]

X-ray: High-energy radiation used in low doses to diagnose diseases and in high doses to treat cancer. [NIH]

X-ray therapy: The use of high-energy radiation from x-rays to kill cancer cells and shrink tumors. Radiation may come from a machine outside the body (external-beam radiation therapy) or from materials called radioisotopes. Radioisotopes produce radiation and can be placed in or near the tumor or in the area near cancer cells. This type of radiation treatment is called internal radiation therapy, implant radiation, interstitial radiation, or brachytherapy. Systemic radiation therapy uses a radioactive substance, such as a radiolabeled monoclonal antibody, that circulates throughout the body. X-ray therapy is also called radiation therapy, radiotherapy, and irradiation. [NIH]

Yeasts: A general term for single-celled rounded fungi that reproduce by budding. Brewers' and bakers' yeasts are Saccharomyces cerevisiae; therapeutic dried yeast is dried yeast. [NIH]

Zoster: A virus infection of the Gasserian ganglion and its nerve branches, characterized by discrete areas of vesiculation of the epithelium of the forehead, the nose, the eyelids, and the cornea together with subepithelial infiltration. [NIH]

Zymogen: Inactive form of an enzyme which can then be converted to the active form, usually by excision of a polypeptide, e. g. trypsinogen is the zymogen of trypsin. [NIH]

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