Magill’s Medical Guide

Fourth Revised Edition

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Covers diseases, disorders, treatments, procedures, specialties, anatomy, biology, and issues in an A-Z format, with sidebars addressing recent developments in medicine and concise information boxes for all diseases and disorders.

The publication of the fourth edition of Salem Press’ bestselling Magill’s Medical Guide is unlike any reference work published before it. This edition grows to five volumes and 3,206 pages. But even better, the purchase of the printed set entitles a school or library to three years of free online access to the Medical Guide’s content.

SCOPE AND COVERAGE

Every entry in Magill’s Medical Guide has been evaluated by our panel of medical consultants and then updated by experts or re-edited to ensure currency and accuracy. The Guide includes over 1,000 essays, ranging in length from 500 to 3,552 words each. 58 topics are new and 37 essays replace those from the previous edition. There are over 400 photographs and drawings plus 644 sidebars and tables. The new edition introduces particularly current topics such as facial transplantation and avian influenza (bird flu). Plus, the Guide includes 44 "In the News" sidebars that evaluate recent media stories about ongoing research and experimental treatments. These boxes both highlight the latest information and provide readers with a critical view of popular reports.

SPECIAL FEATURES

The special features in this set include: "Complete List of Contents," "Entries by Specialties and Related Fields," and "Entries by Anatomy or System Affected." There is also a "Glossary" of medical terms, a list of almost 900 "Diseases and Other Medical Conditions," an appendix detailing the training and duties of various "Health Care Providers," a list of "Medical Journals," a "General Bibliography" and "Web Site Directory," a helpful "Resources" list, the new "Symptoms and Warning Signs" and "Pharmaceutical List," and a comprehensive subject index. For each disease and disorder, a concise information box lists causes, symptoms, duration, and treatments, acting as a quick reference tool for the reader.

At a Glance - PRINT

- 5 volumes, 3,206 pages
- 1,017 Articles
- 644 Sidebars and Tables
- 400+ Photographs and Illustrations
- 44 "In the News" Sidebars
- Glossary
- Symptoms and Warning Signs
- Pharmaceutical List
- 900 "Diseases and Other Medical Conditions" list
- "Health Care Providers" list
- List of "Medical Journals"
- General Bibliography
- Web Site Directory
- Subject Index
- "Resources" list of Organizations

At a Glance - ONLINE

- Free Access with Print Purchase
- Full content of Medical Guide
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- Index with Direct Links
ENVIRONMENTAL DISEASES
DISEASE/disorder
ANATOMY OR SYSTEM AFFECTED: All
SPECIALTIES AND RELATED FIELDS: All
DEFINITION: Sicknesses caused or exacerbated by human exposure to physical, chemical, biological, or social environmental conditions, the duration and in-
tensity of the exposure typically affecting the manifestation of symptoms and fatality-case ratios. Acute environmental diseases may result in rapid decline of health status and warrant emergency response, while chronic conditions often result from long-term exposures to low levels of environmental risk factors.

**Key Terms:**

- **Acute:** referring to exposure to hazardous environmental agents or conditions that occur once or over a short period of time (typically fourteen days or less);
- **Chronic:** referring to exposure to environmental risk factors or agents occurring over a long period of time, typically more than one year; symptoms of environmental diseases that take a long time to appear after first contact with the causative agent.
- **Dose-response:** the relationship between the dose (a quantitative measurement of exposure usually expressed in terms of concentration and duration) and the quantitative expression of change to the status of human health and well-being resulting in disease.
- **Environmental Epidemiology:** the systematic study of the distribution and determinants of environmental diseases in a population.
- **Environmental Infection:** human exposure to infectious agents of diseases (including bacteria, fungi, parasites, and viruses) through contact with environmental media such as contaminated water, air, food, and soil.
- **Environmental Radiation:** human exposure to electromagnetic radiation at doses and durations that can produce adverse impacts on human health.
- **Environmental Toxicity:** human exposure to chemical or biochemical substances at doses that produce harmful modification to the body’s physiological mechanisms, leading to diseases.
- **Exposure Assessment:** a systematic process of discovering the pathway through which humans are exposed to specific environmental agents and risk factors, and of ascertaining the quantity and duration of that exposure.

**Causes and Symptoms**

The modern word “miasma” comes from the Greek *miasma* or *miasain*, meaning “pollution” or “to pollute.” Before scientific theories of disease became entrenched in medical practice, miasma was used to connote bad environments to which human exposure led to various diseases. Even today, one of the most devastating human diseases, malaria, draws its name from references to “bad air.” There is clearly a rich historical record of human recognition of the intimate connection between environmental quality and diseases. It is now known that serious human diseases are caused by numerous chemical, physical, and biological agents (risk factors) that occur naturally or as a result of human actions that modify the environment. In fact, the more that is learned about disease etiology, the more the complex interplay between environmental conditions and root causes of diseases within the body are recognized. Furthermore, some people are more sensitive to environmental risk factors because of their age, gender, occupation, culture, or genetic characteristics.

Environmental diseases are those illnesses for which cause and effect can be reasonably associated through epidemiological studies, preferably verified through laboratory experiments. Therefore, the recognition of environmental diseases draws upon two traditional postulates regarding causation in the study of human diseases, one ascribed to Robert Koch (1843-1910) and the other ascribed to Austin Bradford Hill (1897-1991). The more important set of guidelines for environmental diseases is generally known in epidemiology as Hill’s criteria of causation, based on his landmark 1965 publication entitled “The Environment and Disease: Association or Causation?” Hill warned in the article that cause-effect decisions should not be based on a set of rules. Instead, he supported the view that cost-benefit analysis is essential for policy decisions on controlling environmental quality in order to avoid diseases. It is arguable that Hill’s treatise initiated current trends characterized by the precautionary principle in environmental health science. Nevertheless, Hill’s nine viewpoints for exploring the relationship between environment and disease are worth emphasizing. They are precedence, correlation, dose-response, consistency, plausibility, alternatives, empiricism, specificity, and coherence.

According to the precedence viewpoint, exposure must always precede the outcome in every case of the environmental disease. One of the most famous examples here is the classic epidemiological study of John Snow (1813-1858) on the spread of cholera and its association with exposure to contaminated water in the densely populated city of London.

According to the correlation viewpoint, a strong association or correlation should exist between the exposure and the incidence of the environmental disease. The clustering of diseases within neighborhoods or
among workers at a specific occupation is frequently the beginning of investigations into environmental diseases. Clusters can provide strong evidence of correlations. Bernardino Ramazzini (1633-1714), considered by many to be one of the founders of the discipline of occupational and environmental health sciences, published his treatise De Morbis Artificum in 1700 following critical observations regarding the correlation between environmental exposures of and diseases in workers.

According to the dose-response viewpoint, the relationship between exposure and the severity of environmental disease should be characterized by a dose-response relationship, in which an increase in the intensity and/or duration of exposure produces a more severe disease outcome. "The dose makes the poison" is one of the central tenets of environmental toxicology. This phrase is attributed to Paracelsus (1493-1541). This tenet has proven difficult to interpret for formulating health policy in the case of environmental diseases because the variation in human genetics and physiology means that, in many situations, a single threshold of toxicity cannot be established as safe for every person. Exposure to ionizing radiation is an example of a situation in which it is difficult to establish dose-response relationships that are useful for setting uniformly applicable preventive health policy.

According to the consistency viewpoint, there should be consistent findings in different populations, across different studies, and at different times regarding the association between exposure and environmental disease. This means that the relationship should be reproducible. For example, exposure of people to mercury across civilizations, occupations, and age groups has been consistently associated with certain health effects that helped recognize the special hazards posed by this toxic metal. Mercury was used in various manufacturing processes for several centuries, and where precautions are not taken to prevent human exposure, disease invariably results.

Consistency should cut across not only generations but also occupations and different doses of exposure. For example, "mad hatter's" disease was associated with the use of mercury in the production of fur felt, in which mercurous nitrate was used to add texture to smooth fibers such as rabbit fur to facilitate matting (the process is called carrottin because of the resulting orange color). More recently, the exposure of pregnant women to fish contaminated with methyl mercury from industrial sources in Japan produced developmental diseases in fetuses. The societal repercussions of the so-called Minamata disease are still not completely settled after more than forty years. Mercury is now widely recognized as a cumulative toxicant with systemic effects and organ damage, with symptoms including trembling, dental problems, ataxia, depression, and anxiety.

According to the plausibility viewpoint, compelling evidence of "biological plausibility" should exist that a physiological pathway leads from exposure to a specific environmental risk factor to the development of a specific environmental disease. This does not exclude the possibility of multiple causes, some acquired through environmental exposures and others through genetic processes. For example, lead poisoning has been recognized since the 1950's as a pervasive and devastating environmental disease. The symptoms of lead poisoning vary, from specific organ effects such as kidney disease to systemic effects such as anemia and to cognitive effects such as intelligence quotient (IQ) deficiency. How a single environmental toxicant can produce such wide-ranging diseases was a puzzle until the molecular mechanisms underpinning lead poisoning and the pharmacokinetic distribution of lead in the human body was understood. Lead is temporarily stored in the blood, where it binds to a key enzyme, aminolevulinate dehydratase, which participates in the synthesis of heme. The by-products of that reaction produce anemia and organ effects, including kidney and brain diseases. Long-term storage of lead in the body occurs in bony tissue, where other effects are possible. These biological understandings have helped activists and scientists agitate for environmental policy to reduce lead exposure worldwide.

According to the alternatives viewpoint, alternative explanations for the development of diseases should be considered alongside the plausible environmental causes. These alternative explanations should be ruled out before conclusions are reached about causal relationship between environmental exposures and disease. For example, the typically low doses to which populations are exposed to pesticides, and the long time period between exposure and the typical chronic disease outcomes such as cancers and neurodegenerative disorders, makes it difficult to reconstruct the disease pathways and pinpoint causative agents. This is where it is important to consider all alternatives and to eliminate them before compelling arguments can be made about the effects of pesticide toxicity. Sometimes observing wildlife response to environmental risk fac-
IN THE NEWS:
IRAQ WAR INCREASES DISEASE RISKS

Throughout the history of warfare, diseases have often caused more casualties among both soldiers and civilians than weaponry itself. The conflict in Iraq is no exception. A rare type of lung infection, acute eosinophilic pneumonia (AEP), is occurring at a higher rate among U.S. soldiers in Iraq than in any other segment of the population. The illness, characterized by fever, serious lung impairment, and eventually respiratory failure, has killed several soldiers since 2003. Of the individuals who contracted this pneumonia, all reported exposure to fine sand and dust particles, a common hazard in the desert environments of the Middle East. Administration of corticosteroids proved life-saving for most patients, but many who have recovered complain of residual lung problems.

Veterans of Operation Iraqi Freedom are not alone in fighting disabling illnesses. Brain cancer, amyotrophic lateral sclerosis (ALS), fibromyalgia, and multiple sclerosis are among the serious ailments plaguing the earlier Gulf War veterans from 1991. The demolition of weapons dumps in Iraq in March, 1991, released the deadly nerve agents sarin and cyclosarin. Many Gulf War veterans claim that this incident is to blame for the diseases from which they now suffer. A Department of Defense-sponsored study conducted by the Institute of Medicine of the National Academy of Sciences, published in *American Journal of Public Health* in August, 2005, focused on the nerve gas release event at Al Khamisiyah and subsequent increase in neurological illnesses in those exposed to the chemical contamination which followed.

Inflamed joints, heat and chemical sensitivities, severe headaches, hair loss, and recurrent skin rashes are just a few of the symptoms endured by affected troops who have returned from both Iraq conflicts. Nearly 200,000 Gulf War veterans are currently receiving disability payments from the Veterans Administration, and many of those payments are for battle-related illness rather than for injury.

—Lenela Glass-Godwin, M.WS.

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sure to prion protein remains mysterious, and some environmental causes have been proposed, including exposure to toxic metal ions. Another example is the current concern with the introduction of nanoparticles into commercial products, with concomitant environmental dissemination. Although much has been learned from an understanding of the human health effects of respirable particulate matter, researchers should be sufficiently open-minded to the possibility that nanoparticles will behave differently in the environment and in the human body.

Hill’s nine viewpoints were presented in the context of pitfalls associated with overreliance on statistical tests of “significance” as a justification to base health policy on epidemiological observations. Hill’s viewpoints have been debated extensively, and it is worth noting the following caveats presented in the 2004 article “The Missed Lessons of Sir Austin Bradford Hill,” by Carl V. Phillips and Karen J. Goodman: Statistical significance should not be mistaken for evidence of substantial association, association does not prove causation, precision should not be mistaken for validity, evidence that a causal relationship exists is not sufficient to suggest that action should be taken, and uncertainty about causation or association is not sufficient to suggest that action should not be taken.

The second set of guidelines regarding causality derives from what is generally known as Koch’s postulates, but it is perhaps only useful for precautionary approaches to proactive assessment of potential health impacts of new agents about to be introduced into the environment. This approach complements the epidemiology-based inferences described by Hill, but further refinement is warranted to deal with complicated issues such as interactions between multiple environmental agents, which could have additive, neutral, or canceling effects. The question of dose is also difficult to subject to simple conclusions because of phenomena such as hormesis, in which small doses may show beneficial effects.

For environmental diseases, a modified version of Koch’s postulates can be expressed as follows. First, exposure to an environmental agent must be demonstrable in all organisms suffering from the disease, but not in healthy organisms (assuming predisposition factors). Second, the identity, concentrations in different environmental and physiological compartments, and transformation pathways of the agent must be known as much as possible. Third, the agent should cause disease when introduced into healthy organisms. Fourth, biomarkers showing modification of the physiological target affected by the environmental agent must be observable in experimentally exposed organisms.

**TREATMENT AND THERAPY**

The symptoms of environmental diseases vary widely, and physiological, anatomical, and behavioral characteristics can succumb to the effects of environmental agents. In evaluating treatment and therapy, it is useful to consider two categories of symptoms. Acute symptoms are exhibited in response to human exposure to high doses of toxic agents within a short period of time. Essentially, the body is overwhelmed, and emergency therapy is necessary to avoid death or permanent disability. For toxic air contaminants, respiratory distress is a common symptom, and mortality can occur rapidly. Conversely, chronic symptoms of human exposures to low levels of environmental (particularly air) pollutants are difficult to diagnose, as in the case of cancers attributable to secondhand tobacco smoke or ambient exposure to respirable particulate matter. Similarly, exposure of the skin to rapidly absorbed toxins can produce rapid mortality, but the development of skin cancer due to ultraviolet (UV) light exposure may take decades to manifest. Ingestion of contaminated liquids or food may take minutes to provoke distress and vomiting, whereas it may take years for chronic symptoms to manifest in cases of carcinogenic water pollutants.

Treatment and therapy of environmental diseases requires accurate diagnosis of the causative agent. The first line of response is to limit exposure through flushing the body with clean air or liquids. Chelation therapy can be used to reduce the body burden of certain toxic metals. Curative measures follow the established procedures developed for specific organs. For example, chemotherapy, radiotherapy, and surgery are used to treat cancers regardless of the involvement of known environmental factors in their etiology. Skin diseases such as chloracne associated with exposure to chlorinated aromatic hydrocarbon pollutants, including polychlorinated biphenyls (PCBs), are managed to reduce the severity of lesions and enhance natural healing processes. Cognitive deficits associated with exposure to metals and other environmental pollutants are believed to be reversible as long further exposures are avoided. Finally, environmental diseases associated with infectious agents such as bacteria can be controlled through a combination of source disinfection and antibiotic therapy.
PERSPECTIVE AND PROSPECTS
There has been a resurgence of interest in environmental diseases because of societal changes at regional and international levels. Industrialization demands the use of thousands of potentially hazardous chemicals that ultimately end up polluting human environments and remain an important source of causative agents for environmental diseases. Recent threats associated with global environmental change, bioterrorism, and chemical warfare have all contributed to the need for rapid detection of hazardous environmental agents and tougher laws to protect air, water, soil, and food resources. Prevention is still the crucial solution to reducing the human burden of environmental diseases worldwide.

On June 16, 2006, the World Health Organization (WHO) issued a landmark report estimating that environmental risk factors play a role in more than 80 percent of diseases regularly reported by WHO across fourteen regions globally. The environment has an impact on human health through exposures to physical, chemical, and biological risk factors and through changes in human behavior in response to environmental change at local and global levels. Globally, nearly 25 percent of all deaths and of the total disease burden (measured in disability-adjusted life years, or DALYs) can be attributed to environmental quality. The situation is more dire for children, with environmental risk factors accounting for more than 33 percent of the disease burden. These discoveries have important implications for national and international health policy, because many of the implicated environmental risk factors can be modified by established interventions. The lack of understanding on how to deploy these interventions globally has inspired the involvement of well-funded organizations and institutions in environmental health issues.

In the United States, the National Institute of Environmental Health Sciences (NIEHS), a subsidiary of the National Institutes of Health (NIH), is responsible for studying environmental diseases and guiding policy on how to protect vulnerable populations. A comprehensive five-year strategic plan developed by the NIEHS in 2006 outlined seven specific goals aimed at curtailing environmental exposures and reducing societal burden of environmental diseases.

The first goal is to expand the role of clinical research in environmental health sciences by emphasizing the use of environmental exposures to understand and better characterize complex diseases. This includes the development of research models for human diseases based on the coordinated knowledge of environmental sciences and human biology.

The second goal is to use environmental toxins to gaining more insight into the basic mechanisms in human biology. This goal involves tapping into the rapidly expanding knowledge of the biochemical mechanisms of disease progression and the influence of genetic factors in the path from environmental exposures to disease symptoms.

The third goal is to build an integrative multidisciplinary understanding of complex environmental systems and the various ways in which human diseases are manifested. The fourth goal is to improve the quality of community-based environmental health research. Much can be learned from studying populations that are exposed to high concentrations of environmental agents believed to cause human disease. The hope is to better understand disease clusters in communities and to link the understanding of regional prevalence of environmental diseases to global environmental health concerns.

The fifth goal is to improve the understanding and use of sensitive markers of exposure, susceptibility, and effects of environmental agents. There is also an urgent need to develop technologies for measuring exposures more accurately in order to link exposures more tightly to assessments of toxicity and other physiological impacts.

The sixth goal is to engage the broader biomedical community in research on environmental diseases and to support a pipeline for encouraging new researchers to enter the discipline of environmental health.

The seventh goal is to encourage pathways by which the results of research can inform policy quickly in order to protect vulnerable populations. This requires fostering cooperation with other agencies and organizations such as the U.S. Environmental Protection Agency (EPA), the Food and Drug Administration (FDA), the U.S. Department of Agriculture, and the Occupational Safety and Health Administration (OSHA). In addition, there must be straightforward communication paths between manufacturing industries where environmental agents of disease originate and the community of environmental health physicians, researchers, and regulatory agencies to ensure successful implementation of these laudable goals.

—Oladele A. Ogunsain, Ph.D., M.P.H.

See also Allergies; Asbestos exposure; Aspergillosis; Asthma; Bronchitis; Cancer; Carcinogens; Carpal tunnel syndrome; Chronic obstructive pulmonary
disease (COPD); *E. coli* infection; Emphysema; Environmental health; Epidemiology; Food poisoning; Gulf War syndrome; Lead poisoning; Lung cancer; Melanoma; Mercury poisoning; Multiple chemical sensitivity syndrome; Occupational health; Poisoning; Pulmonary diseases; Radiation sickness; Respiration; Skin cancer; Skin lesion removal; Teratogens; Toxicology; Tularemia.

**FOR FURTHER INFORMATION:**


National Institute of Environmental Health Sciences (NIEHS). *New Frontier in Environmental Sciences and Human Health: The 2006-2011 NIEHS Strategic Plan*. http://www.niehs.nih.gov/external/plan2006. The comprehensive five-year strategic plan developed by the NIEHS.


**ENVIRONMENTAL HEALTH**

**SPECIALTY**

**ANATOMY OR SYSTEM AFFECTED:** All

**SPECIALTIES AND RELATED FIELDS:** Epidemiology, occupational health, preventive medicine, psychology, public health, toxicology

**DEFINITION:** The control of all factors in the physical environment that exercise, or may exercise, a deleterious effect on human physical development, health, and survival and correcting and preventing those effects from adversely affecting future generations. The study of the influence of environment on health and disease.

**KEY TERMS:**
community: a group of people living in the same locality
hygiene: the science of health and the prevention of disease
pollutant: a noxious substance that contaminates the environment
remediation: correcting an evil, fault, or error
sanitation: the application of measures designed to protect public health

**SCIENCE AND PROFESSION**

The environment is the sum of all external influences and conditions affecting the life and development of an organism. For humans, a healthy environment means that the surroundings in which humans live, work, and play meet some predetermined quality standard. The field of environmental health encompasses biological, chemical, physical, and psychosocial factors in the environment. This is the air that humans breathe, the water that they drink, the food that they consume, and the shelter that they inhabit. The definition also includes the identification of pollutants, waste materials, and other environmental factors that adversely affect life and health. The study of environmental health investigates how human health and disease are influenced by the environment. It encompasses the fields of environmental engineering and sanitation, public health engineering, and sanitary engineering. The majority of professionals working in the field of environmental health are trained as civil engineers, environmental engineers, geologists, toxicologists, or preventive medicine specialists. Many are also qualified in subspecialties such as hydrogeology, epidemiology, public sanitation, and occupational health.

Environmental health deals with the control of factors in the physical environment that cause (or may