



MOUNT SINAI  
SCHOOL OF  
MEDICINE

# CENTER FOR CHILDREN'S HEALTH AND THE ENVIRONMENT

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MOUNT SINAI SCHOOL OF MEDICINE

Testimony of  
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Before the  
Committee on Environment and Public Works  
United States Senate

In a Field Hearing at  
Adelphi University, Garden City, New York

June 11, 2001

Chairman Reid, Senator Clinton, and Members of the New York Congressional Delegation,

I am pleased to appear before you today to discuss rising rates of cancer and other chronic diseases in the American population and the linkages between cancer and the environment.

I would like also to discuss with you a blueprint for substantially reducing cancer rates in this nation. The centerpiece of this plan will be the construction of a strong national capacity in public health and preventive medicine that will enable us to locate, track, understand and prevent the environmental causes of cancer.<sup>(1)</sup>

My name is Philip J. Landrigan, M.D. I am Chair of the Department of Community and Preventive Medicine and Professor of Pediatrics at the Mount Sinai School of Medicine in New York City. I direct the Center for Children's Health and the Environment at Mount Sinai, a policy research center supported by The Pew Charitable Trusts. I am a pediatrician and epidemiologist. A copy of my curriculum vitae is attached.

**Rising Rates of Chronic Disease in the American Population.** Today, the leading causes of illness and death in the United States are chronic diseases and injuries.<sup>(2)</sup> Rates of asthma have more than doubled. Incidence of certain birth defects of the reproductive organs such as hypospadias have doubled. Neurodevelopmental disorders such as dyslexia, attention deficit/hyperactivity disorder (ADHD) and autism are highly prevalent and cause untold misery to children and their families. Chronic diseases of the brain and central nervous system such as Parkinson's Disease have increased in frequency.

Cancer is a particular problem. Cancer will kill approximately 550,000 people in the United States this year, according to the American Cancer Society. Cancer is the second leading cause of death, exceeded only by heart disease. It is the second leading cause of lost years of potential life.<sup>(3)</sup>

Breast cancer is a major problem in New York and across the nation. An estimated 182,000 cases of breast cancer are expected to be diagnosed this year among American women, and about

1,400 new cases of breast cancer are expected to be diagnosed in men.<sup>(3)</sup> Rates of breast cancer have risen in the United States, and the cumulative increase in incidence since the early 1970's has been more than 40%.

Pediatric cancer is another major problem. An estimated 12,400 children and young people will be diagnosed with cancer in the United States in the year 2001. Cancer is the third most common cause of death in American children, exceeded only by unintentional injuries and homicide. Thus it is the leading cause of death from disease in our young people. The two most common forms of childhood malignancy are leukemia and brain cancer, and together these two diseases account for about two-thirds of pediatric cancer.<sup>(4)</sup> Although death rates for childhood cancer are down, thanks to early detection and vastly improved treatment, the reported incidence, i.e., the number of new cases of cancer per million children has increased over the past two decades (please see attached graphs).<sup>(4)</sup>

For acute lymphoblastic leukemia (ALL), the most common pediatric malignancy, incidence increased from 23.1 cases per million children in the early 1970s to a peak of 28.2 per million in the 1980s, and then declined somewhat to a level of 26.8 per million in 1996. This represents an overall increase since the early 1970s of about 12%, an increase that is statistically significant.<sup>(4)</sup>

For primary brain cancer (glioma), a sharp and statistically significant increase in incidence has been noted from 23 cases per million children in the early 1970s to 29.0 per million in the late 1990s. This represents an overall increase in incidence over the past three decades of nearly 30%, an increase that is statistically quite significant.<sup>(3)</sup>

For testicular cancer, incidence in young men 15-30 years of age has increased over the past thirty years by 68%. This increase occurred entirely in white men, and was not seen in black men. It is statistically highly significant.<sup>(5)</sup>

The causes of these increases in cancer are incompletely understood. Some have argued that better diagnostic detection and changing definitions of cancer may account for a major fraction of the increase.<sup>(6)</sup> I would agree that new diagnostic technologies have made some contribution

to reported increases in cancer incidence, but I cannot agree that it is the whole story. I would point out that childhood cancer is not a subtle disease. Sadly, it is a devastating and extremely serious illness. It makes children terribly ill, and it brings them to the hospital. Thus it seems unlikely to me that large numbers of children with cancer would have escaped medical detection only twenty-five years ago, at a time when many doctors of my generation were already practicing pediatrics.

A further argument against the notion that better diagnostic detection accounts for the entire reported increase in childhood cancer is that any increase due to better diagnosis would have produced only a temporary rise in reported incidence at the time of introduction of the new technology, reflecting diagnosis at an earlier stage of illness. That temporary increase would then be expected to be followed by a return to baseline. In fact, however, no such return to baseline incidence of childhood brain cancer has occurred in the United States over the past 30 years. In fact, the incidence rate for childhood brain cancer has continued to rise inexorably, and this upward trend is seen in both boys and girls in all regions of the United States.<sup>(7)</sup> These facts argue that most of the reported rise in incidence of childhood cancer is a real increase.

It is highly likely that environmental toxins in air, food, dust, soil and drinking water have contributed to increasing rates of cancer in Americans of all ages, including our children. The known and suspected causes of childhood cancer include benzene, other solvents, radiation, arsenic, parental smoking, certain pesticides and certain chemicals in the environment that have the potential to disrupt the function of the endocrine system. Maternal consumption during pregnancy of cured meats containing nitrites, such as sausage and bacon has been shown to increase risk of childhood brain cancer. There are also protective factors. Maternal consumption of folic acid during pregnancy, and the practice of nursing an infant appear to be protective factors that can reduce incidence of childhood cancer. Those facts are signs of hope.

**Cancer and the Environment – An Historical Perspective.** Considerable progress toward cancer control has stemmed from the recognition that chemical agents in the environment can cause cancer.<sup>(8)</sup> In 1775, Sir Percivall Pott, a British surgeon, reported for the first time an association between childhood cancer and an environmental agent.<sup>(9)</sup> Pott noted that the

“climbing boys of London”, teenage lads employed as chimney sweeps, experienced a devastating incidence of cancer of the scrotum. He correctly attributed the development of those tumors to occupational exposure to soot. In 1895, Rehn noted a high frequency of cancer of the urinary bladder among workers in the aniline dye industry.<sup>(10)</sup> He attributed the causation of those tumors to aromatic amines. More recently etiologic associations have been recognized between benzene and leukemia,<sup>(11)</sup> asbestos and lung cancer,<sup>(12)</sup> bischloromethylether and lung cancer,<sup>(13)</sup> vinyl chloride monomer and angiosarcoma of the liver,<sup>(14)</sup> tobacco and lung cancer,<sup>(15)</sup> and chewing tobacco and cancer of the mouth.<sup>(16)</sup>

Toxicologic studies stimulated by those clinical and epidemiologic observations have led to fundamental advances in the understanding of cancer biology. Benzo(a)pyrene, a polynuclear aromatic hydrocarbon compound found in soot, has been found to induce skin cancer in experimental animals.<sup>(17)</sup> That finding provides a molecular basis for Pott’s observations of the link between soot and scrotal cancer. Likewise  $\beta$ -naphthylamine, a chemical found in aniline dye manufacture, has been shown to cause cancer of the bladder in experimental animals, thus providing an explanation for the observation of Rehn.<sup>(18)</sup> Chemical carcinogens found in tobacco and tobacco smoke provide a biological basis for the observation that cigarette smoking causes lung cancer and that chewing tobacco causes cancer of the mouth and oropharynx.

Common themes that run through these tales of discovery are an (1) the importance of tracking data on cancer incidence, (2) an openness to the possibility that environmental factors can cause cancer and (3) a willingness to pursue clinical and epidemiologic observations to discover the biological mechanisms by which environmental agents cause malignancy.

The recognition of environmental carcinogenesis has had a profound influence on our understanding of human cancer. No longer must cancer be regarded as an inescapable consequence of aging or the result of unexplainable “natural forces.” Quite the contrary. It is now realized that chemical carcinogenesis is not exceptional and that well over half of human cancers - - perhaps as many as 80-90% worldwide - - are caused by environmental exposures.<sup>(19)</sup> I should note that in this context “environmental factors” include not only exposures to industrial

chemicals and pollutants but also exposures to such factors as diet, alcohol, tobacco, drugs, radiation and sexual behavior.

The concept that the environment is responsible for a great majority of human cancer received strong collaboration in a landmark study published recently from Sweden.<sup>(20)</sup> This study which examined patterns of cancer in 44,788 pairs of twins found sharp discrepancies in cancer incidence even between identical twins. These differences, even in persons of identical genetic composition, indicate that environment plays a major role in the causation of malignancy.

The most hopeful implication of the discovery of that many thousands of cancer cases are caused by exposures in the environment is that a very high proportion of all human cancer ought to be preventable. Prevention can be accomplished by reducing exposures to environmental carcinogens.<sup>(8)</sup>

**Chemical Exposures in Today's World.** Americans today face environmental hazards that were neither known nor suspected a few decades ago. Americans today are at risk of exposure to over 85,000 synthetic chemicals, most of which have been invented since World War II. Americans are most likely to be exposed to the 28,000 high-production-volume (HPV) chemicals that the U.S. Environmental Protection Agency estimates are produced in quantities of over one million pounds per year.<sup>(21)</sup> These chemicals are the most widely dispersed in foods, household products, pesticides, air, food and drinking water. The National Academy of Sciences has found that children are the group within the American population most vulnerable to these chemical hazards.<sup>(22)</sup>

No basic toxicity information is publicly available for 43% of the high-production-volume chemicals according to the EPA. And although children are now recognized to be especially vulnerable to chemicals in the environment, only 7% of HPV chemicals have been examined for their potential toxicity to children or to human development.<sup>(21)</sup>

The percentage of cancer in Americans that is caused by toxic chemicals in the environment is not known. We do, however, know that many chemicals are proven human carcinogens, that

many more are suspected human carcinogens on the basis of animal testing, and that most chemicals have never been tested.

**A Blueprint for Cancer Prevention in the United States.** The following are elements of a comprehensive plan for the prevention of environmental cancer in the United States.

Disease and exposure tracking. It will be essential to continue to provide support to the Centers for Disease Control and Prevention (CDC) and to the National Cancer Institute (NCI) to enable these agencies to monitor the number of cases of cancer and other chronic diseases that occur each year among Americans of all ages and in every part of the country.<sup>(1)</sup> The tracking of cancer, asthma, birth defects and other chronic diseases has lagged historically behind the tracking of infectious diseases such as measles and smallpox. Now, however, that the chronic diseases have become the major causes of morbidity and mortality in the United States, we must remedy this situation and arm ourselves with accurate information on the temporal and geographic distribution of cancer and other chronic diseases. Such information is essential for targeting prevention.

Also it will be essential to continue to provide support to the CDC to enable CDC to continue each year to monitor the levels of chemicals in the blood of Americans and to make this information available to the public. The combination of information on chemical exposure with data on cancer incidence will undoubtedly spark research that will identify specific preventable environmental causes of cancer and other chronic diseases.

A classic example of the importance of disease tracking to cancer prevention is provided by the story of oral cancer among women in the American south. In the early 1970's the National Cancer Institute published an Atlas of Cancer Mortality by County in the United States. Examination of the maps in this atlas revealed a strikingly high incidence of oral cancer among women across the southeastern United States from Virginia to Texas. The cause of that increase was initially not known. However, publication of the maps stimulated extensive research, and one of those studies was an epidemiologic investigation undertaken by Dr. Debra Winn. This classic study found an extremely strong association between oral cancer and the use of chewing

tobacco.<sup>(16)</sup> Once this association had been discovered, programs of prevention were put in place. This represents a textbook example of how disease tracking can lead to discovery of the factors responsible for disease and then to prevention.

Premarket testing of the toxic and carcinogenic potential all new chemical compounds is a most effective approach to the prevention of environmental disease. Unfortunately, premarket testing has often not been undertaken. A 1984 analysis by the National Research Council showed that most chemical compounds have never been tested for their carcinogenic potential.<sup>(23)</sup> That unfortunate figure has not improved appreciably in the intervening years, and the number of new chemical substances released into the environment has increased substantially during that time.

In addition to doing more toxicity testing, we also need to develop more sensitive approaches to testing that can reliably detect the long term consequences of exposures to toxic chemicals in early life. Extensive experiences demonstrated that infants and young children are uniquely vulnerable to certain chemicals that are relatively harmless to adults. To detect the unanticipated consequences of early exposures to such chemicals, it will be necessary to develop new approaches to assay prenatal, perinatal and childhood toxicity. For certain classes of chemicals it may, in part, be necessary to undertake experimental studies in which chemicals are administered shortly after birth and the experimental subjects then followed over their entire life span.<sup>(22)</sup> This approach will replicate the human condition in which exposures in the earliest stages in life may produce disease only decades later. It may thus enhance detection of the environmental causes of late illness. Functional tests of neurotoxicity and of immune, endocrine and reproductive toxicity are also need to be much more widely applied than they are at present.

Right-to-know is the concept that American families have the right to be informed of the nature and toxic properties of the chemicals that they may encounter in their air, food, drinking water, schools and communities. It is a powerful tool for cancer prevention, and it complements and extends the efficacy of regulation.

Right-to-know information empowers families and enables them to take intelligent decisions to reduce their own and their children's exposures to toxic substances. Right-to-know has proven



an extremely effective means for reducing toxic exposures. For example, EPA's Toxic Release Inventory (TRI) an annual listing of the nature and amounts of toxic chemicals released to the environment by polluting industries in the United States has highlighted those industries that are the worst actors and has resulted in many of these industries' taking aggressive steps to reduce their toxic emissions. Likewise Proposition 65 in California requires manufacturers to list hazardous materials on the labels of consumer products. This labeling requirement has resulted in the removal of many toxic products from the market in California and nationwide.

It will be necessary now to consider development of national right-to-know legislation in the United States that extends to consumers across this nation the sort of knowledge now available only on the west coast.

Regulatory standards issued by the Environmental Protection Agency and the Occupational Safety and Health Administration are an extraordinarily important mechanism for the prevention of environmental cancer. These standards regulate permissible uses of carcinogenic chemicals and establish levels above which workers and the public may not legally be exposed. Standards have brought about substantial reductions in exposures to carcinogens, including asbestos, benzene, vinyl chloride and PCBs. All standards are however, inherently arbitrary – they imply safety when safety does not exist. There is no bright line between the level of exposure to a toxic substance that causes cancer and that which is safe; there is instead a continuum of toxicity. Standards therefore need continually to be re-examined in the light of new data, and when necessary revised.

Traditionally, regulatory standards in this nation have been built on the assumption that the entire American population is comprised of 70-kilogram young adult males. Estimates of risks have been based on the exposures and the sensitivities of this “average” person, and standards have been set at levels to protect this person's health. The only federal environmental law that specifically acknowledges the unique sensitivities of infants and children is the Food Quality Protection Act of 1996. This legislation, which governs the use of pesticides in agriculture, requires that standards be set at levels that will specifically protect infants and children from harm to their health. In the years ahead, it will be necessary to extend the model of the Food

Quality Protection Act to other environmental legislation so that all environmental standards are set at levels that will protect the health of the most vulnerable among us.

Research. A vigorous national research program is an essential element of a comprehensive blueprint for cancer prevention. In this nation we have traditionally directed the major portion of our cancer research portfolio into discovering new cancer treatments. This approach has yielded great benefits. Death rates from many cancers, in particular pediatric cancers and testicular cancer, have been substantially reduced. Thirty years ago when I was still a pediatric resident, every child with leukemia died of their disease. Today more than three-fourths of children with leukemia survive and live to play another day.

Now it is time to open a second front on the war on cancer. We need to increase substantially our investment in prevention oriented research. It may be instructive to contrast our approach to cancer research with our approach to research on cardiovascular disease. The national portfolio on Cardiovascular Disease has long emphasized a search for the preventable causes of disease. This tradition began in 1948 when the U.S. Public Health Service established the Framingham Heart Study in Framingham, Massachusetts with the specific goal of identifying the preventable causes of heart disease and stroke. The study was initiated in the years after World War II when Americans had returned home to new prosperity, were eating a diet extremely high in cholesterol, were smoking at unprecedentedly high rates and experiencing massively increasing rates of heart disease and stroke. The Framingham Study and other studies like it identified the preventable environmental risk factors for heart disease such as hypertension, cholesterol, obesity, cigarette smoking, diabetes and sedentary lifestyles. Once these risk factors had been identified, aggressive programs of prevention were put into place. The result has been a reduction in heart disease rates among American men and women of nearly 50% over the past five decades. That reduction represents one of the great triumphs of public health in the past half century. We need now to do the same for cancer.

Conclusion. Cancer is a complex, multifactorial, profoundly frightening and often deadly disease. But also cancer is a preventable disease. Many thousands of cancer deaths in this

nation every year are caused by toxins in the environment, and those are cases that can and should be prevented.

Cancer prevention requires a carefully orchestrated, precisely targeted series of programs in prevention and research. These programs can result in enormous reductions in cancer incidence, suffering and death. The challenge before us as a nation is to craft such programs. We must track disease. We must test chemicals. We must educate and inform our citizens. We must commit to research in cancer prevention resources of the magnitude that we have historically committed to research in cancer treatment. Cancer prevention is cost-effective. Cancer prevention makes sense. And cancer prevention is the right thing to do.

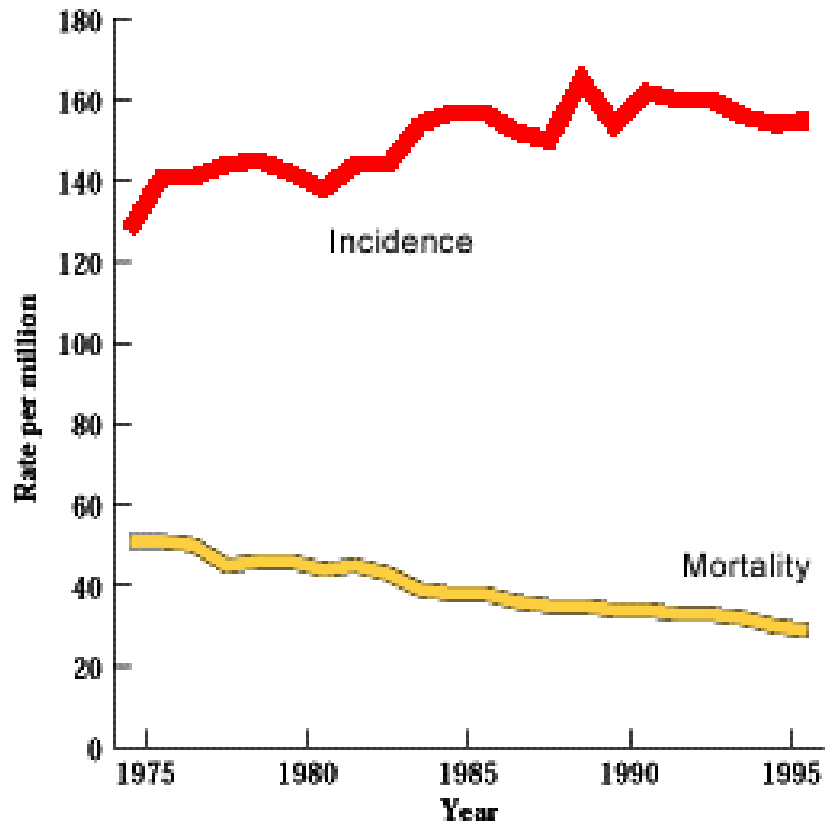
Thank you. I shall be pleased to answer your questions.

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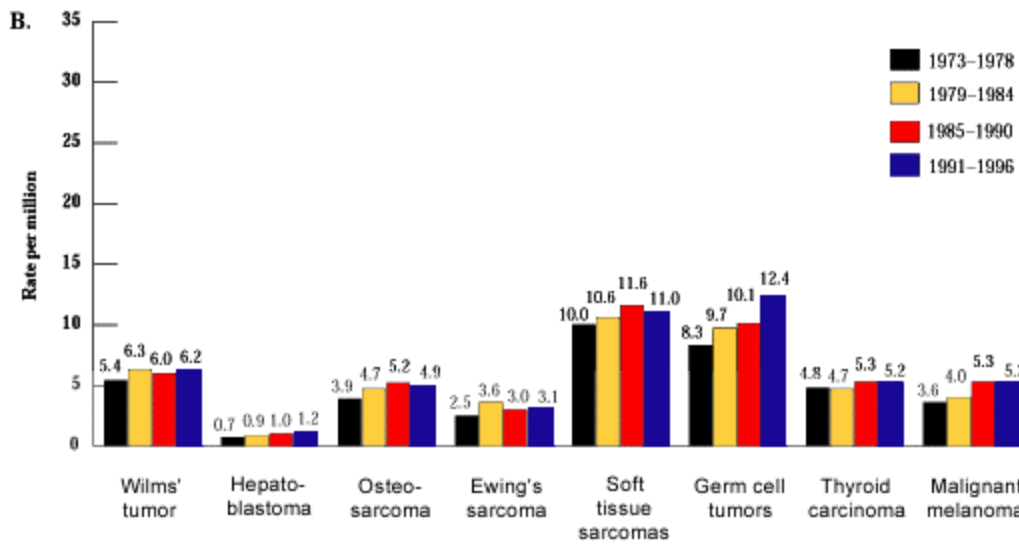
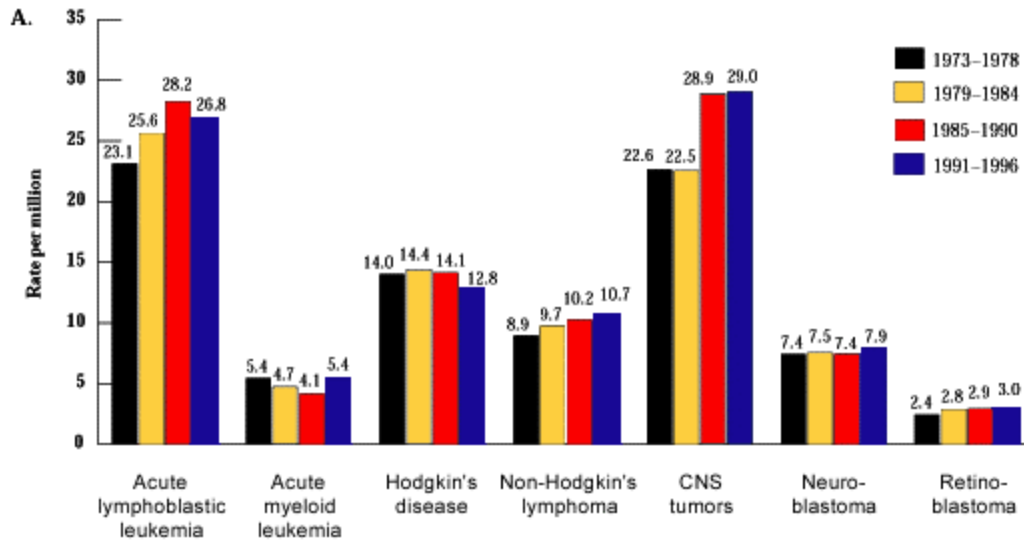
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Childhood Cancer (Age 0-19), Age-Adjusted Incidence and Death Rates, 1975-1996



Source: American Cancer Society

ACS Statistics 2000 Cancer Facts & Figures Graphical Data Age-Adjusted Incidence Rates, Childhood Cancer (Age 0-19), By Period of Diagnosis, 1973-1996



Source: American Cancer Society

## CURRICULUM VITAE

**Name:** Philip J. Landrigan, M.D., M.Sc., D.I.H.  
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**Born:** Boston, Massachusetts, June 14, 1942

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### Education:

**High School:** Boston Latin School, 1959  
**College:** Boston College, A.B. (magna cum laude), 1963  
**Medical School:** Harvard - M.D., 1967  
**Internship:** Cleveland Metropolitan General Hospital, 1967-1968  
**Residency:** Children's Hospital Medical Center, Boston,  
(Pediatrics), 1968-1970  
**Post Graduate:** London School of Hygiene & Tropical Medicine, 1976-77  
Diploma of Industrial Health (England), 1977  
Master of Science in Occupational Medicine,  
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### Positions Held:

**Current:** **Mount Sinai School of Medicine**, Ethel H. Wise Professor of Community and Preventive Medicine and Chairman of the Department of Community and Preventive Medicine, 1990-Present.  
**Mount Sinai School of Medicine**, Director, Division of Environmental and Occupational Medicine, Department of Community and Preventive Medicine, 1985-Present.  
**Mount Sinai School of Medicine**, Professor of Pediatrics, 1985-Present.

**Previous:** **U.S. Environmental Protection Agency**, Senior Advisor to the Administrator on Children's Health and the Environment, 1997-1998.  
**National Institute for Occupational Safety and Health**, Director, Division of Surveillance, Hazard Evaluations and Field Studies, 1979-1985.  
**Centers for Disease Control**, Chief, Environmental Hazards Activity, Cancer and Birth Defects Division, Bureau of Epidemiology, , 1974-1979.

- Director, Research and Development, Bureau of Smallpox Eradication, 1973-1974.
- Epidemic Intelligence Service (EIS) Officer, 1970-1973.



### **Adjunct Positions :**

**University of Washington School of Public Health and Community Medicine**, Clinical Professor of Environmental Health, 1983 - Present.

**Harvard Medical School**, Visiting Lecturer on Preventive Medicine and Clinical Epidemiology, 1982 - Present.

**Harvard School of Public Health**, Visiting Lecturer on Occupational Health, 1981 - Present.

**University of Cincinnati**, Department of Environmental Health, College of Medicine, Assistant Clinical Professor of Environmental Health, 1981 - 1986.

**London School of Hygiene and Tropical Medicine**, Visiting Fellow, TUC Institute of Occupational Health, 1976 - 1977.

**Harvard Medical School**, Clinical Instructor in Pediatrics, 1969 - 1970.

### **Memberships :**

American Academy of Pediatrics, Fellow

Society for Epidemiologic Research, Member

American Public Health Association, Member

Occupational Health Section, Chair, 1989-90.

Royal Society of Medicine, Elected Fellow

International Commission on Occupational Health, Member

Scientific Committee on Epidemiology

American College of Epidemiology, Fellow

Board of Directors, 1990 - 1993.

American Epidemiological Society, Elected Member

Collegium Ramazzini, Fellow

President, 1997-present.

Herman Biggs Society, Member, 1986-1992.

New York Academy of Sciences, Fellow

New York Occupational Medicine Association, Member

Board of Directors, 1988 - 1990.

American College of Occupational and Environmental Medicine, Fellow

New York Academy of Medicine, Elected Fellow

Physicians for Social Responsibility, Member

Board of Sponsors, 1994-95; Board of Directors 1996-1999.

### **Specialty Certifications :**

American Board of Pediatrics - 1973

American Board of Preventive Medicine:

General Preventive Medicine - 1979

Occupational Medicine - 1983

### Awards and Honors:

**Institute of Medicine**, National Academy of Sciences, Elected to membership, 1987  
**U.S. Department of Health, Education and Welfare**, Volunteer Award, 1973  
**U.S. Public Health Service**, Career Development Award, 1976  
**Centers for Disease Control**, Group Citation as Member of Beryllium Review Panel, 1978  
**U.S. Public Health Service**, Meritorious Service Medal, 1985  
**New York Committee for Occupational Safety and Health**, Annual Honoree, 1985  
**New England College of Occupational and Environmental Medicine**, Harriet Hardy Award, 1993  
**United Brotherhood of Carpenters**, William Sidell Presidential Award, 1995  
**American Public Health Association**, Herbert L. Needleman Medal and Award for Scientific Contributions and Advocacy on Behalf of Children, 1995.  
**International Association of Fire Fighters**, Occupational Health and Safety Award, 1995  
**Physicians for Social Responsibility**, Broad Street Pump Award in Environmental Health, 1996  
**International Society for Occupational and Environmental Health**, Vernon Houk Award, 1998  
**American College of Preventive Medicine**, Katherine Boucot Sturgis Award, 1999  
**Mothers & Others for a Livable Planet**, Award for Advocacy on Behalf of the Health of Children, 1999  
**Earth Day New York**, Award for Excellence in Environmental Medicine, 1999  
**Russian Academy of Medical Science**, Elected as Foreign Member, 2000  
**American Conference of Governmental Industrial Hygienists**, William Steiger Memorial Award, 2000  
**Environmental Advocates (New York)**, Award for Environmental Advocacy on Behalf of Children, 2000

### Visiting Professorships and Lectureships:

**University of Tokyo**, Visiting Professor of the Faculty of Medicine, September 1989  
**University of Tokyo**, Visiting Professor of the University, July 1990  
**University of Cape Town Medical School**, Visiting Professor, Department of Community Health, March 1992  
**Medical College of Pennsylvania**, Catherine Boucot Sturgis Visiting Professor in Community and Preventive Medicine, March 1992  
**National University of Singapore**, Visiting External Examiner in Occupational Medicine, 1994  
**Duke University Medical School**, Visiting Professor, NIEHS Clinical Training Program in Environmental Medicine, 1995  
**Mayo Clinic**, Department of Pediatrics, Amberg-Helmholtz Lecturer in Pediatrics, 1998  
**Centers for Disease Control and Prevention**, Langmuir Memorial Lecturer, 1999  
**University of Rochester**, 44<sup>th</sup> Annual Paul W. Beaven Lecturer, 2000  
**Royal College of Physicians (London)**, Faculty of Occupational Medicine Richard Schilling Memorial Lecturer, 2000

## **Committees:**

### **The White House**

Presidential Advisory Committee on Gulf War Veterans' Illnesses, 1995-1996.

### **American Academy of Pediatrics**

Committee on Environmental Hazards, 1976 - 1987. Chairman, 1983-1987.

### **National Research Council**

National Academy of Sciences, Assembly of Life Sciences. Board on Toxicology and Environmental Health Hazards, 1978-1987; Vice-Chairman, 1981-1984.

National Academy of Sciences, Assembly of Life Sciences, 1981-1982;  
Commission on Life Sciences, 1982-1984.

Institute of Medicine, Committee for a Planning Study for an Ongoing Study of Costs of Environment-Related Health Effects, 1979-1980.

National Academy of Sciences, Panel on the Proposed Air Force Study of Herbicide Agent Orange, 1979-1980.

National Academy of Sciences, Committee on the Epidemiology of Air Pollutants, Vice-Chairman, 1984-1985.

National Academy of Sciences, Committee on Neurotoxicology in Risk Assessment, 1987-1989.

National Academy of Sciences, Committee on the Scientific Issues Surrounding the Regulation of Pesticides in the Diets of Infants and Children, Chairman, 1988-1992.

National Academy of Sciences, Board on Sustainable Development, 1995-1998.

### **National Institutes of Health/U.S. Public Health Service**

National Institutes of Health, Study Section on Epidemiology and Disease Control, 1986-1990.

National Institute of Environmental Health Sciences, Third Task Force for Research Planning in the Environmental Health Sciences; Chairman, Subtask Force on Research Strategies for Prevention of and Intervention in Environmentally Produced Disease, 1983-1984.

National Institute for Occupational Safety and Health, Board of Scientific Counselors, 1995-1997.

Food and Drug Administration, Ranch Hand Advisory Committee, 2000-2001

### **Department of Defense**

Armed Forces Epidemiological Board, 2000-Present

### **State and Local Government**

State of New York, Governor's Blue Ribbon Committee on the Love Canal, 1978-1979.

State of New Jersey, Meadowlands Cancer Advisory Board, Chair, 1987-1989.

State of New York, Asbestos Advisory Board, Chair, 1987 - Present.

State of New York, New York State Advisory Council on Lead Poisoning Prevention, Chairman, 1993 - Present.

City of New York, Mayor's Lead Paint Poisoning Advisory Committee, 1991-1993.

State of New York, Public Health Priorities Committee, 1996.

State of New York, Health Research Science Board, 1997 - Present.

## **Academic**

Harvard School of Public Health, Occupational Health Program, Residency Review Committee, 1981-1983; Chairman, 1981.

New York Academy of Medicine, Working Group on Housing and Health, 1987-1989; Chairman, 1989.

Association of University Programs in Occupational Health and Safety, 1985 – Present; President, 1986-1988.

New York Lung Association, Research and Scientific Advisory Committee, 1986-1989. Board of Directors, 1987-1990.

Milbank Memorial Foundation, Technical Board, 1986-1988.

Mickey Leland National Urban Air Toxics Research Center, National Advisory Committee, 1994-1995.

Cornell University, Dean's Advisory Council in Veterinary Medicine, 1996-1997.

## **International Organizations**

World Health Organization. Contributor to the WHO Publication: "Guidelines on Studies in Environmental Epidemiology" (Environmental Health Criteria, No. 27), 1984.

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## **Environmental Organizations**

INFORM, Board of Directors, 1991 - Present.

Environmental Health Foundation, Board of Directors, 1993 - 1996.

Colette Chuda Environmental Fund, Scientific Advisory Committee, 1994 - Present.

Children's Health Environment Coalition, Board of Directors, 1996 - Present.

Children's Environmental Health Network, Board of Directors, 1995 - Present.

## **Labor Unions**

United Automobile Workers (UAW) - Chrysler Corporation, Joint Scientific Advisory Committee, Member, 1990 - Present.

United Brotherhood of Carpenters, National Health and Safety Fund, Medical Advisory Committee, 1990 - Present; Chairman, 1994 - Present.

International Association of Fire Fighters, John Redmond Foundation, Medical Advisory Committee, 1989 - Present.

International Brotherhood of Teamsters, National Health and Safety Advisory Committee, 1994 - Present.

George Meany Center for Labor Studies, Board of Trustees, 1994-1997.

## **Other Organizations**

Health Insurance Plan (HIP) of Greater New York, Board of Directors, 1992-1994.

American Legion, Science Panel, Chairman, 1988 - Present.

## Editorial Boards

Editor-in-Chief: *American Journal of Industrial Medicine*, 1992 - Present; Consulting Editor, 1979-1992.

Editor-in-Chief: *Environmental Research*, 1987-1994.

Consulting Editor: *Archives of Environmental Health*, 1982 - Present.

Editorial Board: *Annual Review of Public Health*, 1984-1990.

Senior Editor: *Environmental Research*, 1985-1987.

Editorial Board: *American Journal of Public Health*, 1987 - Present.

Editorial Board: *New Solutions: A Journal of Environmental and Occupational Health Policy*, 1990 - Present.

Editorial Board: *The PSR Quarterly: A Journal of Medicine and Global Survival*, 1990-1994.

Editorial Board, *Journal of Public Health Management and Practice*, 1995-1996.

## National Service

United States Public Health Service, Commissioned Corps, 1970-1985. LCDR (04) to CAPT (06).

United States Naval Reserve, Medical Corps, 1996 - Present.

LCDR (0-4) 1996-98; CDR (0-5) 1 April, 1998 - Present.