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## *Airborne Toxic Pollutants*

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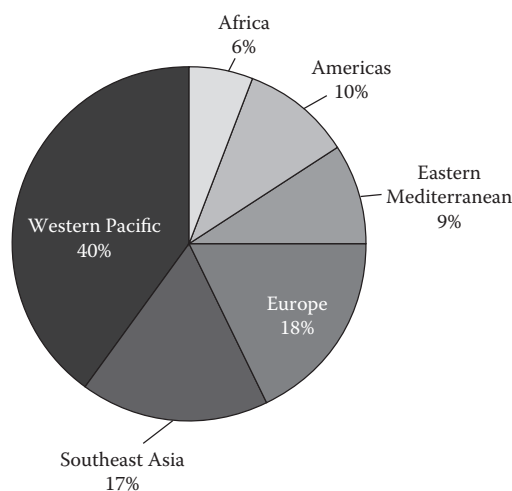
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### Introduction

Most of the deaths linked to environmental exposure are attributed to air pollution (World Health Organization [WHO]/IER 2009). Atmospheric emissions are theoretically able to disperse maximally when in gaseous state. This is a property that has been put to use in chemical warfare using sarin (Murakami 2003) or pentafluorophenylarsenic oxide among many other toxic war gases (Rettenmeier 2004). However, the atmosphere is not a perfect gas, and precipitation, wind, and topography lead to a heterogeneous distribution of both pollutants in the air and deposition.

More than 2 million premature deaths in 2008 were due to indoor smoke from solid fuels and 1.34 million deaths were attributed to urban outdoor air pollution (Figure 12.1). The main causes of death by outdoor air pollution are cardiopulmonary failure, lung cancers, and respiratory infections (WHO 2011c). Several of the air pollutants may occur naturally, but most are the result of human activities. Fine particle pollution often originates from



**FIGURE 12.1**

Percentage of deaths due to outdoor air pollution in 2008 by region. (Data calculated from World Health Organization [WHO], 2011, Public health and environment: Outdoor air pollution, number of deaths, 2008. [http://gamapserver.who.int/gho/interactive\\_charts/phe/oap\\_mbd/atlas.html](http://gamapserver.who.int/gho/interactive_charts/phe/oap_mbd/atlas.html).)

combustion sources such as power plants and motor vehicles. The great majority of urban populations have an average annual exposure to PM10 particles (diameter  $<10\ \mu\text{m}$ ) in excess of the WHO Air Quality Guidelines recommended maximum level of  $20\ \mu\text{g}/\text{m}^3$  (WHO 2011d).

### Exposure to Pollutants

Air pollutants can enter the human body through the skin and mucosal tissues (including the mouth) from different environmental sources: water, soil, gases, dust, and radiation, as well as the food chain (Florea et al. 2004; Stoltenburg-Didinger 2004). Similarly, their bodily storage, metabolism, and excretion may follow different paths (Hirner et al. 2004) and cause interactions (Fender and Wolf 2004; Hartwig et al. 2004; Stoltenburg-Didinger 2004). Hitherto, environmental monitoring and follow-ups of human subjects have seldom factored in this diversity of paths and interactions of mixtures. This likely has resulted in underestimates of exposure; the most regrettable consequence is probably that damages from this exposure may be attributed to nonenvironmental risk factors such as individual frailty and behavior. Of particular concern is exposure in the working environment and added exposures from outdoor and indoor air pollution.

Toxicity, or the amount needed for damage to occur at the organism, cell, or molecular levels, is measured as concentration or dose of a toxicant (micrograms per deciliters [ $\mu\text{g}/\text{dl}$ ] of blood, nanogram [ $\text{ng}$ ] or microgram

per kilogram [ $\mu\text{g}/\text{kg}$ ] of bodily mass). Inhibition of biological processes and lethality are the most widely used indicators of toxicity and are often summarized as  $\text{LC}_{50}$  (lethal concentration affecting 50% of the population under study), on account of variation in resistance among individual cells or organisms. Responses to acute and chronic exposures may differ considerably. Lead acute effects are localized in the cerebellum, whereas chronic effects are a subtle axodendritic disorganization in the hippocampus leading to hyperactivity, aggression, and seizures (Stoltenburg-Didinger 2004). There are other factors that make the dose-response vary such as age (mainly <5 years or >80 years), physical condition or genetic problems, and interactions of different compounds.

Health effects are changes in an individual or population, identifiable either by clinical diagnosis or statistical epidemiological methods. Risk analysis is a process that incorporates three components: risk assessment, risk management, and risk communication. The assessment of human health risk requires identification, compilation, and integration of information on the health hazards of a chemical; human exposure to the chemical; and relationships among exposure, dose, and adverse effects (WHO 2010b). The description of these steps is presented in Table 12.1; however, multiple exposures to different pollutants may alter the exposure (International Programme on Chemical Safety [IPCS] 2004). For example, in rural areas usually it is possible to find a mix of compounds such as cooking fuel,

**TABLE 12.1**

Risk Assessment Process

Step	Description	Content
Hazard identification	Identifies the type and nature of adverse health effects	Human studies Animal-based toxicology studies <i>In vitro</i> toxicology studies Structure–activity studies
Hazard characterization	Qualitative or quantitative description of inherent properties of an agent having the potential to cause adverse health effects	Selection of critical data set Modes/mechanisms of action Kinetic variability Dynamic variability Dose–response for critical effect
Exposure assessment	Evaluation of concentration or amount of a particular agent that reaches a target population	Magnitude Frequency Duration Route Extent
Risk characterization	Advice for decision making	Probability of occurrence Severity Given population Attendant uncertainties

Source: World Health Organization (WHO) (2010b), WHO human health risk assessment toolkit: Chemical hazards, IPCS harmonization project document No. 8, Geneva: WHO.

herbicides, pesticides, fertilizers, and combustion of vegetation and garbage that may generate a combination of symptoms in the population.

## **Main Air Pollutants**

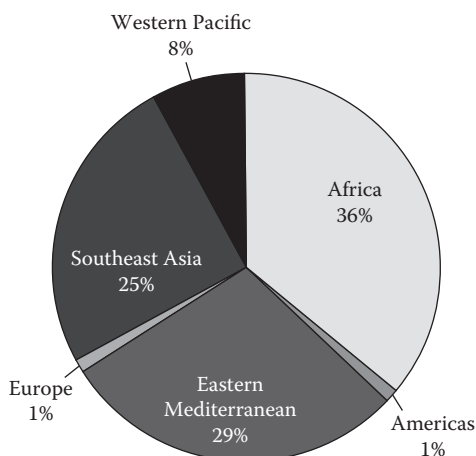
### ***Aerosols and Particulate Matter***

Recently, the difference between aerosols in global change studies and particulate matter in urban and outdoor air pollution research has vanished. There is now evidence of the global atmospheric circulation as a factor in dispersing and concentrating toxic pollutants. This is clearly demonstrated by aviation, which accounts for 8000 premature deaths per year (1% of all air pollution deaths and 10% of all deaths linked to particulate matter inter-continental transport). Due to atmospheric circulation, the United States incurs 7 times fewer casualties than expected from their aircraft emissions, whereas India's mortality is sevenfold the amount attributable to its aircraft emissions. India and China emit 2% and 8% of global aviation emissions, respectively, but combined represent 35% of aviation-attributable deaths. Long-term exposure to black carbon and particulate organic carbon less than 2.5  $\mu\text{m}$  in diameter (also called PM<sub>2.5</sub> or fine particulate matter) are deemed responsible (Barrett et al. 2010). The PM<sub>2.5</sub> particles are more dangerous since, when inhaled, they may reach the peripheral regions of the bronchioles and interfere with gas exchange inside the lungs (WHO 2011a).

PM<sub>10</sub> can penetrate into the lungs and the bloodstream, can cause heart disease, lung cancer, asthma, and acute lower respiratory tract infections (WHO 2011d). The major components of PM are sulfate, nitrates, ammonia, sodium chloride, carbon, mineral dust, and water suspended in the air (WHO 2011a).

Urban air pollution is mostly derived from fossil fuel combustion sources releasing a complex mixture of thousands of compounds to the atmosphere, which includes carcinogens such as benzo(a)pyrene, benzene, 1,3-butadiene, and lead. Considering 3200 cities with more than 100,000 inhabitants around the globe, urban PM air pollution causes 3% of cardiopulmonary disease in adults; 5% of cancers of trachea, bronchus, and lung; and 1% of acute respiratory infections in children 4 years old and younger. This is equivalent to 0.80 million premature deaths (1.4% of the global total) and occurs largely in the western Pacific Rim, Southeast Asia, and eastern Europe. Energy use is held responsible for particulate matter emissions and an associated 14,000 preventable deaths in Europe (Bollen et al. 2009). Similarly, mortality in Latin American cities such as Mexico City and Sao Paulo increase in response to higher PM concentrations (Cohen et al. 2004).

Age-wise, almost half the premature deaths (1 million) by indoor air pollution are due to pneumonia in children under 5 years of age. Exposure is difficult to pinpoint as the sensitivity of children and their number differ across regions in response to nutrition and health conditions and fertility trends; these differences in the populations of children are compounded in

**FIGURE 12.2**

Percentage of deaths of children <5 years old due to outdoor air pollution in 2008 by region. (Data calculated from World Health Organization [WHO], 2011, Public health and environment: Outdoor air pollution, number of deaths, 2008. [http://gamapserver.who.int/gho/interactive\\_charts/phe/oap\\_mbd/atlas.html](http://gamapserver.who.int/gho/interactive_charts/phe/oap_mbd/atlas.html).)

the regional pattern of deaths in children compared to the general regional patterns of deaths due to outdoor air pollution (Figure 12.1 and Figure 12.2). There are international, national, and local institutions that have established different guideline values to limit the air pollutant emissions in various parts of the world. The WHO Air Quality Guidelines represent the most widely accepted and up-to-date assessment of health effects of air pollution and recommends targets to reduce health risks. However, exposure to air pollutants is largely beyond the control of individuals and requires public support at the local, national, and international levels (WHO 2011a).

Heavy industries illustrate the link between global atmospheric change emissions and toxic air pollutants. The cement industry atmospheric emissions include but are not limited to benzene, tin, chromium, lead, chlorine, mercury, cadmium, and nonmethane organic volatile compounds. A salient feature of emission inventories is the heterogeneity with which different cement plants declare pollutants (Chen et al. 2010). This complicates the estimates of global pollution and personal exposure.

Notable among PM<sub>10</sub> particles are the carcinogenic polycyclic aromatic hydrocarbons (PAHs), which have been the focus of several molecular epidemiological investigations. Several studies of populations exposed to PAHs showed increased levels of several markers of genotoxicity, including DNA adducts, chromosome aberrations, sister chromatid exchanges, and *ras* oncogene overexpression. These results indicate that at least in some regions PAHs are a major source of the genotoxic and embryotoxic activities of organic mixtures associated with air pollution. In addition to the formation of DNA

damage by exogenous exposures to carcinogens, such as PAHs, it is known that DNA is modified, often oxidatively, by radicals (Farmer et al. 2003).

### **Ozone ( $O_3$ )**

Tropospheric  $O_3$  is one of the major constituents of photochemical smog. It is formed by the reaction with sunlight of pollutants such as nitrogen oxides ( $NO_x$ ) and volatile organic compounds (VOCs) emitted by vehicles, solvents, and industry. The highest levels of ozone pollution occur during periods of sunny weather (WHO 2011a). European studies have reported that the daily mortality rises by 0.3% and heart diseases by 0.4% per  $10 \mu\text{g}/\text{m}^3$  increase in ozone exposure. The main effects of ozone and other pollutants are described in [Table 12.2](#).

### **Volatile Organic Compounds**

Volatile organic compounds (VOCs) can generally be understood as solvents containing carbon that evaporate easily at  $20^\circ\text{C}$ , such as used in dry cleaning and painting; in the plastic coating industries; in adhesives, inks, and printing; in the footwear industry, and in vegetal and animal fat processing. They contribute to the formation of photochemical oxidants such as  $O_3$  and include carcinogens, mutagens, or agents toxic to human reproduction (Council of the European Union 1999). VOCs are one of the reportable categories of criteria pollutants in the United States, along with  $PM_{10}$ ,  $PM_{2.5}$ , and Pb (U.S. Environmental Protection Agency [EPA] 2008).

### **Nitrogen Dioxide ( $NO_2$ )**

$NO_2$  is a toxic gas, highly reactive, oxidant, and corrosive. The primary sources are combustion processes in vehicles, gas stoves, vented appliances with defective installations, as well as welding and tobacco smoking.  $NO_2$  acts mainly as an irritant affecting the eyes, nose, throat, and respiratory tract. Extremely high-dose exposure to  $NO_2$  may result in pulmonary edema and diffuse lung injury. Continued exposure to high  $NO_2$  levels can contribute to the development of acute or chronic bronchitis. Low  $NO_2$  exposure may cause increased bronchial reactivity in some asthmatics, decreased lung function in patients with chronic obstructive pulmonary disease, and increased risk of respiratory infections, especially in young children (EPA 2012a).

### **Sulfur Dioxide ( $SO_2$ )**

$SO_2$  is a highly reactive colorless gas. It can be oxidized to sulfur trioxide, which in the presence of water vapor is readily transformed to sulfuric acid aerosol.  $SO_2$  is a precursor of sulfates, which are one of the main components of respirable particles in the atmosphere. It also damages trees and crops.

**TABLE 12.2**

## Source and Effects of Selected Air Pollutants

Pollutant and Sources	Harmful Effects in Humans
Particulate matter (PM): PM10 and PM2.5—Indoor air pollution due to biomass fuel and coal for heating and cooking.	Premature death by pneumonia in children <5 years of age, and deaths by chronic obstructive pulmonary disease (e.g., chronic bronchitis) among adults. Risk of acute lower respiratory infections and associated mortality among young children. Major risk factor for chronic obstructive pulmonary disease and lung cancer among adults. Asthma, cataracts, stroke, tuberculosis, adverse pregnancy outcomes, in particular low birth weight, ischemic heart disease, interstitial lung disease, and nasopharyngeal and laryngeal cancers (WHO 2006, 2010a, 2011a, 2011b).
Particulate matter (PM): PM10 and PM2.5. Black carbon—Rural and urban outdoor air pollution, generated mainly from motor transport, small-scale manufacturers and other industries, burning of biomass and coal for cooking and heating, as well as coal-fired power plants. Residential wood and coal burning for space heating, especially in rural areas in colder months. PM can also be generated by mechanical grinding processes during industrial production, and by natural sources such as natural wind-blown dust.	Chronic exposure to particles contributes to the risk of developing cardiovascular and respiratory diseases, as well as lung disease. PM2.5 are more dangerous since, when inhaled, they may reach the peripheral regions of the bronchioles, and interfere with gas exchange inside the lungs. Animal data suggest that exposure to chemicals including tributyltin, bisphenol A, organochlorine and organophosphate pesticides, lead, diethylstilbestrol, perfluorooctanoic acid, monosodium glutamate, and nicotine can lead to altered cholesterol metabolism and weight gain later in life. In rodents also, pesticides and air pollutants have been shown to contribute to diabetes-related effects following adult exposures. Similarly, adult male rats exposed to fine particulate matter (PM2.5) in conjunction with a high-fat diet developed insulin resistance. Allergies, asthma, and airway disorders, endometriosis, and autoimmune thyroid disease in humans may have roots in endocrine disrupting chemical exposure (WHO 2011b; UNEP/WHO 2013).
Ozone (O <sub>3</sub> )—Vehicles, solvents, and industry.	Breathing problems, triggered asthma, reduced lung function, lung and heart diseases, and mortality. Increased incidence of type 1 diabetes (WHO 2011a; UNEP/WHO 2013).
Nitrogen dioxide (NO <sub>2</sub> )—Combustion processes (heating, power generation, and engines in vehicles).	Inflammation of the airways. Symptoms of bronchitis in asthmatic children increase in association with long-term exposure. Reduced lung function (WHO 2011a).

*Continued*

TABLE 12.2 (Continued)

## Source and Effects of Selected Air Pollutants

Pollutant and Sources	Harmful Effects in Humans
Sulfur dioxide (SO <sub>2</sub> )—Burning of fossil fuels (coal and oil) and the smelting of mineral ores that contain sulfur. Burning of sulfur-containing fossil fuels for domestic heating, power generation, and motor vehicles.	Affects the respiratory system and the function of the lungs, and causes irritation of the eyes. Irritation of the respiratory tract causes coughing, mucus, secretion, aggravation of asthma and chronic bronchitis and makes people more prone to infections of the respiratory tract. Cardiac disease and mortality. Increases the incidence of type 1 diabetes. People with asthma or chronic lung or heart disease are the most sensitive to SO <sub>2</sub> (WHO 2011a; Ontario Ministry of the environment 2010; UNEP/WHO 2013).
VOCs—DEHP-phthalate, formaldehyde, indoor PVC-phthalate release.	Increases the risk for bronchial obstruction, asthma, and wheezing in children (UNEP/WHO 2013).
Polybrominated diphenyl ethers (PBDEs)—Flame retardants in textiles, electronics, electric articles, furniture, and building materials.	Limited evidence for cognitive disorders, earlier age at menarche and cryptorchidism. Strong experimental evidence for suppression of thyroid hormone (UNEP/WHO 2013).
PAHs—Cigarette smoke, motor vehicle exhausts, industrial activities, and fossil fuel and wood burning.	Carcinogenic (Farmer et al. 2003; UNEP/WHO 2013).
Bacteria, fungi, algae, and some protozoa (their β-glucans, toxins, spores, pollen, cell fragments, methane, and VOCs emissions)—Microbial indoor air pollution when sufficient moisture is available.	Increases prevalence of respiratory symptoms, wheezing, cough, infections (bronchitis, fungal), allergies (alveolitis) and asthma, upper respiratory tract symptoms as well as perturbation of the immunological system (WHO 2009a).
Organometals and organometalloids—Metal and waste industries.	Mutagenic, carcinogenic, cytotoxic, neurotoxic (Hirner et al. 2004).
Radioactive materials—Radiation from nuclear power plants.	High doses (>100 mSV): Affect fetal development or outcome of pregnancy, spontaneous abortion, miscarriage, perinatal mortality, congenital defects, cognitive impairment and death. Also, significant associations between radiation exposure, and both cerebrovascular and cardiac disease mortality. Low doses (<100 mSV): Leukemia, breast cancer, thyroid cancer, and all solid cancers (WHO 2013).
Radon—Indoor radon exposure in buildings.	Lung cancer (WHO 2009b).



SO<sub>2</sub>, along with nitrogen oxides, are the main precursors of acid rain. As such it contributes to the acidification of lakes and streams, accelerated corrosion of buildings, and reduced visibility. SO<sub>2</sub> also causes the formation of microscopic acid aerosols, which have serious health implications and contribute to climate change (Ontario Ministry of the Environment 2010). The largest sources of SO<sub>2</sub> emissions are from fossil fuel combustion at power plants (73%) and other industrial facilities (20%). Smaller sources of SO<sub>2</sub> emissions include industrial processes such as extracting metal from ore, and the burning of high-sulfur-containing fuels by locomotives, large ships, and nonroad equipment. SO<sub>2</sub> is linked with a number of adverse effects on the respiratory system (EPA 2012b).

### **Organometal(loid)s**

Organometals and organometalloids are volatile, mobile, and toxic metals with a molecule that has a carbon moiety, most often a methyl group, rendering them lipophilic and so allowing them to bind more easily to living cells and become bioaccumulated.

Most organometal(loid)s yield metabolites that are both hydrophilic and lipophilic, facilitating their transport in bodily fluids and cell wall penetration. Interference with enzymes, proteins, hemoglobin, cytochromes, DNA, and the immune system ensue, but neurotoxic diseases dominate, including harm to muscle and sensory functions (paralysis and seizures), and harm to mood and cognitive functions (learning, memory, speech, and behavior) (Gruner et al. 2004).

Some organometals have been used as industrial catalysts, gasoline antiknock agents, biocides and antitumoral agents (Yonchev et al. 2004), preservatives, and antifouling paints (Gruner et al. 2004). Natural and anthropogenic areas (wetlands and ponds, waste dumps, and sewage systems) also host anaerobic microorganisms able to methylate heavy metals. Organometal(loid)s enter the atmosphere as gases or aerosols, and are exchanged with the hydrosphere and soil (Florea et al. 2004). Metal recycling plants are also hazardous working places (Fender and Wolf 2004).

Metals interfere with cell functions. Methylation in the human liver and kidney or by anaerobic gut microorganisms yields more cytotoxic compounds (affecting cells) than those entering it (Hirner et al. 2004); metals accumulate along the food chain (Stoltenburg-Didinger 2004).

High levels of arsenic exist in smelting, glass production, and other working environments. Arsenic inhalation increases the risk of lung cancer and may inhibit DNA repair and generate reactive oxygen species leading to elevated oxidative DNA damage at very low doses. Some of the induced lesions are mutagenic and lead to carcinogenesis (Hartwig et al. 2004).

Increased chromosomal aberrations have been found in workers in most exposure studies. Chromosomal aberrations and cancer risk have been linked. Organometal(loid) exposure includes tin, antimony, arsenic, and

lead in landfill gases, two order of magnitudes higher than in the background environment (Fender and Wolf 2004). Waste site workers had frequent multiaberrant cells, without influence of the duration of employment. In copper smelters, elevated lead and dioxin/furans (from plastics) were found, as well as aberrations, but significant only in cells with dicentric chromosomes. More sister chromatid exchanges were found than in waste site workers.

The brain concentrates metal ions such as lead (Pb) and mercury (Hg) in astroglia. Pb enters the human body mainly through inhalation and ingestion. It kills cells and interferes with normal neuronal development. Lead often mimics and inhibits the action of calcium. Lead produces irreversible learning and memory deficits in children at 15  $\mu\text{g}/\text{dl}$  blood but not in adults so the limit tolerable serum level was fixed at 10  $\mu\text{g}/\text{dl}$  blood by WHO and the U.S. Centers for Disease Control. Astroglia seem to be the main accumulation site in the brain, as well as for other neurotoxic metals (Stoltenburg-Didinger 2004). Astroglia participate in neurotransmitter metabolism in the blood–brain barrier, stress, and injury responses.

Lead can be found in all parts of our environment—the air, the soil, the water, and even inside our homes. Much of our exposure comes from human activities including the use of fossil fuels including past use of leaded gasoline, some types of industrial facilities, and past use of lead-based paint in homes. Lead and lead compounds have been used in a wide variety of products found in and around our homes, including paint, ceramics, pipes and plumbing materials, gasoline, batteries, ammunition, and cosmetics (EPA 2012c).

Lead emissions from volcanoes or fires are dwindled by anthropogenic emissions: 2000 to 18,000 t per year as compared to 300,000 to 450,000 t. Lead has been banned from gasoline and paints in many countries but even after production declined in developed countries, dust, water, and blood still contain lead beyond WHO recommended limits in 5% to 15% of the samples. Children absorb 50% of the lead intake and adults 10%, meaning possible intakes of up to 170  $\mu\text{g}$  per day per person. Until the late 1960s, 80  $\mu\text{g}$  were allowed by law (60  $\mu\text{g}$  in children). WHO established admissible limits in 1987 when it was already known since 1979 that these levels were conducive to impaired development, memory, and behavior. Furthermore, it was known since 1974 that lead accumulates in the hippocampus, an essential brain structure for memory and learning. Reduction of hemoglobin synthesis at the set admissible levels was known since 1982. It was already known since 1984 that 25  $\mu\text{g}/\text{dL}$  shortens pregnancy and reduces birth weight. In fact, lead neurotoxicity in children was clinically known since 1897. Many other cellular processes are disturbed by lead at low doses. As to lead from gasoline, it is metabolized in the human body to even more toxic products that depress several cellular processes (Hirner et al. 2004). According to a New York State Department of Health 1993 report, more patients were treated for lead than alcohol toxicity. Cities in developing countries are most at risk (Büsselberg 2004).

Aluminum has been linked to dementia and Alzheimer's disease. Zinc plays several important roles in mammal cells with reports of toxicity at even low doses. Glutamate is the most important excitatory neurotransmitter and its receptors are involved in learning, memory, development, and synaptic plasticity, which are blocked or disturbed by lead and aluminum. Mercury is used in tooth fillings and as antiseptic in vaccines, despite known cognition, tremor, coordination, and reflex issues of mercury intake, the mechanisms of which have been widely studied (Hirner et al. 2004). According to the 2008 U.S. EPA National Emissions Inventory, coal-fired electric power plants cause 48% of the total anthropogenic mercury emissions, followed by electric steel furnaces (8%), industrial boilers (7%), waste combustion in cement manufacturing (7%), and gold mining (3%).

Subclinical nerve injuries were found in Swedish copper smelter workers due to long-term exposure. Chronic exposure to combustion of coal containing elevated arsenic led to loss of hearing, taste, blurred vision, and limb numbness. Neurotoxic pesticide effects, ranging from chronic subclinical to acute clinical, have been reported around the world (Gruner et al. 2004).

### **Radiation**

*Radiation* is a general term referring to any sort of energy that can travel through space either as a wave or a particle. In considering related health risks, radiation may be classed as: (1) nonionizing radiation (lower energy, such as UV, visible light, infrared, microwaves, radio and radar waves, wireless Internet connections, mobile phone signals) and (2) ionizing radiation (higher energy, such as  $\alpha$ -particles,  $\beta$  particles,  $\gamma$ -rays, x-rays, cosmic protons, neutrons in accidental emissions of nuclear power plants) (NHS 2013).

Radiation damage to tissue or organs has been shown to depend on the type of radiation, the sensitivity of different tissues and organs, the dose, and the dose rate. Adverse health effects of radiation result from two mechanisms: (1) cell killing, which may cause functional impairment of the exposed tissue or organ only if a sufficient number of cells are affected; and (2) nonlethal changes in molecules of a single cell, most commonly in the DNA molecule, which may result in an increased risk of disease long after exposure. The first type of effects is determined mostly at the time of radiation. The second type of effect occurs through a random process that is not entirely determined at the time of radiation. These effects include cancer and inheritable effects. At low doses, radiation risks are primarily related to the second type, rather than to the deterministic effects characteristic of higher-dose exposure (WHO 2013).

Most radiation exposure is from natural sources (85% of the annual human radiation dose), mainly radioactivity in rocks and soil; radon, a radioactive gas given out by many volcanic rocks and uranium ore; and cosmic radiation. Radiation arising from human activities typically accounts for up to 15% of the public's exposure every year. This radiation is no different from

natural radiation except that it can be controlled. X-rays and other medical procedures account for most exposure from this quarter. Less than 1% of exposure is due to the fallout from past testing of nuclear weapons or the generation of electricity in nuclear plants, as well as coal and geothermal power plants (World Nuclear Association 2012). This does not account for local exposure following nuclear plant accidents.

Radon is a radioactive gas that emanates from rocks and soils, and tends to concentrate in enclosed spaces like underground mines or houses. Soil gas infiltration is the most important source of residential radon; other sources, including building materials and water extracted from wells are of lesser importance. The proportion of all lung cancers linked to radon is estimated between 3% and 14%, depending on the average radon concentration in the country and on the method of calculation. Radon is the second most important cause of lung cancer after smoking in many countries. Radon is much more likely to cause lung cancer in people who smoke or who have smoked in the past than in lifelong nonsmokers. However, it is the primary cause of lung cancer among people who have never smoked. There is no known threshold concentration below which radon exposure presents no risk. Addressing radon measures in construction of new buildings (prevention) and in existing buildings (mitigation or remediation) is needed. The primary radon prevention and mitigation strategies focus on sealing radon entry routes and on reversing the air pressure differences between the indoor occupied space and the outdoor soil through different soil depressurization techniques (WHO 2009b).

### ***Endocrine Disrupting Chemicals***

There is global transport of endocrine disrupting chemicals (EDCs) through air currents, leading to worldwide exposure of humans and wildlife. EDCs have the capacity to interfere with tissue and organ development and function, and therefore they may alter the susceptibility to different types of diseases throughout life. Nondescended testes in young boys are linked with exposure to diethylstilbestrol (DES) and polybrominated diphenyl ethers (PBDEs), and with occupational pesticide exposure during pregnancy. Exposures to polychlorinated dioxins and certain polychlorinated biphenyls (PCBs) are risk factors in breast cancer. Prostate cancer risks are related to occupational exposures to pesticides (of an unidentified nature), to some PCBs and to arsenic. Developmental neurotoxicity with negative impacts on brain development is linked with PCBs. Attention deficit/hyperactivity disorder (ADHD) is overrepresented in populations with elevated exposure to organophosphate pesticides. An excess risk of thyroid cancer was observed among pesticide applicators and their wives, although the nature of the pesticides involved was not defined (UNEP-WHO 2013).

Halogenated (chlorin-containing) organic substances include dioxins and furans and the most toxic compound yet known to man:

2,3,7,8-Tetrachlorodibenzodioxin (TCDD). Dioxins, furans, heavy metals, some PCBs, and pentachlorinated phenol (PCP) are carcinogens found in incineration atmospheric emissions (Council of the European Union 2000).

### ***Dampness and Mold***

Indoor environments contain a mixture of live and dead microorganisms, fragments thereof, toxins, allergens, volatile microbial organic compounds, and other chemicals, which in some concentrations are known or suspected to be elevated in damp indoor environments and may affect the health of people living or working there. In particular, it has been suggested that dust mites and fungi play a major role, since these produce allergens, toxins, and irritants known to be associated with allergies, asthma, and respiratory diseases. Dampness may also promote bacterial growth and the survival of viruses, but this has received little attention in the literature. The prevalence of home dampness indicated by occupants and inspectors is in the order of 10% to 50% in the most affluent countries. For less affluent countries, the prevalence sometimes exceeds 50%. Climate change and the rise in sea level and increased frequency and duration of floods are likely to increase the proportion of buildings with damp problems. In addition, inadequate design will prevent adequate ventilation in winter in many houses, leading to increased condensation and indoor dampness. Preventing persistent dampness and microbial growth on interior surfaces (through ventilation) is the most important means of avoiding harmful effects on health (WHO 2009a).

There are many evidences of health effects in humans due to air pollutants, which are summarized in [Table 12.2](#). This list is not exhaustive and the guideline values of the pollutants must be consulted in the WHO reports. The variation range of exposure in the scientific literature is high. The limits or guideline values change according to new evidence (WHO 2011a).

### **Air-Cleaning Technologies**

The most immediate method of improving air quality would be the use of biogas, solar, and wind energy, and hybrid vehicle technologies which apart from reducing toxic pollutants may help to reduce greenhouse emissions (WHO 2010a). Particle filtration and sorption of gaseous pollutants are among the most effective air-cleaning technologies (Figure 12.3), although used particle filters can be a source of pollution and there is insufficient information regarding long-term performance and proper maintenance (Zhang et al. 2011). Cool roofs and pavements, and urban trees can have a substantial effect on urban air temperature and, hence, can reduce cooling-energy use and smog (Akbari et al. 2001).



**FIGURE 12.3** (See color insert.)

A vortex creates an ascendant flow to suspend the particles imitating wind. Then the suspended particles are trapped in the drops imitating rain, and absorbed in a filter that imitates deposition and soil filtration. (© Photo by Marina Islas-Espinoza.)

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