Special Section: Environmental Pollutants and Cancer

Introduction

Environmental pollution in relation to cancer is a topic of considerable public interest and scientific debate. There are two major classes of factors that influence the incidence of cancer: hereditary factors and acquired (environmental) factors. Hereditary factors come from our parents and cannot be modified. Environmental factors are potentially modifiable. They include tobacco use, poor nutrition, inactivity, obesity, certain infectious agents, certain medical treatments, sunlight, cancer-causing agents that occur naturally in food, cancer-causing agents in the workplace, and cancer-causing agents that exist as pollutants in our air, water, and soil.

These environmental factors account for an estimated 75%-80% of cancer cases and deaths in the US. Exposure to pollutants in occupational, community, and other settings is thought to account for a relatively small percentage of cancer deaths, about 4% from occupational exposures and 2% from environmental pollutants (man-made and naturally occurring). Although the estimated percentage of cancers related to occupational and environmental pollutants is small compared to the cancer burden from tobacco smoking (30%) and the combination of nutrition, physical activity and obesity (35%), the relationship between pollution and cancer is important for several reasons.

First, even a small percentage of cancers can represent many deaths: 6% of cancer deaths in the United States each year corresponds to approximately 33,900 deaths. Second, exposure to occupational and environmental pollutants is borne disproportionately by lower-income workers and communities, contributing to disparities in the cancer burden across the population. For example, it was epidemics of occupationally related cancer, primarily among industrial workers, that led to identification of a number of chemicals known to cause cancer in humans. Finally, although considerable research has been done to understand the relationships between environmental pollutants and cancer, some important research questions remain. These include the role of exposures to certain classes of chemicals (such as hormonally active agents) during critical periods of human development, and the potential for pollutants to interact with each other as well as with genetic and acquired factors.

While this special section focuses on the relationship between environmental pollutants, particularly air pollutants, and cancer, pollution may adversely affect the health of humans and ecosystems in many other ways. Cancer is only one dimension of environmental health. Environmental pollution may have other serious consequences for human health, including developmental or reproductive toxicity, respiratory and cardiovascular disease. The progress made in reducing exposures to occupational carcinogens and some air and water pollutants in the United States since the 1970s has been substantial, and has contributed to the protection of the public and the preservation of the environment for future generations. It is important that this progress be recognized and sustained.

What Is a Carcinogen?

The term *carcinogen* refers to exposures that can increase the incidence of malignant tumors (cancer). The term can apply to a single chemical such as benzene; fibrous minerals such as asbestos; metals and physical agents such as x-rays or ultraviolet light; or exposures linked to specific occupations or industries (e.g., nickel refining). It is now known that such agents or exposures can increase risks of cancer by acting in several different ways throughout the continuum of molecular and biological events that may eventually result in a tumor.

A carcinogen like ultraviolet light can damage DNA and directly affect the structure or function of critical genes in the genetic material of a cell, either by causing mutations or inactivating or activating genes that control cell differentiation and survival. Some carcinogens, such as estrogens, act by increasing the rate of cell division that, in turn, may increase the odds of survival and proliferation of genetically damaged cells. The genetic material in cells is constantly being damaged and repaired, even in the absence of man-made pollutants because of factors such as inflammation and energy metabolism.[1] Exposure to external carcinogens adds to this background burden and increases the likelihood that some of these errors will not be corrected before the cell divides and no longer recognizes the damage as abnormal.

Exposure to pollutants can result from inhalation of contaminants in air, intake of contaminated food or water, or by absorption following direct contact of skin or mucous membranes. The probability that exposure to

a carcinogen will result in development of a cancer depends on many things. In general, the intensity and duration of exposure strongly influences eventual risk, but the relative importance of these factors, as well as other circumstances that affect risk, may vary by carcinogen and cancer site. For example, the lung cancer risk from cigarette smoking is influenced more by the duration of smoking than by the number of cigarettes smoked per day, whereas the breast cancer risk from high-dose radiation depends mostly on the intensity of exposure as well as the age at exposure.

In this special section, we focus on cancer related to air pollutants. The lung is usually the most important route of entry for airborne pollutants.[2] Once inhaled, pollutants can affect the lung itself, as in the case of tobacco smoke, asbestos, radon, and several metals, such as cadmium and arsenic. Some inhaled carcinogens, however, can also induce tumors in distant organs, such as the liver or bladder, because of the transport of inhaled pollutants throughout the body after absorption through the lung or other organs.

Occupational Exposures

Studies of occupational exposures have yielded important information about cancer risks associated with pollutants. This is because workplace exposures are often considerably higher than those in the general environment and because exposed occupational groups can be clearly identified and followed for long periods of time. Many of the chemicals known to cause cancer in humans were identified through studies of workers. Such studies, for example, identified the carcinogenic risks of arsenic, asbestos, cadmium, chromates, chloromethyl ethers, coke oven emissions, nickel, polycyclic aromatic hydrocarbons, and radon. Two of these substances may pose special hazards for the general public: asbestos and radon.

Asbestos

Asbestos is a group of naturally occurring minerals that contains fibrous silicates that are highly resistant to heat and chemical degradation. Asbestos was widely used in the US and other industrialized countries even after definitive epidemiological evidence of carcinogenicity was published in the 1950s and 1960s. Government regulation ended asbestos production and use in the US after 1980.

Asbestos is an established cause of lung cancer, asbestosis (a serious nonmalignant lung disease), and malignant mesothelioma, a rare cancer that can affect the lining of the chest, abdominal cavity, and covering of the heart. Workers exposed to asbestos in several occupations – including asbestos mining, production, and application of asbestos insulation – have greatly increased risks of all three conditions. In addition, environmental exposures to asbestos, usually resulting from living near an asbestos mine or natural outcropping of asbestos-containing rock, have been linked with mesothelioma.

Nearly all cases of mesothelioma are attributable to asbestos exposure. However, most of the increased cancer cases among workers exposed to asbestos are from lung cancer rather than mesothelioma, and there is a strong interaction between tobacco smoking, asbestos exposure, and lung cancer.[2] (Interaction means that cigarette smokers who are exposed to asbestos have a greater risk of developing lung cancer than would be expected from either smoking or asbestos alone.) Other cancer sites, such as the pharynx, larynx, esophagus, stomach, and colorectum are associated with asbestos exposure in some studies, although the increase in risk is substantially less than for lung cancer and causality is less certain.[3]

Asbestos products remain in most buildings constructed between 1930 and 1975, as well as in older buildings that were repaired or altered prior to 1975. This source of asbestos can present a danger during renovations or demolition, because the asbestos-containing materials (such as insulation) can become a dust and be inhaled. Because of the hazards associated with the continued presence of asbestos, there are federal programs for asbestos abatement. Contractors involved in the removal, enclosure, or encapsulation of asbestos must be specially licensed and must comply with federal, state, and local laws. The US Environmental Protection Agency (EPA) also describes how buildings containing asbestos should be maintained to minimize exposures (see: http://www.epa.gov/asbestos/buildings.html).

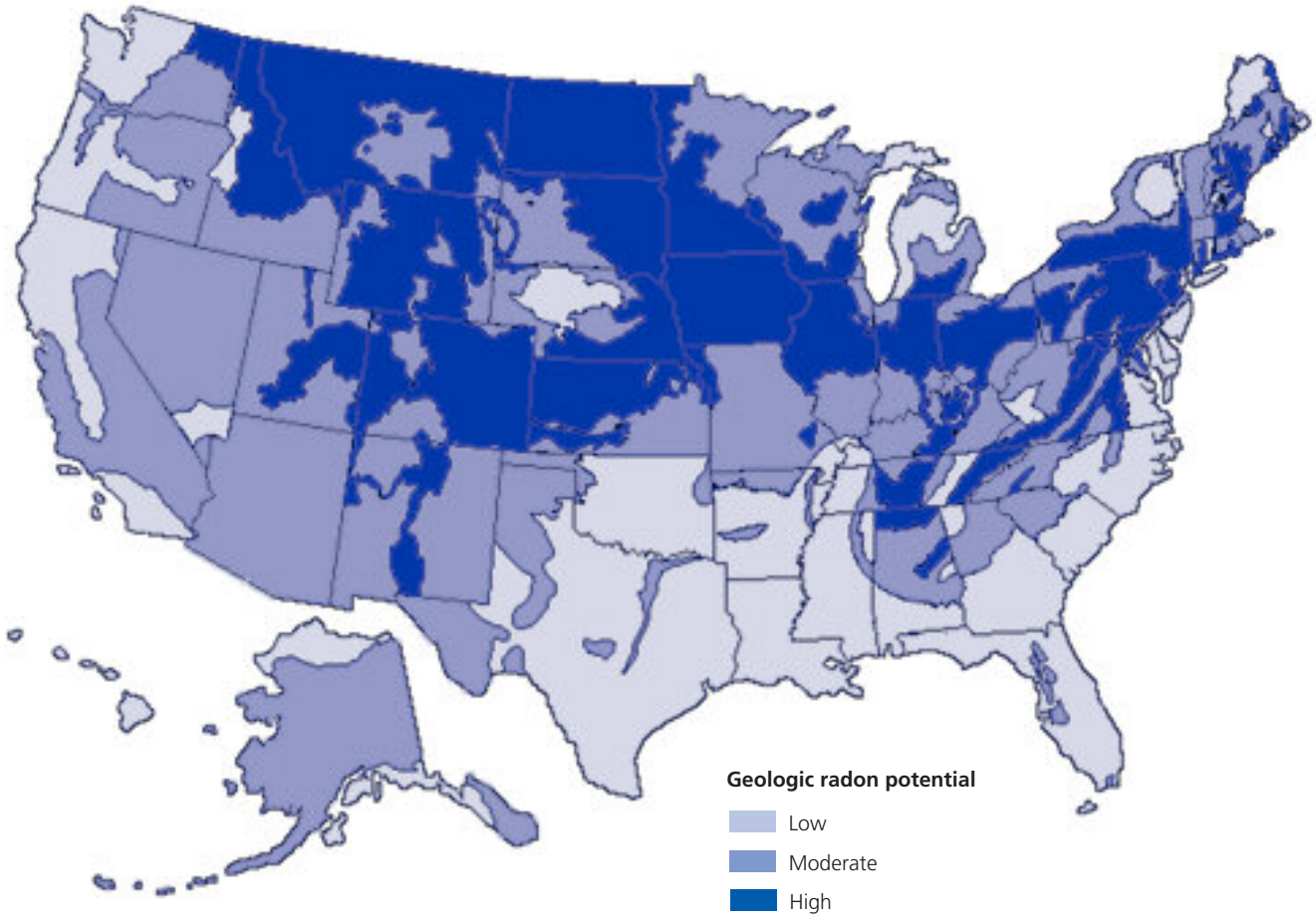
Although use of asbestos has declined in many developed countries, its use continues to present a hazard in other parts of the world. More than 70% of the world's asbestos production is used in Eastern Europe, Latin America, and Asia.[4] Non-occupational (household or residential) sources of exposure to asbestos can arise from dust brought home on the clothing of workers exposed to asbestos-containing products. Residential exposure can also occur from asbestos mining or manufacturing or erosion of asbestos or asbestos-containing rocks.[5]

Radon

Radon is an inert (i.e., not chemically reactive) gas that is produced naturally from radium in the decay of uranium. Two of the decay products of radon (called radon daughters) emit α particles when they decay. Unlike x-rays, α particles cannot penetrate very far into human tissue, but can cause local damage to tissues because of their high energy and mass. When radon daughters are inhaled and undergo nuclear decay in the lung, the α particles can damage genetic material in the cells lining the lung, potentially resulting in lung cancer.[2] Since uranium occurs in soil and rock throughout the world, radon exposure is universal. The level of uranium present in rock and soil, however, varies considerably throughout the US (Figure 1) and the world. Once produced, radon can diffuse through cracks in walls and presents a hazard if it accumulates in basements and unventilated underground spaces. It was found to cause lung cancer in studies of underground miners who were exposed to very high concentrations. Studies of miners provide good information on the relationship between increasing radon exposure and lung cancer risk at high levels of exposure.[6]

Average radon concentrations in the indoor air of most homes are considerably lower than those in uranium mines, although this varies widely.[2] Within buildings, radon levels are usually highest in the basement. In regions where radon emissions from soil are higher, houses with poorly ventilated basements can yield substantial exposures. The EPA sets recommended levels for maximum concentration of radon in homes, and provides information about how it is measured and how levels can be reduced (for more information, see: http://www.epa.gov/radon/pubs/consguid.html). The EPA standard was initially set based on extrapolation from high occupational exposures to the generally much lower residential exposures. Researchers have subsequently confirmed the reliability of these risk

Figure 1. Generalized Geologic Radon Potential of the United States



Source: US Geologic Survey: http://energy.cr.usgs.gov/radon/usrnpot.gif

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estimates by combining the results of multiple studies of indoor radon exposures. The observed risk of lung cancer from indoor radon exposures approximates that which was predicted by studies of uranium miners exposed at much higher doses.

In 1998, the Biological Effects of Ionizing Radiation (BEIR VI) Committee of the National Research Council estimated that between 10% and 14% of lung cancer deaths in the US could be attributed to radon.[7] Most of the radon-related lung cancers occur among smokers, although an estimated 2,100-2,900 of the 11,000 deaths from lung cancer among nonsmokers in the US each year are estimated to be radon-related.

Indoor Air Pollution

Indoor air pollution (exposures that occur inside buildings) results from pollutants that enter buildings from ambient or outside air, as well as pollutants that may result from interior sources, such as stoves, heating, products used in building materials and furnishings, and ventilation systems. Thus, depending upon the pollutant, its concentration in residential or office settings may be higher or lower than outdoor concentrations. Because of the large amount of time Americans spend indoors, virtually the entire population is exposed.

Some indoor air contaminants are known to increase cancer risk, including radon and secondhand tobacco smoke. Exposure to deteriorating insulation or other asbestos-containing products can disperse fibers in the home. Other sources of pollution include combustion of oil, gas, kerosene, coal, wood, and tobacco products; building materials and furnishings, products used for household cleaning, maintenance, and pest control; and chemical and microbial contaminants from central heating and cooling systems and humidification devices.[8] Most indoor contaminants are not associated with cancer.

Secondhand tobacco smoke

Secondhand exposure to tobacco smoke affects nonsmokers in environments where others are smoking. Secondhand smoke is a complex mixture that contains both sidestream smoke – the material emitted from smoldering tobacco products between puffs – and exhaled mainstream smoke. It contains most of the 4,000 chemicals and at least 50 known carcinogens generated during active smoking, although at lower concentrations.[9]

Numerous studies have documented increased risk of lung cancer among nonsmokers exposed to secondhand smoke at work and at home. An analysis combining results of 46 epidemiological studies of spousal exposure and 19 studies of workplace exposure estimated that the relative risk for lung cancer was 1.24 for a nonsmoking woman exposed to her husband's smoking and 1.16 for a nonsmoking woman exposed in the workplace compared to a nonsmoking woman with no exposure to secondhand smoke.[10] Secondhand smoke exposure at home or at work is also associated with an increased risk of death from heart disease. It was recently estimated that exposure to secondhand smoke causes approximately 35,000 heart disease deaths and 3,000 lung cancer deaths among nonsmokers in the United States every year.[11]

Implementing policies that establish smoke-free environments is the most effective way to reduce secondhand smoke exposure among nonsmokers. Fifteen states and numerous municipalities now restrict public smoking in restaurants, workplaces, and/or bars. (For more information on secondhand smoke, see page 38.)

Burning of solid fuel for heating and cooking

In some countries, a major source of indoor air pollution is wood or other fuel used for heating and cooking. Very high lung cancer rates have been observed among nonsmoking women in regions of China where unventilated indoor coal stoves traditionally have been used for cooking and heating.[5] Combustion products of coal include fine particulates, sulfur dioxide, arsenic, and polycyclic aromatic hydrocarbons.

Fumes from cooking oil are another source of indoor air pollution.[12] Burning of crude biomass fuels, such as crop residues, animal dung, and wood, for cooking and heating has not been associated with lung cancer, but results in high levels of indoor air pollution and increased risk for acute respiratory infections and chronic obstructive pulmonary disease.[8]

Outdoor Air Pollution

Outdoor (ambient) air pollution is a highly complex and variable mixture. Exposures to ambient air pollution at current levels in the US and other countries have been associated with numerous effects on health, including respiratory problems, premature mortality, hospitalization for heart and nonmalignant lung diseases, and lung cancer. The sources of pollutants in ambient air include such mobile sources as cars, buses, planes, trucks, and trains. Large stationary sources of air pollution include factories, fossil-fuel-powered electrical generating plants, incinerators, recycling facilities, and metal

smelting. Some smaller but still significant sources include dry cleaning processes and degreasing operations. In addition, there are important natural sources such as windblown dust storms and wildfires.[13]

As the federal entity charged with primary responsibility for regulation of air pollutants, the EPA has set national air quality standards for six principal air pollutants (also called the criteria pollutants): nitrogen dioxide, ozone, sulfur dioxide, particulate matter, carbon monoxide, and lead. However, many urban areas in the US fail to meet EPA's air quality standards for ozone and/or particulate air pollution (Figure 2).[13] A separate set of EPA regulations pertains to hazardous air pollutants, or air toxics.

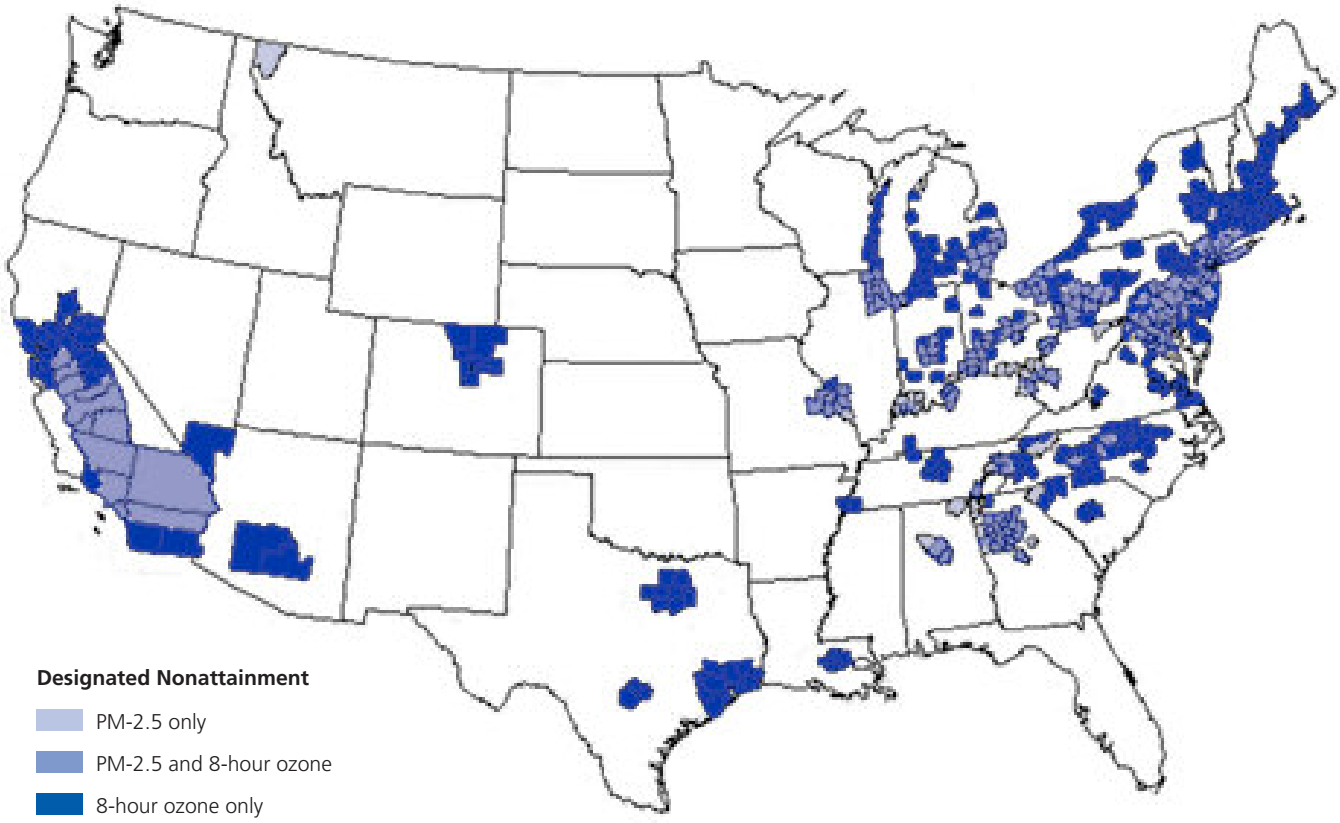
Particulate air pollution

Particulate matter (PM) is a general term for a mixture of solid particles and liquid droplets found in the air. PM is defined by size, with some particles large enough to be seen as dust or dirt, while others are so small that they can be seen only with an electron microscope. Only particulates which are less than or equal to 10 micrometers (um) in diameter (about one-seventh the diameter of a human hair) are small enough to be inhaled. These particulates are further subdivided into fine particulates, with a diameter of 2.5 um or less, and coarse particulates, with a diameter of 2.5 to 10 um. Exposure to fine particulates appears to be more related to cancer than exposure to coarse particulates.

In many industrialized countries, motor vehicle exhaust represents the most widespread source of air pollution, including ozone-producing volatile organic chemicals (VOCs) and fine particulates.[14] Coal-fired power plants are an important source of sulfur dioxide, and the byproducts of sulfur dioxide are a major component of fine air particles in the Eastern United States.[13]

American Cancer Society epidemiologists have collaborated with air pollution experts to assess the health

Figure 2. Counties That Fail to Meet EPA Standards for PM-2.5 and/or 8-Hour Ozone



Several counties have only a portion of their county designated nonattaiinment. These counties are represented as whole counties on the map.
Source: US Environmental Protection Agency: http://www.epa.gov/oar/oaqps/greenbk/mappm25o3.html

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effects of exposure to outdoor air pollution in the Society's Cancer Prevention Study II (CPS II) cohort. The first study, published in 1995, examined information about air pollution levels for 151 US metropolitan areas in which study participants were residing, as well as information about individual risk factors, such as cigarette smoking.[15] The study found that people living in the most polluted areas had higher death rates from all causes, nonmalignant heart and lung disease, and lung cancer than those in the least polluted areas. Results of this study contributed to the decision by the US EPA to issue more stringent standards in 1997, reducing concentrations of pollutants, including the first standard for fine particulates.

An updated and expanded analysis of the ACS study was published in 2002.[16] This study estimated that each 10 ug/m^3 elevation in fine particulate air pollution (PM2.5) was associated with a 4%, 6%, and 8% increased risk of all-cause, heart and lung disease, and lung cancer mortality, respectively, although the lung cancer estimates are the least precise.[17]

Ozone

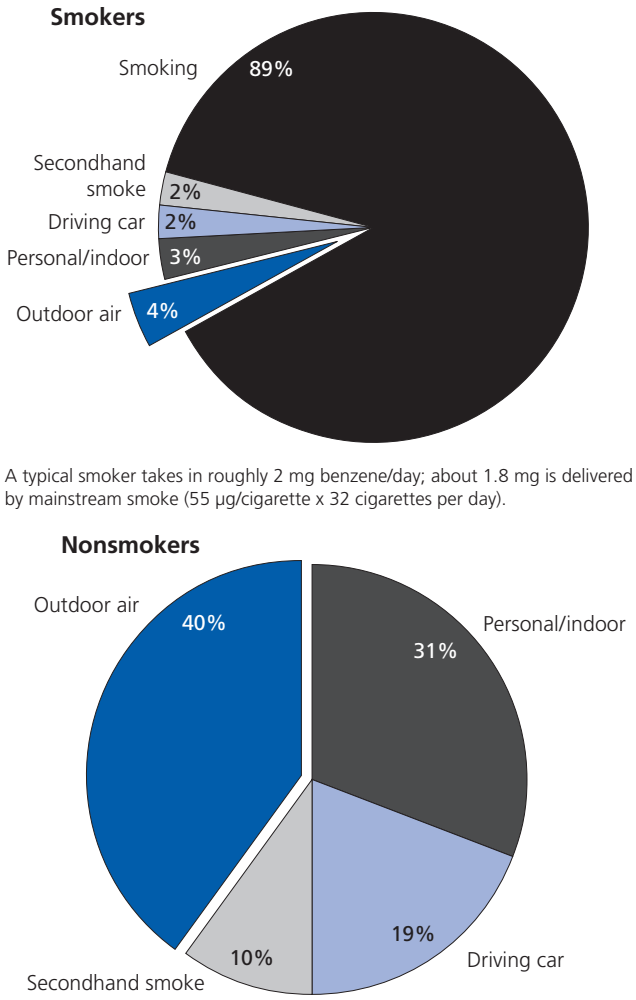
Ground-level ozone is the main chemical component of "smog." Ozone is formed by the reaction of volatile organic compounds (VOCs) and nitrogen oxides in the presence of heat and sunlight. These are most often produced by the incomplete combustion of gasoline and other fuels, and are thus highly associated with traffic. Ozone exposure has not been linked to cancer. However, exposure to high levels of ozone has been linked to respiratory diseases as well as damage to vegetation and ecosystems.

Hazardous air pollutants

In addition to the six criteria air pollutants mentioned above, there are additional toxic pollutants which the EPA regulates. Air toxics are released from motor vehicles and other sources. For example, when a car is refueled, gases escape from liquid gasoline and form a vapor in a process called vaporization or evaporation. To reduce such releases and exposures, gas stations are now required to install vapor recovery systems at the pump. Air toxics are also released from stationary sources, such as oil refineries, dry cleaners, and auto paint shops.

Some of the 188 substances on the list of air toxic pollutants have been found to cause cancer in animals or humans. Benzene has long been recognized as a human carcinogen because of strong evidence that high levels of occupational exposure increase risk of leukemia. Benzene is derived from gasoline combustion and other combustion sources in the environment. Elevated benzene exposures occur in proximity to certain industries, such as oil refineries, as well as in the homes of smokers, the interiors of automobiles and buses, and in the vicinity of gasoline stations and heavily traveled highways.[17] The overwhelming source of benzene exposure for smokers is cigarette smoke. Smokers have an average benzene body burden about 6 to 10 times that of nonsmokers and receive about 90% of their benzene exposure from smoking (Figure 3).[17]

Figure 3. Sources of Benzene Exposure



A typical smoker takes in roughly 2 mg benzene/day; about 1.8 mg is delivered by mainstream smoke (55 µg/cigarette x 32 cigarettes per day).

A typical nonsmoker inhales about 0.2 mg benzene/day, assuming an average exposure of 15 µg/m^3 and an alveolar respiration rate of 14 m^3/day. Outdoor air contributes about 40% of that amount, assuming an average outdoor level of 6 µg/m^3. The remaining 9 µg/m^3 are split between driving (100 min. at 30-40 µg/m^3), indoor sources such as automobile vapor emissions in attached garages or storage of gasoline or kerosene in the garage or the basement, and environmental tobacco smoke exposures at home or work.

Source: EPA TEAM Studies.[17]

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Although epidemiologic studies do not provide direct evidence that exposure to benzene at levels present in urban air, motor vehicles, or gasoline stations increases the risk of leukemia, benzene in cigarette smoke is thought to be a factor in the increased relative risk of leukemia in smokers.[18]

A number of studies have investigated whether the proximity of residential neighborhoods to busy roads or gasoline stations increases risk of childhood leukemia, but results have been inconsistent.[19] Studies of leukemia in adults with occupational exposure to gasoline fumes and motor vehicle exhaust have generally not found elevated risks. For many years, estimates of leukemia risk associated with low levels of benzene exposure had a wide range of uncertainty because the only exposure-response data in humans came from a single study of 1,136 men with very high exposures.[20] More recently, a study of a large (more than 70,000) occupationally exposed population in China demonstrated an increased risk of leukemia at lower levels of exposure, including exposure at the current US occupational standard – one part per million (1 ppm) – for 40 years.[21] A separate study in China found decreased numbers of white blood cells (an indication of toxicity to blood and bone marrow) associated with shorter term (1-2 years) exposures as low as 1 ppm.[22] Average benzene concentrations in outdoor air are considerably lower, in the range of 0.0003 to 0.006 ppm.[17]

Diesel particulate matter and diesel exhaust from internal combustion engines are also listed by the EPA as hazardous air pollutants. Diesel fuel has been used in cars, trucks, locomotives, and other mechanized machinery for over 100 years. It is estimated that approximately 1.4 million US workers are exposed to diesel exhaust through their occupation, and it is also a component of urban air pollution.[23] High levels of diesel particulate exposure produce lung cancer in rats, although some argue that this may be due to very high exposures overwhelming the mechanisms that clear foreign substances from the lung. Research is continuing on this issue.

Most studies of occupational groups with high exposures, such as truck drivers and railroad workers, find an elevated risk of lung cancer. The potential carcinogenicity of diesel exhaust has been reviewed by the US National Toxicology Program and classified as "reasonably anticipated to be a human carcinogen."[23] Measures to reduce diesel emissions from mobile sources include use of low sulfur fuel, pollution controls on vehicles, reduced idling times, and use of alternative fuels, such as compressed natural gas.[24]

The EPA takes a two-phase approach to reducing emissions of air toxics from large sources of air pollution.[25] The first phase is a "technology-based" approach, where EPA develops standards for controlling the emissions of air toxics from a particular industry (or "source category"). These Maximum Achievable Control Technology (MACT) standards are based on emission levels that are already being achieved by the better controlled and lower emitting sources in an industry. In the second phase, EPA is required to assess the remaining health risks from each source category within 8 years of setting the MACT standards to determine whether the MACT standards appropriately protect public health. Using this "risk-based" approach, EPA must make a determination whether more health-protective standards are necessary.

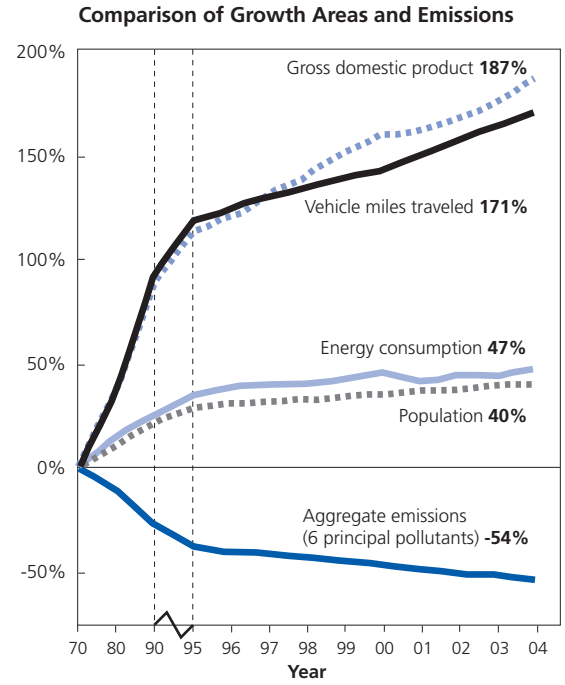
Progress in Reducing Air Pollution

Since 1970, considerable progress has been made in controlling air pollution in the US. For example, the aggregate emissions of the six principal air pollutants decreased 54% between 1970 and 2004, while gross domestic product increased 187%, vehicle miles traveled increased 171%, energy consumption increased 47%, and population increased 40% (Figure 4). Nonetheless, in 2002, 146 million people lived in counties with pollution levels above at least one of the National Ambient Air Quality Standards.[13]

Stratospheric ozone

While ground-level ozone is harmful to health, stratospheric ozone (located 6 to 30 miles above the earth) protects living organisms from harmful ultraviolet radiation (UV-B) from the sun. Certain chemicals emitted from commercial air conditioners, refrigerators, insulating foam, and some industrial processes can cause this protective ozone layer to break down, increasing the amount of UV-B radiation that reaches the earth's surface. In humans, UV-B radiation is linked to skin cancer, including melanoma. In 1989, the US and 28 other countries signed the Montreal Protocol, a treaty that recognized the international nature of ozone depletion and committed the world to limiting the production of ozone-depleting substances. Today, more than 180 nations have signed the protocol. In the US, the Clean Air Act Amendments established a US regulatory program to protect the stratospheric ozone layer, and in January, 1996, US production of many ozone-depleting

Figure 4. Air Emission Trends, 1970-2004



Source: US Environmental Protection Agency: http://www.epa.gov/airtrends/2005/econ_emissions.html.

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substances, including chlorofluorocarbons, carbon tetrachloride, and methyl chloroform, virtually ended.[13]

How Carcinogens Are Identified

Carcinogens are usually identified on the basis of epidemiologic studies or by testing in animals. Studies of occupational groups (cohorts) have played an important role in understanding many chemical carcinogens as well as radiation because, as noted above, exposures are often higher among workers and they can be followed for long periods of time. Some of our information has also come from studies of persons exposed to carcinogens during medical treatments (such as radiation and estrogen), as well as from studies conducted among individuals who experienced large, short-term exposure to a chemical or physical agent due to an accidental or intentional release (such as survivors of the atomic bomb explosions of Hiroshima and Nagasaki).

Important epidemiologic studies have been done to examine the relationship between exposure to potentially carcinogenic substances in the general population and risk of cancer, but such studies are much more difficult, often because of uncertainties about exposure and the challenge of long-term follow up. Moreover, relying upon epidemiological information to determine cancer risks does not fulfill the public health goal of prevention. Thus, for the past 40 years, the US and many countries have developed methods for identifying carcinogens through animal testing using the "gold standard" of a 2-year or lifetime bioassay in rodents. This test is expensive and time consuming, but it can allow us to take prudent public health actions prior to collecting data from exposed populations.

Many substances that are carcinogenic in rodent bioassays have not been adequately studied in humans, usually because an acceptable study population has not been identified. Among the substances that have proven carcinogenic in humans, all have shown positive results when tested in well-conducted 2-year bioassays.[26] Moreover, between 25% and 30% of established human carcinogens were first identified through animal bioassays. Since animal tests necessarily use high-dose exposures, in most cases, human risk assessment requires extrapolating the exposure-response relationship observed in rodent bioassays to predict effects in humans at lower doses. Typically, regulatory agencies in the United States and abroad have adopted the default assumption that no threshold level (level below which there is no increase in risk) of exposure exists for carcinogenesis.

Evaluation of Carcinogens

The National Toxicology Program (NTP) plays an important role in the identification and evaluation of carcinogens in the US, and the International Agency for Research on Cancer (IARC) plays a similar role internationally.

The National Toxicology Program was established in 1978 to coordinate toxicology testing programs within the federal government, including tests for carcinogenicity. The NTP is also responsible for producing the *Report on Carcinogens*, an informational scientific and public health document that identifies agents, substances, mixtures, or exposure circumstances that may increase the risk of developing cancer.[27] For a list of substances listed in the *11th Report on Carcinogens* as known or reasonably anticipated to be human carcinogens, see: http://ntp.niehs.nih.gov/ntp/roc/toc11.html.

The International Agency for Research on Cancer is a branch of the World Health Organization that regularly convenes scientific consensus groups to evaluate the carcinogenic potential of chemicals. After reviewing published data from laboratory, animal, and human research, these committees reach consensus about

whether the evidence should be designated "sufficient," "limited," or "inadequate" to conclude that the substance is a carcinogen. For a list of substances that have been reviewed by the IARC monograph program, see: http://www-cie.iarc.fr/.

Although the relatively small risks associated with low-level exposure to carcinogens in air or water are difficult to detect in epidemiologic studies, scientific and regulatory bodies throughout the world have accepted the principle that it is reasonable and prudent to reduce human exposure to substances shown to be carcinogenic at higher levels of exposure.[27]

What Is the American Cancer Society's Role?

Cancer prevention is central to the mission of the American Cancer Society that states:

"The American Cancer Society is the nationwide, community-based voluntary health organization dedicated to eliminating cancer as a major health problem by preventing cancer, saving lives, and diminishing suffering from cancer through research, education, advocacy, and service." With respect to cancer prevention, the major focus of the Society's efforts is to reduce the use of tobacco products, promote a healthy diet and physical activity, and encourage the use of screening tests that may prevent cancer or detect it at an early, treatable stage because the Society believes that these activities have the greatest potential to reduce the burden of cancer in our lifetimes.

Although the Society does not play a direct role in the identification and classification of carcinogens, it does provide information and guidance on environmental cancer risks. For example, the Society provides summary information on its web site about topics related to environmental pollutants, and directs readers who want more detailed information to other sources. In the legislative arena, advocating for smoke-free legislation to reduce indoor air pollution is an important priority for the American Cancer Society. The Society has also supported national and state laws, such as the Clean Air Act and particulate matter standard, designed to safeguard the public from cancer-causing substances in outdoor air and water. The Society has established an Environmental Advisory Committee which convenes twice a year to provide advice on scientific and policy issues related to environmental pollution.

The Society's Divisions are also responsive to local concerns. In some Divisions, issues related to environmental pollution are of high interest to constituents, and the Division receives frequent requests to participate in coalitions or endorse state legislation. The California Division has established a Cancer and the Environment Workgroup, with the support of the National Home Office, to provide advice on these issues.

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