The purpose of the attached Consensus Statement on Cancer and the Environment is to forge a national and international consensus on how to prevent environmentally-related cancers. This Consensus Statement has been prepared for submission to the President's Cancer Panel, which is holding the four national meetings in 2008/2009 on cancer and the environment.

We welcome the decision to focus the hearings of the President's Cancer Panel on cancer and the environment. The three distinguished Panel Members will review the scientific evidence on the contribution of environmental exposures to cancer causation and on the strategies that can reduce the environment's role in causing cancer. The timing is fortuitous: the Panel will have the unique opportunity to present its findings to a new President at a time when an expanded perspective on cancer causation and prevention could significantly contribute to cancer research, policy and education.

The drafting and sign-on process is being facilitated by the Collaborative on Health and the Environment (www.healthandenvironment.org). If you are a scientist, researcher, health professional or advocate and you agree with the content of the Consensus Statement on Cancer and the Environment, please join us and sign the Statement. As of Nov 26, 2008, over 160 people and organizations have signed the Statement, indicating their support.

To sign the Statement, visit: www.healthandenvironment.org/cancersignon

To view the growing list of people and organizations who have signed the Statement, visit: www.healthandenvironment.org/cancersigners

Consensus Statement on Cancer and the Environment

Creating a National Strategy to Prevent Environmental Factors in Cancer Causation

October 2008

Introduction

Cancer is the second most common cause of death in the United States, accounting for 1 of every 4 deaths.[1] Data from the National Cancer Institute's Surveillance Epidemiology End Results Program (SEER) estimates that 45% percent of men and 40% of women in the U.S. will be diagnosed with cancer at some point in their lives. The incidence of childhood cancer has increased steadily since 1975.[2]

In the 37 years since President Nixon signed the National Cancer Act, the major national voluntary and governmental cancer organizations have focused on more effective cancer treatments, improved access to care, improved quality of life for cancer survivors, discouraging smoking and reducing obesity. When prevention has been added to these objectives, the word has sometimes been used as a synonym for early detection, like mammograms and colon cancer screenings, and sometimes been used to describe the actions individuals can take to prevent cancer, like smoking cessation, exercise, and maintaining a healthy weight.

These important measures have achieved significant successes. The number of cancer deaths and the incidence of certain cancers including those of the lung, colo-rectum and breast, have dropped. But these successes must not lead to complacency: many tentacles of the cancer epidemic continue their deadly spread.

These frightening trends call for new efforts and new ways of thinking. We call for a major initiative to identify and eliminate environmental contributors to cancer to augment continued work to improve cancer treatments, access to care and quality of life for cancer survivors. There is good evidence to support actions to reduce "environmental exposures" to chemical contaminants and ionizing and non-ionizing radiation including electromagnetic fields, as well as risk factors in our diet, social and psychological stressors, and infectious agents.

The American public cares about cancer and the environment. Opinion polls repeatedly show that Americans are very concerned about exposures in air, water, and food, and in the home, the workplace, and schools that may contribute to cancer incidence. And, as this consensus statement will show, a growing body of scientific evidence supports their concern.

This consensus statement is not intended to be an exhaustive review of the evidence linking environmental factors to many different types of cancer. Instead, this statement summarizes the most important evidence and conclusions for policymakers, scientists, medical professionals and public health advocates.

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[1] American Cancer Society. *Cancer Facts & Figures 2008*. Atlanta: American Cancer Society; 2008.

[2] Ries LAG, Melbert D, Krapcho M, et al (eds). SEER Cancer Statistics Review, 1975-2004, National Cancer Institute. Bethesda, MD; based on November 2006 SEER data submission, posted to the SEER web site, 2007. Accessed 12/17/07 at: http://seer.cancer.gov/csr/1975_2004/

Why Environmental Exposures Matter

I. Although cancer death rates in the U.S. have decreased, the number of people who suffer from cancer continues to grow. We are learning through experience that reducing exposure to environmental cancer risk factors [3] saves lives.

There are over 10 million people living with cancer in the U.S. The lifetime risk (up to age 95) of being diagnosed with cancer has increased; data from the National Cancer Institute's Surveillance Epidemiology End Results Program (SEER) estimates that 45% percent of men and 40% of women in the U.S. will be diagnosed with cancer at some point in their lives. Cancer is the second most common cause of death in the US, exceeded only by heart disease.

Over the last 30 years, rates of some cancers have risen. These include:

- Kidney, liver and esophageal cancer as well as multiple myeloma in men.
- Melanoma, non-Hodgkin's lymphoma, leukemia and cancers of the bladder, thyroid and kidney in women.[4]
- Childhood cancers overall. [5]

The age-adjusted cancer incidence rate in the U.S. has begun to decline, mostly because of the recent decline in lung cancer in males, colo-rectal cancer in males and females, and very recently, breast cancer in females. However, incidence rates of several other cancers have remained steady or continued to rise. Among the top 15 cancers in men, multiple myeloma, cancers of the kidney, liver and esophagus increased from 1975 to 2004.[6] Over that same time period and among the top 15 cancers in women, melanoma, non-Hodgkin's lymphoma, leukemia and cancers of the bladder, thyroid and kidney have increased. In the last decade, thyroid cancer among women has nearly tripled.[7]

Many of the cancers whose rates have been rising in the past decade are unrelated to cigarette smoking. With few exceptions, improved diagnostic techniques and changes in disease classification do not explain the increased incidences of these cancers. However, the national investment in tobacco-related cancers and the subsequent decline or stabilization of lung cancer and several other tobacco-related cancers show us that preventing exposure to risk factors saves lives.

II. The methods that have been used to attribute cancer risk to environmental exposures are outdated and flawed, and should no longer be used to determine policy or set research priorities.

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[3] The term "risk factor" is used throughout this statement to capture the spectrum of causal and mechanistic pathways (tumor promotion, tumor initiation, tumor enabling, developmental disruption, endocrine disruption, developmental susceptibility, etc.) of various agents known or suspected to contribute to cancer. The more commonly used "carcinogen" is a subset of "risk factor" and does not adequately capture the complexity of cancer causation.

[4] Espey DK, Wu X, Swan J, et al. Annual report to the nation on the status of cancer, 1975-2004, featuring cancer in American Indians and Alaska Natives, Cancer. 2007; 110(10):2119-2152.

[5] Ries LAG, Melbert D, Krapcho M, et al (eds). SEER Cancer Statistics Review, 1975-2004, National Cancer Institute. Bethesda, MD; based on November 2006 SEER data submission, posted to the SEER web site, 2007. Accessed 12/17/07 at: http://seer.cancer.gov/csr/1975_2004/

[6] Espey DK, Wu X, Swan J, et al. Annual report to the nation on the status of cancer, 1975-2004, featuring cancer in American Indians and Alaska Natives, Cancer. 2007; 110(10):2119-2152.

[7] Ries LAG, Melbert D, Krapcho M, et al (eds). SEER Cancer Statistics Review, 1975-2004, National Cancer Institute. Bethesda, MD; based on November 2006 SEER data submission, posted to the SEER web site, 2007. Accessed 12/17/07 at: http://seer.cancer.gov/csr/1975_2004/

In 1981, Richard Doll and Richard Peto[8] estimated the percentage of cancer deaths that would be avoided if certain risk factors were addressed, including occupational and environmental exposures, tobacco smoking, diet, alcohol consumption, and others. The Harvard Center for Cancer Prevention [9] used this same method of calculating attributable fractions in 1996 and it was used again by Doll in 1998.[10] These well-cited sources estimated that only a small percentage of cancer deaths could be prevented by reducing exposure to cancer risks in the environment (~2%) or in workplaces (~4% - 5%), while the majority of cancers would be avoided by improvements in diet and eliminating exposure to tobacco smoke—results which are reflected in over half of our statewide cancer prevention programs.[11]

These estimates of attributing cancer risk do not reflect our new understanding of the complexity of cancer causation. As acknowledged later by Sir Richard Doll, the calculation of attributable fractions fails to account for the fact that exposures interact with each other, making the attribution of cancers to single causes highly speculative and uncertain. National cancer research and intervention priorities over the past 37 years have been strongly influenced by Doll and Peto's attributable fractions. Those priorities need to be reset to reflect our new understanding of the complex causes of cancer.

Carcinogenesis is a multistage process that may begin as early as embryonic development and unfolds over a person's life. It includes tumor initiation, promotion and progression. Risk factors for cancer can act alone to trigger a single stage in this process, or through the interaction with other factors to either directly or indirectly initiate one or more of the stages of carcinogenesis.

We now know that complex interactions among genetic inheritance, diet, social circumstances and exposures to a variety of environmental agents – including chemicals known to cause cancer, radiation, and some biological organisms – influence cancer risk. The presence of one or more risk factors, such as exposure to dioxin,[12] or agents such as bisphenol A that disrupt endocrine signaling,[13] can profoundly influence the extent of risk posed by other factors.

Given this complex cancer model, simple calculations of the fractions of cancer attributable to single agents are misleading. Single agents always act within multidimensional causal webs reflecting the cumulative interaction among risk factors at various levels of organization (biological, social and ecological) and scales (individual, family, community, society and ecosystem). While on a population-level, there are certainly risk factors such as tobacco smoke that increase the risk of disease dramatically, more often we are challenged by many concurrent exposures, each of which may pose only a modest increase in cancer risk, but which can add up to harm and represent significant threats to public health.[14]

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[8] Doll R and R Peto. The Causes of Cancer: Quantitative estimates of Avoidable Risks of Cancer in the United States Today, Journal of the National Cancer Institute. 1981; 66: 1191-1308.

[9] Harvard Reports on Cancer Prevention, Vol 1: Human Causes of Cancer. Cancer Causes & Control, November 7, 1996. Accessed June 9, 2008 at: www.hsph.harvard.edu/cancer/resources_materials/reports.index.htm

[10] Doll R. Epidemiological Evidence of the Effects of Behavior and the Environment on the Risk of Human Cancer, Recent Results in Cancer Research. 1998; 154: 3-21.

[11] Centers for Disease Control and Prevention. Comprehensive Cancer Control Plans: A Content Review. December 2005.

[12] Webster TF and Commoner B. Overview: the dioxin debate. In Dioxins and Health, 2nd Edition. John Wiley & Sons, Inc (Hoboken, NJ) 2003.

[13] Ho S-M, Tang W-Y, Belmonte de Frausto J et al. Developmental Exposure to Estradiol and Bisphenol A Increases Susceptibility to Prostate Carcinogenesis and Epigenetically Regulates Phosphodiesterase Type 4 Variant 4, Cancer Research. 2006; 66 5624-5632.

[14] Poulsen HE, Prieme H, Loft S. Role of oxidative DNA damage in cancer initiation and promotion, European Journal of Cancer Prevention. 1998 Feb; 7(1):9-16.

Successful cancer prevention will depend on addressing other co-occurring and interactive risk factors[15] [16] along with the broader set of social conditions that influence these exposures.

How We Can Prevent Cancer: Six Actions That Will Reduce the Incidence of Environmentally-Related Cancer

The following actions are justified by the existing scientific evidence linking environmental factors to cancer. An effective national cancer prevention strategy will incorporate these goals prominently into research, policy and education agendas.

Action 1: Expand cancer goals to include changing the conditions that contribute to cancer.

As outlined in the introduction, cancer organizations in the U.S. have largely focused their efforts on detection, treatment, lifestyle choices, and quality of life. In recent years, though, both governmental and non-governmental organizations in the U.S. have set goals of reducing cancer incidence and mortality. But even these efforts have a way to go.

The Centers for Disease Control and Prevention (CDC) set a target as part of its Healthy People 2010 initiative to "reduce the number of new cancer cases as well as the illness, disability and death caused by cancer."[17] Although a target of a 21% reduction in cancer mortality was outlined in addition to targets for the reduction of cancer deaths associated with seven specific cancer types, no target was outlined for reduction of cancer *incidence*, nor was there a target for the reduction of specific disparities in cancer incidence.

The CDC, through multiple programs and initiatives, has a prevention agenda that includes objectives and strategies for lung cancer (tobacco cessation) and melanoma/skin cancer (sun exposure education). But it does not set goals for environmental and occupational exposures that increase cancer risk more broadly.[18] However, some states such as Maryland, which receives CDC funding through its National Cancer Control Program are implementing a model program for cancer prevention associated with environmental and occupational exposures.[19] And the CDC's Health Protection Goals take a step in the right direction by focusing on the health of workplaces and communities.[20]

Many other countries actively track progress toward more upstream cancer-prevention goals, emphasizing the importance of creating conditions that foster health and reduce the risk of disease.[21] An effective

[15] Hardell L. Pesticides, soft-tissue sarcoma and non-Hodgkin lymphoma - historical aspects on the precautionary principle in cancer prevention, Acta Oncologica. 2008; 47(3): 347-354. Appropriate citations

[16] Coombs NJ, Taylor R, Wilcken N, et al. HRT and breast cancer: impact on population risk and incidence, European Journal of Cancer. 2005; 41(2): 1775-1781. Appropriate citation?

[17] Centers for Disease Control and Prevention. Healthy People 2010. Accessed 12/13/07 at: http://www.healthypeople.gov/Document/HTML/Volume1/03Cancer.htm

[18] Centers for Disease Control and Prevention. Preventing and Controlling CancerThe Nation's Second Leading Cause of Death At A Glance 2008. Accessed 8/11/08 at: http://www.cdc.gov/nccdphp/publications/aag/dcpc.htm

[19] Maryland Family Health Administration, Maryland Department of Health and Mental Hygiene. The Maryland Comprehensive Cancer Control Plan Our Call to Action 2004-2008. Chapter 8: Environmental Issues and Cancer. Accessed 8/11/08 at: http://www.fha.state.md.us/cancer/cancerplan/plan/Ch8_Environmental_Issues%20and%20Cancer.pdf

[20] Centers for Disease Control and Prevention. Health Protection Goals. Accessed 7/10/08 http://www.cdc.gov/osi/goals/places.html

[21] Canadian Public Health Association. Public Health Goals for Canada: A Federal, Provincial and Territorial Commitment to Canadians. Accessed http://www.healthycanadians.ca/index_e.html

national cancer prevention strategy will address the myriad factors at multiple levels that collectively create the conditions out of which cancer patterns emerge.

Action 2: Take action now on what we already know by supporting policy and market-based efforts to reduce exposures to cancer-causing agents.

We still don't understand everything about the causal web of cancer, with its countless tangled strands of stressors and risk factors. Additional research is crucial. But this doesn't mean we can't take action now. Indeed, it is imperative that we act on what we do know.

The most direct way to prevent cancer is to stop putting cancer-causing agents into our indoor and outdoor environments in the first place. Known and suspected cancer contributors should be prioritized based on preliminary evidence of harm, shared mechanisms with chemicals known to be harmful, and potential exposures to vulnerable populations. A significant investment in green chemistry and the development of less hazardous products and materials is required. National goals must be set that aim to reduce the upstream causes of cancer.

We can begin to act on other major known risk factors by reducing socioeconomic disparities, improving lifelong fitness and nutrition, and reducing exposures to harmful chemicals, radiation, and other environmental contributors.

We can also take a precautionary approach to unknown risk factors. We have concerns with the dramatic increases in exposure to electromagnetic fields, especially cell phones, and nanoparticles, which are being widely added to consumer products. Both contaminants are examples of rapidly proliferating exposures for which the evidence of safety and harm is limited. We believe nanoparticles require a prudent safety testing regime before they are widely introduced. We believe that the weight of the evidence on the potential cancer hazard from cell phones is sufficient to justify an urgent increase in independent research and precautionary recommendations to reduce EMF exposures from cell phones, especially for children. Above all, we believe that a prudent approach to the use of these and any other new technologies will take seriously the possibility that they may contribute to cancer risk.[22] [23]

Action 3: Create a new way of managing chemicals so we can determine which chemicals are safe and which are harmful.

Of the industrial chemicals in use in the U.S. today, the National Toxicology Program (NTP) has published long-term carcinogenicity studies of only 556 chemicals[24]– a very small portion of the total number in use today. And because of severely limited financial resources and the high cost of standard animal testing methods, the NTP identifies only about seven new carcinogens each year.[25]

The Toxic Substances Control Act (TSCA), the basis of U.S. chemical policy today, has allowed more than 60,000 chemicals to be in use without requiring the research necessary to determine their safety. We need comprehensive chemical policy reform. The burden must be on chemical manufacturers and users to test their products for health effects and argue that the benefits of their use outweigh the risks.

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[22] Hardell L, Carlber M, Hansson Mild K. Pooled analysis of two case-control studies on use of cellular and corless telephones and the risk for malignant brain tumours diagnosed in 1997-2003, International Archives of Occupational and Environmental Health. 2006; 79(8): 630-639.

[23] Takagi A, Hirose A, Nishimura T, et al. Induction of mesothelioma in p53+/- mouse by intraperitoneal application of multi-wall carbon nanotube, Journal of Toxicological Sciences. 2008;33(1):105-116.

[24] National Toxicology Program. Long-term study reports and abstract. Accessed on June 16, 2008 at: http://ntp.niehs.nih.gov/index.cfm?objectid=0847DDA0-F261-59BF-FAA04EB1EC032B61

[25] Report on Carcinogens, Eleventh Edition; U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program .

Simultaneously, we need to promote alternatives assessment and green chemistry to expedite the "sunsetting" of carcinogenic and endocrine-disrupting chemicals.

Action 4: Reduce cancer disparities in low income communities, communities of color, and workers with high exposures to cancer-causing agents.

The incidence of most cancers, with the exception of breast cancer and melanoma, increases with declining socioeconomic status (SES). Socioeconomic status, ethnicity and gender can significantly influence cancer risk at the individual, community, and societal levels, and must therefore be addressed at multiple levels. Genetic inheritance and environmentally mediated gene expression may also impact the biological effects of single and mixed exposures. Studying this phenomenon is complex and challenging, but there are clear associations that can guide inquiry and direct prevention efforts.

For example, in the U.S., low-income communities and communities of color are generally exposed to more environmental contaminants, poorer nutrition and poorer health care than more affluent communities. According to the American Cancer Society, lower-income workers and communities are disproportionately affected by environmental pollutants and occupational exposures.[26] Recent studies have shown that people of color, recent immigrants and the poor are far more likely to work with carcinogens, have less access to institutions that protect them, and suffer disproportionately from exposure to environmental contaminants where they live.[27] [28] Moreover, studies report increased levels of markers of inflammation and oxidative stress that are almost certain to be linked to increased risk of cancer, as well as a variety of other degenerative diseases, in people of lower socioeconomic status.[29] [30]

Disparities in income have increased markedly in the United States in recent decades and represent a major contributor to cancer incidence and mortality. No serious cancer prevention agenda can neglect the importance of returning to a more equitable society.

Action 5: Expand research into the complex webs of cancer causation including multiple exposures, low dose effects, and how early life exposures and other critical windows of vulnerability can increase cancer risk.

In the past, epidemiologic research has focused on measuring the cancer risk of one contaminant or exposure at a time. Where studies do examine the effect of multiple exposures, practical limitations in epidemiology often mean that researchers can examine only two or three agents at a time. Yet in our daily lives, we are not exposed to single agents, but rather to combinations of a large number of agents. New methods are needed to understand the cancer risks associated with exposure to combinations of chemicals and other factors, including dietary components and psychosocial stressors.

We must also continue to study the health effects of low-dose exposures. Science has already shown the inadequacy of the old dictum "the dose makes the poison." Linear dose-response modeling does not

[26] American Cancer Society. *Cancer Facts and Figures 2006.* Atlanta, GA: American Cancer Society; 2006.

[27] Morello-Frosch R, Pastor M, Porras C, Sadd J. Environmental justice and regional inequality in Southern California: implications for future research, *Environmental Health Perspectives. 2002; 110(2):149-154.*

[28] Brulle RJ and Pellow DN. Environmental Justice: Human Health and Environmental Inequalities, Annual Reviews of Public Health. 2006; 27:103-124.

[29] Harris RE. Cyclooxygenase-2 (cox-2) and the inflammogenesis of cancer. Subcell Biochem. 2007;42:93-126.

[30] Pollitt RA, Kaufman JS, Rose KM, Diez-Roux AV, Zeng D, Heiss G. Cumulative life course and adult socioeconomic status and markers of inflammation in adulthood. J Epidemiol Community Health. 2008 Jun;62(6):484-91.

capture all cancer risk. We know from many new studies that very low levels of exposure may correspond to an increased cancer risk and sometimes have even more of an effect than higher levels of exposure.[31] [32]

Age is another important factor in determining the cancer risk of a given exposure. Research has repeatedly documented examples of differential cancer risk with age at exposure.[33] [34] [35] [36] These examples and others [37] also demonstrate that early life exposures are likely to significantly impact later cancer risk, and exposure may even convey risk on future generations.[38] [39] [40] Understanding the timing of exposures that increase cancer risk later in life is critical to our success in prevention. Large prospective studies like the National Children's Study and the biomonitoring activities of CDC's National Center for Environmental Health will shed light on this issue.

Understanding the complexity of interactive cancer risk factors is not enough. An important body of research points to the importance of understanding the systemic conditions that contribute to cancer.[41] [42] This approach is well-recognized in social epidemiology, where researchers examine how different forms of social organization impact health, and in the ecological sciences, where ecologists study the conditions that lead to systemic resilience or system collapse.

We need research paradigms that recognize how critical system conditions are to preventing cancer and many other diseases. The systemic conditions that will reduce cancer incidence will also reduce the incidence of the many other epidemic diseases of our time.

Action 6: Implement a comprehensive cancer prevention strategy that sets the course for new directions in research, policy and individual action incorporating all of the aforementioned actions.

We need a comprehensive, effective U.S. cancer prevention strategy that promotes health, reduces the incidence of cancer, and protects the most vulnerable members of society.

The current cancer prevention work in the U.S. focuses primarily on smoking cessation, reducing obesity, early detection and increasingly, chemoprevention. Current policies call for increases in the screening

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[31] Hertz-Picciotto and Smith AH. Observations on the dose-response curve for arsenic and lung cancer, Scandinavian Journal of Work, Environment and Health. 1993; 19: 217-226.

[32] Durando M, Kass L, Piva J, et al. Prenatal bisphenol-A exposure induces preneoplastic lesions in the mammary gland in wistar rats, Environmental Health Perspectives. 2007; 115(1):80-86.

[33] Kneale GW, Stewart AM. Reanalysis of Hanford data: 1944-1986, American Journal of Industrial Medicine. 1993; 23(2):371-389.

[34] Richardson DB, Wing S. Greater sensitivity to ionizing radiation at older age: follow-up of workers at Oak Ridge National Laboratory through 1990, International Journal of Epidemiology. 1999; 28:428-436.

[35] Gilman EA, Kneale, GW, Knox EG, et al. Pregnancy x-rays and childhood cancers: effects of exposure age and radiation dose, Journal of Radiological Protection. 1988; 8(1):2-8.

[36] Cohn, BA, Wolfe MS, Cirillo PM, et al. DDT and breast cancer in young women: new data on the significance of age at exposure, Environmental Health Perspectives. 2007;115:1406–1414.

[37] Durando M, Kass L, Piva J, et al. Prenatal bisphenol-A exposure induces preneoplastic lesions in the mammary gland in wistar rats, Environmental Health Perspectives. 2007; 115(1):80-86.

[38] Anway MD, Leathers C and Skinner MK. Endocrine disruptor vinclozolin induced epigenetic transgenerational adult onset disease, Endocrinology. 2006; 147(12): 5515-5523.

[39] Newbold RR, Padilla-Banks E, Jefferson WN. Adverse effects of the model environmental estrogen diethylstilbestrol are transmitted to subsequent generations, Endocrinology. 2006; 147:s11-s17.

[40] Newbold R. Lessons learned from perinatal exposure to diethylstilbestrol, Toxicology and Applied Pharmacology. 2004; 199:142-150.

[41] Krieger N. Theories for social epidemiology in the 21st century: an ecosocial perspective, International Journal of Epidemiology. 2001; 30:668-677.

[42] Karpati A, Galea S, Awebuch T et al. Variability and vulnerability at the ecological level: implications for understanding the social determinants of health, American Journal of Public Health. 2002;92(11): 1768-1772.

rates for breast cancer, colon cancer and prostate cancers. But early detection is not prevention. While early detection strategies are critically important because they allow treatment to begin early in the course of disease, true cancer prevention strategies aim to reduce the incidence of cancer by lessening the number of stressors that cause disease.

This strategy would:

- Aim to reduce the incidence of cancer by lessening the number of stressors which contribute to it;
- Embrace the inherent complexity and integrate current knowledge on the role of ionizing and non-ionizing radiation, biological organisms, genetic inheritance, endocrine disruptors and chemical carcinogens in water, air, sediment food and consumer products; and the role of psychosocial factors like stress, socioeconomic status and income disparities.
- Set the course for new directions in research, policy and individual action; and
- Set concrete goals for reduction in the exposures that have already been implicated in cancer causation.

We owe it to ourselves, our children, our grandchildren, and the people around the world who are living and dying with cancer to expand our efforts to prevent the environmental causes of this devastating disease.

To sign this statement, please visit www.healthandenvironment.org/cancersignon. As of Nov 26, 2008, over 160 people and organizations have signed (see www.healthandenvironment.org/cancersigners).

The following scientists, researchers, health professionals and advocates affirm that they each agree with the content of the Consensus Statement on Cancer and the Environment:

- **Dr. Eula Bingham** (Professor of Environmental Health, University of Cincinnati College of Medicine; former Administrator, Occupational Safety and Health Administration)
- **Dr. Julia Brody** (Executive Director, Silent Spring Institute)
- **Dr. Richard Clapp** (Professor of Environmental Health, Boston University School of Public Health)
- **Dr. Devra Davis** (Executive Director, Center for Environmental Oncology, University of Pittsburgh Medical Center, Cancer Pavilion; Professor of Epidemiology, University of Pittsburgh Graduate School of Public Health)
- **Dr. Ronald Herberman** (Director, University of Pittsburgh Cancer Institute; Hillman Professor of Oncology and Professor of Medicine, University of Pittsburgh School of Medicine)
- **Dr. Robert Hiatt** (Professor and Co-Chair of Epidemiology & Biostatistics, University of California San Francisco School of Medicine)
- **Dr. David Kriebel** (Professor & Acting Chair of Work Environment, University of Massachusetts Lowell; Co-Director Lowell Center for Sustainable Production)
- **Dr. Nancy Krieger** (Professor of Society, Human Development & Health, Harvard University)
- **Dr. Phil Landrigan** (Professor & Chair of Community and Preventative Medicine & Professor of Pediatrics, Mt. Sinai Medical Center)
- **Dr. Phil Lee** (Professor Emeritus, Stanford University; Professor & Chancellor Emeritus University of California San Francisco School of Medicine; former U.S. Assistant Secretary of Health & Human Services)

Organizational signers include:

- Breast Cancer Fund
- Commonweal
- Science and Environmental Health Network