Cancer

Air and Water Pollutants

A Need for Action
Does breathing air and drinking water contribute to cancer?

Beginning with the landmark Clean Air Act of 1970 and Safe Drinking Water Act of 1974, the Federal government has recognized the need to safeguard two of our most fundamental natural and public health resources: the water we drink and the air we breathe.

Thirty years of regulations and technological advances have made cleaner, healthier air and water for millions of Americans. Yet in the United States:

- Over 105 million people in 2006 lived in counties with air pollutant levels exceeding Clean Air Act standards.¹
- About 24 million people in 2007 were served by community water systems that reported violations of health-based drinking water standards.²
- In 2006 alone, industries reporting to the Toxics Release Inventory released or disposed of 820 million pounds of known or suspected carcinogens,³ 70 million pounds of which went directly into the air or water.⁴

Mounting evidence links dozens of air and water pollutants—including two widespread categories of pollutants, particulate pollution in air and disinfection byproducts in water—with cancer.

Water pollution and cancer

**What do we know?**

Many different water pollutants increase cancer risk. Exposure to these pollutants can occur by drinking contaminated water or bathing, showering or swimming in it. Ironically, one major water pollutant that increases cancer risk comes from disinfection, a process that protects our health from other diseases.

Disinfecting public water supplies with chlorine reduces illness and death associated with waterborne microbes. But, when chlorine interacts with organic compounds often found in surface water, hundreds of different chemical mixtures—called disinfection byproducts—can form. In experimental animal studies, several disinfection byproducts were found to cause cancer, including chloroform, other trihalomethanes, and some haloacetic acids.⁵ Strong evidence from epidemiologic studies suggests that long-term exposure to disinfection byproducts in drinking water increases the risk of bladder cancer and possibly colon, rectal and esophageal cancers.⁶ Given the vast number of people who get their water from outdated treatment and delivery systems,
even modest elevations in cancer risk from disinfection byproducts can create a significant public health impact.

Disinfection byproducts are not the only water pollutants that may increase cancer risk. Radon in drinking water can contribute to a small but significant increase in the risk of lung cancer by contributing to radiation levels inside buildings. Exposure to arsenic is a well-established cause of bladder cancer as well as of lung, kidney, and non-melanoma skin cancers. In addition, prostate and liver cancers may also be linked with arsenic in drinking water although the evidence is only suggestive. In some areas of California, Nevada, Alaska, Michigan, New England, New Mexico and Utah, high levels of arsenic occur naturally in groundwater sources. Arsenic may also contaminate public and private water supplies in mining and ore processing regions. Public and private water supplies can be contaminated by many different pollutants from hazardous waste sites and industrial, commercial, agricultural and domestic sources. Studies of water contamination by these sources have observed that a range of pollutants—among them perchloroethylene, trichloroethylene, chlorophenols, and nitrates—are associated with elevated risks of several cancer types.

**WHAT CAN WE DO?**

How can we reduce the public health risks associated with water pollutants? Here are five broad steps we can take.

- Update water treatment facilities and delivery infrastructure to reduce disinfection byproducts.
- Enhance watershed protection programs to reduce contamination of surface waters.
- Reduce pollution by promoting green chemistry, alternatives assessment, and the sunsetting of cancer-contributing substances.
- Ensure that current drinking water standards truly protect public health. For example, EPA recently reduced the standard for arsenic from 50 parts per billion (ppb)—first set in 1942—to 10 ppb. Yet the estimated cancer risk associated with this new standard is still about 1 in 333 people: a risk 30 times greater than EPA usually considers acceptable.
- Create more protective standards for a range of common water pollutants, such as radon, atrazine, arsenic and various disinfection byproducts.
Outdoor air pollution and cancer

What do we know?

Outdoor air pollution—also called ambient air pollution—is a mixture of gases and particulates, the exact composition of which depends upon the pollutant sources and meteorological conditions of a given area. Major sources of outdoor air pollutants can include industrial sources, power plants and motor vehicles.

Many outdoor air pollutants are known or suspected to increase the risk of cancer. For example, a large number of carcinogenic pollutants are emitted in the combustion of fossil fuels and as constituents of airborne soot, including: diesel exhaust; benzo[a]pyrene and other polycyclic aromatic hydrocarbons (PAHs); 1,3-butadiene; benzene; inorganic compounds such as arsenic and chromium; radionuclides; formaldehyde; and fine particulate matter (called PM_{2.5} or particles with diameters less than 2.5 micrometers)—particles which often have other carcinogens adsorbed on their surfaces. Several studies provide evidence that outdoor air pollution can increase cancer risk, among them:

- Occupational studies demonstrating increased risk of lung cancer among railroad, bus garage, trucking, and dock workers exposed to diesel exhaust;
- Studies examining populations near specific air pollution point sources such as waste incinerators or metal smelters;
- Studies comparing cancer rates in rural and urban populations, which have consistently found excesses of lung cancer in urban areas, some of which implicate specific industrial air pollution point sources and motor vehicle traffic density.

Strength of the evidence linking specific cancers with human exposure to water contaminants

- **Bladder**: Strong: arsenic, water disinfection byproducts
- **Breast**: Suspected: organic solvents
- **Colorectal**: Suspected: water disinfection byproducts, nitrate
- **Esophageal**: Suspected: water disinfection byproducts
- **Kidney**: Strong: arsenic, nitrate, organic solvents
- **Leukemia**: Strong: arsenic, nitrate, organic solvents
- **Liver**: Suspected: arsenic, organic solvents, pesticides
- **Lung**: Strong: arsenic, radon
- **Prostate**: Suspected: arsenic
- **Skin**: Strong: arsenic
- **Non-Hodgkin’s Lymphoma**: Suspected: organic solvents, pesticides
Some of the strongest evidence supporting a link between air pollution and lung cancer emerged in the 1990s from three large prospective cohort studies. These studies and subsequent follow-up analyses showed elevations in lung cancer risk associated with exposure to specific constituents in air pollution, including: particulate matter (PM$_{10}$ or particles with diameters less than 10 micrometers), SO$_2$, PM$_{2.5}$, and sulfate particulate exposure. These findings were not attributable to confounding factors such as tobacco smoke. Estimates suggested that PM$_{2.5}$ was causing or contributing to a range of respiratory and cardiac conditions, killing 60,000 people each year. These staggering numbers led EPA to set new regulatory standards for PM$_{2.5}$ in 1997.

Since 1980, air quality has improved nationwide through compliance with the National Ambient Air Quality Standards (which sets limits for carbon monoxide, lead, nitrogen oxides, ozone, particulate matter and sulfur dioxide). The improvement can be attributed largely to pollution-control devices and reducing the use of materials and products that emit toxic air pollutants. But we still have a long way to go in reducing the risk of cancer from outdoor air pollutants.

- Despite overall reductions, levels of certain pollutants—including some carcinogens—have not declined in some areas of the country because of local or regional releases.
- In 2006, over 100 million people were living in communities where at least one of the six criteria air pollutants exceeded the regulatory limits. EPA estimates more than 270 million people live in census tracts for which the lifetime cancer risk from hazardous air pollutants exceeds a 10-in-one-million risk.

Unfortunately, controlling pollutants at the “end of pipe” after they have been created can never be fully effective, and the costs of such controls become prohibitive as their efficiency goes up. Eliminating pollutants at the source through redesigning technologies to make them inherently non-polluting is more effective.

**What can we do?**

There are a number of things we can do to reduce the risk of cancer from outdoor air pollutants.

- Increase enforcement of current regulatory standards.
- Continue to improve those standards based upon monitoring data and related health studies.
- Support the development and adoption of innovative, non-polluting technologies.
- Expand, support, and incentivize the use of public transportation systems.
Indoor air pollution

What do we know?

Adults in the United States spend approximately 90% of their time indoors. As a result, the health risks—including cancer risk—associated with exposure to indoor air pollutants can be significant. The very places we consider safe—our homes, workplaces, schools and health care facilities—may be a major source of exposure to air pollutants.

During the early 1980s, EPA conducted the Total Exposure Assessment Methodology (TEAM) Study to determine exposure to toxic air pollutants on a population basis in a range of microenvironments, including ambient and indoor air as well as exposure via water and food. Results from this study were striking: personal and indoor exposures often greatly exceeded the outdoor concentration of dozens of air toxics, leading to the conclusion that, “indoor air in the home and at work far outweighs outdoor air as a route of exposure to these chemicals.”

Indoor air pollution comes from many sources, including outdoor pollution filtering into buildings; contaminants in construction materials; consumer and office products containing or producing volatile or aerosolized compounds; gases or volatile compounds emanating from the soil or water near or below buildings; showers or other hot-water sources containing volatile contaminants; vehicular exhaust from attached garages; tobacco smoke; and combustion byproducts from heating and cooking. The levels of indoor air contaminants are also

EVIDENCE: Strength of the evidence linking specific cancers with human exposure to agents in air pollutants

<table>
<thead>
<tr>
<th>Cancer Type</th>
<th>Strength</th>
<th>Suspected Contaminants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bladder</td>
<td>Strong</td>
<td>coal tars, diesel exhaust, PAHs</td>
</tr>
<tr>
<td>Breast</td>
<td>Strong</td>
<td>environmental tobacco smoke, dioxin, PAHs</td>
</tr>
<tr>
<td>Esophageal</td>
<td>Suspected</td>
<td>soot</td>
</tr>
<tr>
<td>Laryngeal</td>
<td>Strong</td>
<td>asbestos</td>
</tr>
<tr>
<td>Leukemia</td>
<td>Strong</td>
<td>benzene and other solvents, pesticides</td>
</tr>
<tr>
<td>Lung</td>
<td>Strong</td>
<td>air pollution, arsenic, asbestos, chromium, coal tars, diesel exhaust, environmental tobacco smoke, nickel, PAHs, particulate air pollution, radon, soot, wood dust</td>
</tr>
<tr>
<td>Multiple myeloma</td>
<td>Strong</td>
<td>benzene, dioxin</td>
</tr>
<tr>
<td>Mesothelioma</td>
<td>Strong</td>
<td>asbestos</td>
</tr>
<tr>
<td>Non-Hodgkin's Lymphoma</td>
<td>Strong</td>
<td>benzene, dioxin</td>
</tr>
<tr>
<td>Soft-tissue sarcoma</td>
<td>Strong</td>
<td>dioxin</td>
</tr>
<tr>
<td>Skin</td>
<td>Strong</td>
<td>arsenic, coal tars, PAHs</td>
</tr>
<tr>
<td>Prostate</td>
<td>Suspected</td>
<td>arsenic, dioxin, PAHs</td>
</tr>
</tbody>
</table>

* For a complete list of the 187 compounds regulated as Hazardous Air Pollutants by the 1990 Amendments to the Clean Air Act and information about their links with cancer, see: http://www.epa.gov/ttn/atw/hltheb/hapindex.html
highly dependent on ventilation characteristics of the building as well as the season.

Indoor air pollutants can contribute to a wide range of acute and chronic health conditions, including cancer. Agents detected in the indoor environment with known or suspected links to cancer include radon, environmental tobacco smoke, asbestos, formaldehyde, chloroform and pesticides.\textsuperscript{6,25–27} Environmental tobacco smoke alone contains approximately 50 known carcinogens.\textsuperscript{28}

**What can we do?**

Eliminating the pollutant source is key to improving air quality and reducing associated health risks. Although EPA has the authority to address indoor air quality indirectly through regulation of outdoor air under the Clean Air Act, and the Occupational Safety and Health Administration has rules limiting some airborne hazards in workplaces, the United States lacks regulations providing the umbrella coverage needed to fully address indoor air quality.\textsuperscript{29} Local and state policies have been extremely effective at controlling exposure to environmental tobacco smoke in public spaces including restaurants and bars—policies that have dramatically reduced exposure to carcinogens in indoor air that impact both workers and patrons. Yet more action is needed to protect people from toxic consumer products and building materials, including labeling laws that require complete information about the chemical ingredients in products and their possible health effects; market incentives to develop cleaner and safer products, technologies, and buildings; and pre-market testing requirements to ensure the safety of products on the market.

**How cancer develops**

*MIXTURES, LOW DOSES, AND WINDOWS OF VULNERABILITY*

Most of us are probably exposed to dozens of carcinogens at low concentrations in the food we eat, the air we breathe, the water we drink, and the materials we encounter. But chemicals are tested for their carcinogenicity one at a time, and regulatory standards for specific chemicals are set based on the mistaken assumption that exposures to chemicals occur and deliver their risks one at a time. However, carcinogens almost certainly act within complex causal webs reflecting the cumulative interaction among risks across the life course and at various levels of organization (biological, social, and ecological) and scales (individual, family, community, society, and
Cancer risk is influenced by exposures to many different factors, including agents that can contribute to the disease in work environments, the general environment, diet, genetic inheritance, reproductive factors, and other lifestyle factors. Studies show that environmental contaminants may increase the risk of cancer through a variety of mechanisms, including genotoxicity, tumor promotion, hormonal action and immunotoxicity.\textsuperscript{30,31} Many of these effects can occur at low levels of exposure which depend not only on what a person is exposed to, but also the timing of exposures. For example, exposure to toxicants at low levels during periods of rapid growth and cell differentiation from fetal life through puberty can be an important contributor to cancer risk later in life.

Preventing cancer depends on addressing the broader set of conditions that influence risk in both our research and cancer prevention and control programs. New methods are needed to understand cancer risks associated with exposure to chemical mixtures, the effects at low doses and during critical windows of vulnerability. Although studies are beginning to reveal the interaction of chemical mixtures on a molecular level and new statistical methods are under development,\textsuperscript{32} it will take many years before experimental science fully reveals the effects of the complex mixtures to which we are all exposed in daily life. Fortunately, we know enough now to take action to reduce these cancer risks.

**Contaminated communities**

**WHAT CAN WE LEARN FROM CANCER CLUSTERS?**

In thousands of communities across the nation, people suspect they may be living in the midst of a cancer cluster, such as the infamous clusters linked with contaminated drinking water in Woburn, Massachusetts, and Tom’s River, New Jersey.\textsuperscript{33,34} Every year, local and state health departments respond to more than 1,000 inquiries regarding suspected cancer clusters.\textsuperscript{35,36} Clusters are verified approximately 5% to 15% of the time, although a causal explanation for the elevated cancer rates is often never identified.\textsuperscript{37}

Given the growing recognition that most cancers have multifactorial origins, there may be more to public concerns about cancer clusters than current science and regulatory practice generally recognize. Communities with multiple chemical exposures, poor nutrition, and other cancer risk factors, often experience higher rates of cancer and other diseases. Also, our dependence on statistical significance testing means that elevations of cancer incidence within small geographic areas are often dismissed.\textsuperscript{38} Unfortunately, government agencies often respond to cancer concerns
in connection with local environmental contamination by saying there is no evidence of a “statistically significant” elevation in cancer risk, or by explaining away a cancer cluster as a statistical fluke or as the result of high smoking rates or poor diets in the community.

History shows us that sometimes cancer clusters are indeed signals that a preventable exposure to a cancer-contributing substance or substances has occurred, and so it is inappropriate for cancer cluster investigations to dismiss exposure to local environmental contamination out of hand. Instead, the appropriate response to a proven or suspected cancer cluster is to use these investigations to engage concerned citizens and public health representatives in honest communication about the range of exposures, including local environmental contamination, that may increase a community’s cancer risk and identify ways to reduce that risk. Reducing or eliminating environmental pollution in a community is just as important to the health of its members as expanding programs focused on better nutrition, increasing exercise, and tobacco cessation. Not one but many interventions are needed to reduce the cancer burden in our society.

Environmental injustice

**DISPARITIES IN EXPOSURE TO AIR AND WATER POLLUTANTS**

In 2005, a headline-grabbing analysis by the Associated Press revealed that black Americans are 79% more likely than whites to live in neighborhoods where industrial pollution is suspected of posing the greatest health threat. While striking, these findings are not new. Over 20 years ago, The United Church of Christ published a landmark study, “Toxic Waste and Race in the United States” finding that the most important factor predicting the siting of toxic waste facilities across the United States was race.

Over the last two decades, dozens of environmental equity studies have examined whether sources of environmental risk are concentrated among racial and ethnic minorities and the poor. A recent review of the literature on differential exposures to environmental pollution found significant relationships between the ethnic and class characteristics of a

> “The poor and especially the nonwhite poor bear a disproportionate burden of exposure to suboptimal, unhealthy environmental conditions in the United States.”
community and levels of exposure to a variety of environmental risks, including proximity to hazardous waste sites, exposures to air and water pollution, as well as other sources such as housing and the work environment. These authors concluded that “the poor and especially the non-white poor bear a disproportionate burden of exposure to suboptimal, unhealthy environmental conditions in the United States.” Similarly, a comprehensive meta-analysis of 49 environmental inequality studies found “ubiquitous evidence” supporting race as the main factor associated with environmental inequities.

In 1994, President Clinton’s Executive Order 12898 directed the government to ensure equality in protecting Americans from pollution. The order stated: “Each Federal agency shall make achieving environmental justice part of its mission by identifying and addressing, as appropriate, disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations.”

During the years that followed, EPA and other federal agencies made substantial progress towards addressing the interrelated environmental, public health, economic and social inequities by ensuring greater access to and responsiveness of government for communities and ensuring the integration of environmental justice principles into all agencies’ programs, activities and rule-making processes. But in 2006, when EPA’s Office of Inspector General surveyed EPA’s programs to determine the extent to which the issue of environmental justice had been considered in various activities, it found major gaps. The Inspector General’s report stated, “Until these program and regional offices perform environmental justice reviews, the Agency cannot determine whether its programs cause disproportionately high and adverse human health or environmental effects on minority and low-income populations.”

A 2005 Governmental Accountability Office report reached a similar conclusion, stating that EPA devoted only minimal attention to environmental equality when developing three major rules to implement the Clear Air Act.

**What can we do?**

Despite greater awareness of environmental injustices that have contributed to cancer disparities in the United States, we need more progress to ensure equitable environmental, social and economic conditions that promote health. Three broad steps will help us move forward:

- Acknowledge that some populations suffer disproportionate exposures to and effects from air and water pollutants and other environmental hazards.
- Include achieving a more equitable society among the goals of a nationwide cancer prevention agenda.
- Make sure those among us who are disproportionately affected by air and water pollutants and other environmental hazards have a voice and a hand in creating and implementing our nationwide cancer prevention agenda.
A short prescription to prevent cancer associated with air and water pollutants

Air and water pollution are often direct consequences of the industrial society in which we live. If we want to prevent cancer associated with air and water pollutants, we need to control or eliminate the release of cancer-contributing substances along the entire lifecycle of products and materials, from manufacturing to use to disposal. A comprehensive U.S. cancer prevention agenda that promotes health, prevents cancer and protects the most vulnerable members of society must address industrial and other sources of air and water pollutants. To implement such an agenda, we must act to:

■ **Enforce** and update environmental regulations to ensure that everyone breathes clean air and drinks safe water where they live, work and play.

■ **Upgrade** water treatment facilities and distribution infrastructure across the United States in order to minimize drinking water contamination by disinfection byproducts and other contaminants found in outdated systems.

■ **Support** cancer research that captures the complexities of cancer causation, including multiple exposures, low-dose effects and critical windows of vulnerability.

■ **Create** a new chemicals regulation system that expands toxicity testing of new and existing industrial agents and consumer products and avoids releasing substances that increase cancer risk into our environments.

■ **Identify** safer alternatives to cancer-contributing substances used in products and processes.

■ **Acknowledge** that while scientific certainty is seldom possible, when sufficient evidence of harm exists, we have a duty to act to prevent harm.
REFERENCES


