Environmental Determinants of Health

37

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Introduction

Since the discovery of DNA's structure in 1953, researchers have debated the relative influence of genetic versus environmental factors as determinants of health. Estimates of the environmental contribution to disease have ranged from as low as 13% [1] to as high as 90% [2]. These differences arise in part due to varying definitions of "environment." For example, a recent World Health Organization (WHO) assessment of the environmental contribution to preventable disease defined the environment as including "exposure to pollution and chemicals (e.g., air, water, soil, products), physical exposures (e.g., noise, radiation), the built environment, other anthropogenic changes (e.g., climate change, vector breeding places), related behaviors and the work environment" [1]. The WHO estimates that 13–32% of the global disease burden is attributable to these environmental determinants. In contrast, thought leaders have suggested that in the extreme, all diseases are environmental because "genetic factors are actually also environmental, but merely on a different time scale" [3]. An intermediate viewpoint defines the environment as all factors external to the genome. However, based in part on prior studies of twins that computed the fraction of diseases attributable to genetic versus nongenetic factors, somewhere between 70% and 90% of disease risks may be attributable to differences in environments [2].

This chapter adopts a perspective of environmental determinants of health consistent with that of the WHO and focuses on chronic diseases related to pollutants in outdoor air, household indoor air, workplaces, and drinking water. Like the WHO, the chapter also considers exposure to lead—which can occur through ingestion of dust, soil, air, water, or food—as an environmental determinant. In addition, consistent with the concept of the built environment as a health determinant, the chapter also discusses the mounting evidence of the profound health impacts unintentionally created through automobile-centric urban designs of the post-World War II era. Overall, the chapter emphasizes environmental factors that are potentially modifiable by changes in individual behaviors or public policies, which physicians may be able to influence.

The chapter begins with an overview of how WHO and others have estimated the burden of chronic diseases attributable to environmental factors. Next, it provides background information on the environmental determinants included in this discussion: outdoor air pollution, household air pollution, drinking water contamination, occupational exposure to hazardous materials, lead exposure, and built environments that discourage physical activity. The final section provides guidance for physicians on incorporating concerns about environmental determinants into their healthcare practices.

Estimating the Burden of Disease from Environmental Determinants

In 1990, the World Bank commissioned the first comprehensive study to characterize the contribution of various risk factors to preventable diseases, in order to help define intervention packages for countries in different development stages [4]. Carried out by the WHO and published in 1996, the study assessed the global and regional disease burden attributable to ten different risk factors, including four environmental determinants (poor water supply and sanitation, air pollution, occupational exposures, and physical inactivity) [4, 5]. A follow-up burden of disease study, published in 2004, added an additional 16 risk factors [6]. Subsequent updates, the most recent published in 2015, were prepared by the Institute for Health Metrics and Evaluation (IHME)

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[7, 8]. The global studies have led to similar efforts at the national level [9–11], including in the United States.

Method for Estimating the Environmental Burden of Disease

All of the global burden of disease projects and their national-level counterparts have used a similar process that involves combining epidemiologic, environmental, and public health data. Disease burden studies begin by compiling evidence linking exposure to a given risk factor to specific health outcomes. Typically, these risk factor-disease pairs are identified through a comprehensive review of epidemiologic studies. Table 37.1 summarizes the health outcomes linked to risk factors discussed in this chapter, as determined from a review of evidence in previous global burden of disease studies [7, 8, 12].

Table 37.1 Selected environmental determinants of health

Risk factor	Associated health outcomes
Built environment not conducive to walking or cycling for transportation (leading to physical inactivity)	Breast cancer
	Colorectal cancer
	Diabetes
	Ischemic heart disease
	Ischemic stroke
Outdoor air pollution (particulate matter)	Chronic obstructive pulmonary disease (COPD)
	Ischemic heart disease
	Lower respiratory infections
	Lung cancer
	Stroke
Lead exposure (via corrosive water, soil, dust, and/or food)	Mild mental retardation (childhood exposure)
	High blood pressure (adults)
Household air pollution from second-hand smoke	Hemorrhagic stroke
	Ischemic heart disease
	Ischemic stroke
	Lower respiratory infections (children)
	Lung cancer
	Otitis media (children)
Household air pollution from radon	Lung cancer
Occupational carcinogens	Lung cancer
	Ovarian cancer
	Leukemia
	Nasopharynx cancer
Occupational particulate matter	COPD
Occupational asthmagens	Asthma
Waterborne carcinogens	Bladder cancer (disinfection byproducts)
	Lung/bronchus cancer (arsenic)
	All cancer (gross alpha radiation)
Waterborne pathogens	Diarrheal diseases

Once these risk factor-health outcome relationships are determined, the next step is to estimate a quantity known as the population attributable fraction (AF)—the fraction of observed diseases that could be prevented if exposure to a specific risk factor were curtailed. AF can be estimated from the following equation [9–11, 13, 14]:

$$\mathrm{AF} = \frac{\int_{x=0}^{m} \mathrm{RR}(x)P(x)dx - \int_{x=0}^{m} \mathrm{RR}(x)P'(x)dx}{\int_{x=0}^{m} \mathrm{RR}(x)P(x)dx} \quad (37.1)$$

where x is the pollutant exposure concentration or dose, RR(x) is the relative risk of an adverse health outcome at exposure concentration or dose x, $P(x)$ is the current population exposure distribution, and $P'(x)$ is an alternative (or counterfactual) exposure distribution. When the exposure is eliminated, then RR($x = 0$) = 1, and the integral on the right side of the numerator reduces to 1. The number of observed cases attributable to the exposure of concern (D_{attrib}) then can be calculated from

$$D_{\mathrm{attrib}} = \mathrm{AF} \times D_{\mathrm{total}} \quad (37.2)$$

where D_{total} is the total number of observed cases. Relative risk functions for each exposure and health outcome are estimated from meta-analyses or systematic reviews of prior epidemiologic studies. The population distribution of exposure is typically estimated from a combination of environmental data collected by state and federal agencies, along with behavioral data from a number of sources, such as the Behavioral Risk Factor Surveillance System [15].

To provide a common metric for comparing disparate health outcomes, such as premature mortality and chronic diabetes, or chronic diabetes and chronic asthma, the WHO developed a concept called the disability-adjusted life year (DALY). The DALY combines two quantities: the years of life lost due to premature mortality (YLL) and the years of life lived with "disability" (YLD). For each affected population age group, these quantities are calculated as

$$\mathrm{YLD} = I \times \mathrm{DW}\ X\ L \quad (37.3)$$

$$\mathrm{YLL} = N \times L \quad (37.4)$$

where I is the annual number of incident cases, L is the illness duration (for YLD) or the remaining life expectancy at the age of death (for YLL), and DW is the "disability weight," intended to represent the relative level of discomfort and interference with daily activities of life from each disease. The WHO has developed standard disability weights for different conditions. The weights were developed from surveys asking health professionals how many imaginary patients with a specific condition they would trade for 1000 healthy,

Table 37.2 Disability weights used in global burden of disease studies

	Untreated form					Treated form				
	Age group (years)					Age group (years)				
Sequela	**0–4**	**5–14**	**15–44**	**45–59**	**60+**	**0–4**	**5–14**	**15–44**	**45–59**	**60+**
Diarrheal episode	00.119	00.094	0.086	00.086	00.088	00.119	00.094	00.086	00.086	00.088
Mild mental retardation	00.361	00.361	00.361	00.361	00.361	00.361	00.361	00.361	00.361	00.361
Lower respiratory infections										
Episodes	00.280	00.280	00.276	00.276	00.280	00.280	00.280	00.276	00.276	00.280
Chronic sequelae	00.099	00.099	00.099	00.099	00.099	00.099	00.099	00.099	00.099	00.099
Upper respiratory infections										
Episodes	00.000	00.000	00.000	00.000	00.000	00.000	00.000	00.000	00.000	00.000
Pharyngitis	00.070	00.070	00.070	00.070	00.070	00.070	00.070	00.070	00.070	00.070
Cancers—preterminal										
Colon and rectum	00.217	00.217	00.217	00.217	00.217	00.217	00.217	00.217	00.217	00.217
Trachea, bronchus and lung	00.146	00.146	00.146	00.146	00.146	00.146	00.146	00.146	00.146	00.146
Bladder	00.085	00.085	00.085	00.085	00.085	00.087	00.087	00.087	00.087	00.085
Leukemia	00.098	00.098	00.108	00.112	00.112	00.083	00.083	00.093	00.097	00.097
Cancers—terminal	00.809	00.809	00.809	00.809	00.809	00.809	00.809	00.809	00.809	00.809
Diabetes mellitus										
Cases	00.012	00.012	00.012	00.012	00.012	00.033	00.033	00.033	00.033	00.033
Diabetic foot	00.137	00.137	00.137	00.137	00.137	00.129	00.129	00.129	00.129	00.129
Neuropathy	00.078	00.078	00.078	00.078	00.078	00.064	00.064	00.064	00.064	00.064
Retinopathy—blindness	00.600	00.600	00.600	00.600	00.600	00.493	00.491	00.488	00.488	00.488
Amputation	00.155	00.155	00.155	00.155	00.155	00.068	00.068	00.068	00.068	00.068
Ischemic heart disease										
Acute myocardial infarction	00.491	00.491	00.491	00.491	00.491	00.395	00.395	00.395	00.395	00.395
Angina pectoris	.0.227	00.227	00.227	00.227	00.227	00.095	00.095	00.095	00.095	00.095
Congestive heart failure	00.323	00.323	00.323	00.323	00.323	00.171	00.171	00.171	00.171	00.171
Cerebrovascular disease—										
First-ever stroke	00.262	00.262	00.262	00.268	00.301	00.224	00.224	00.224	00.224	00.258
COPD	00.428	00.428	00.428	00.428	00.428	00.388	00.388	00.388	00.388	00.388
Asthma—cases	00.099	00.099	00.099	00.099	00.099	00.059	00.059	00.059	00.059	00.059

Sources: World Health Organization. All outcomes other than mild mental retardation: http://www.who.int/healthinfo/global_burden_disease/tools_national/en/. Mild mental retardation: http://www.who.int/healthinfo/global_burden_disease/GBD2004_DisabilityWeights.pdf

imaginary people [16]. Table 37.2 shows disability weights for some of the health outcomes discussed in this chapter.

Current Estimates of the Environmental Burden of Disease

Globally, the most recent burden of disease estimate attributed 11.4 million annual deaths (21.2% of total deaths globally) and 354 million DALYs (16.3% of the global total) in the year 2013 to the environmental determinants discussed in this chapter. The published global estimate does not provide details for each country; however the IHME published a separate estimate for the United States for the year 2010 [12]. Figure 37.1 combines IHME estimates of the burden of disease from outdoor air pollution, household air pollution, occupational exposures, and built environment factors (through their influence on physical inactivity) with our own estimates for drinking water pollution, described below in the section entitled "Drinking Water Pollution." In total, 15% of all 2.6 million US deaths in 2010 and 8.9% of all 82 million DALYs are attributable to these determinants. The following sections provide background information on each determinant shown in Fig. 37.1.

Outdoor Air Pollution

Deadly smogs in Donora, Pennsylvania, in 1948 and London in 1952 spurred research to understand the impacts of air pollution on public health in the United States and Europe [20, 21]. In Donora, a smog so thick that daytime was as dark as night sickened about half of the population of 14,000 and led to 20 deaths [20]. In London, a similar smog led to a death toll estimated at the time to be 4000; later reanalysis placed the toll as high as 12,000 [21].

A large body of epidemiological, toxicological, and clinical research since the smogs of the mid-twentieth century has provided strong evidence linking adverse health

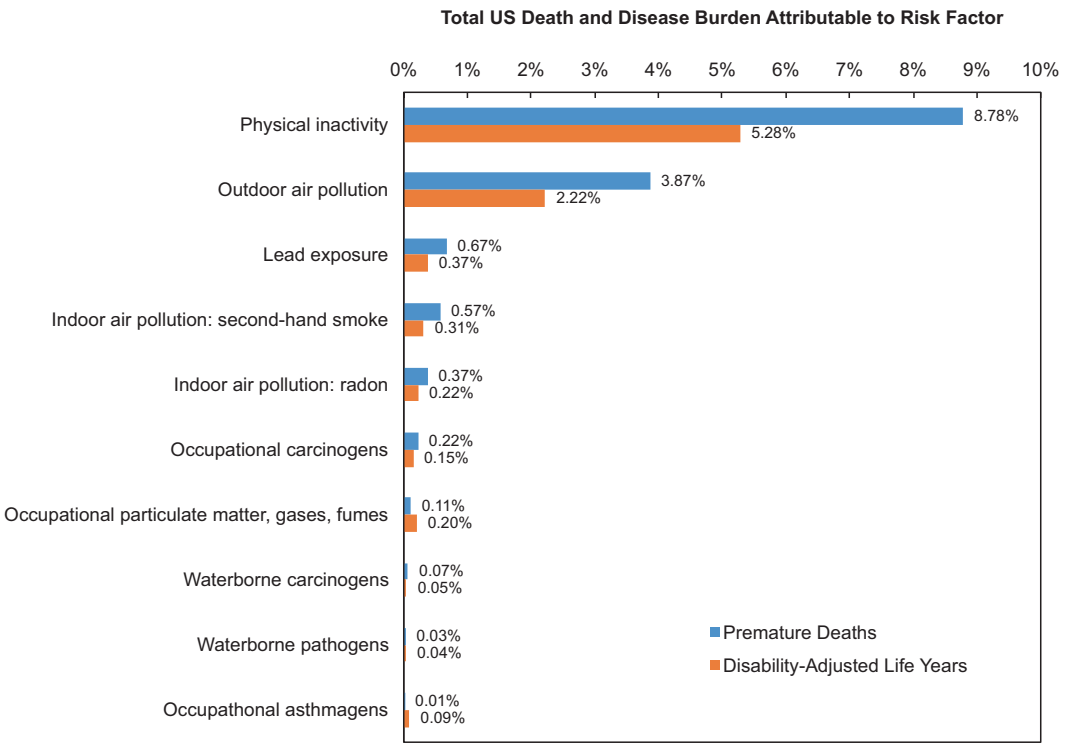

Fig. 37.1 Estimated contribution of environmental determinants to premature deaths and disability-adjusted life years in the United States (Developed from data in [12, 17–19])

impacts to exposure to three categories of common air pollutants: particulate matter (PM), ozone (O_3), and nitrogen dioxide (NO_2) [21, 22]. All three pollutants are strong oxidants that can affect health directly through oxidation of lipids and proteins and indirectly through activation of intracellular oxidant pathways [23]. Strong evidence supports causal associations between these pollutants and all-cause mortality, cerebrovascular disease (including stroke), ischemic heart disease, chronic obstructive pulmonary disease (COPD), lower respiratory tract infections, and trachea, bronchus, and lung cancers. Evidence also supports associations with bronchitis in children and adults and with elevated incidence of asthma symptoms in asthmatic children [24].

The Global Burden of Disease Project estimated that in 2013, more than 2.9 million deaths (5.4% of total deaths) and 70 million DALYs (2.8% of total DALYs) globally were attributable to ambient air pollution [7]. The IHME study attributed 103,000 US deaths (3.9% of total deaths) and 1.8 million DALYs (2.2% of total DALYs) in the year 2010 to ambient air pollution. To avoid double counting due to co-occurrence of pollutants, these estimates include only risks from particulate matter pollution so should be considered conservative.

Indoor Air Pollution

Insufficient ventilation has been recognized as dangerous to health since biblical times. However, until relatively recently, concerns about indoor air quality were driven by the need for odor control and comfort [25, 26]. During the 1980s, however, indoor air pollution rose to prominence, at first due to concerns about radon. Radon pollution of indoor air made national news in 1984 when a worker at the Limerick nuclear power plant in Pennsylvania triggered the radiation monitoring system at the power plant when he arrived at work; tests revealed that the source of his exposure was not occupational, but instead the air inside his household was contaminated with radon originating from underlying geologic formations [26, 27]. This incident focused national attention not just on radon but also on other sources of indoor air pollution, including formaldehyde, mold, and, more recently, environmental tobacco smoke. In addition, recent research in the developing world has spotlighted household air pollution arising from combustion of solid fuels indoors for cooking and heating.

In developed countries, recent evidence suggests that the household indoor air pollutants with the largest impacts on chronic disease are environmental tobacco smoke, radon,

and mold. A meta-analysis found that children of parents who smoke have twice the risk of hospitalization for serious respiratory infections as those with nonsmoking parents [28]. Similarly, studies have found elevated risks of asthma in children and chronic lymphocytic leukemia, lung cancer, and cardiovascular disease in adults among nonsmokers living with smokers [29–34]. Multiple studies, including several meta-analyses, have found consistent associations between visible mold in the home and the development and exacerbation of asthma in the United States and Europe [35–37]. A meta-analysis of studies from North America and Europe showed consistent associations between the presence of visible mold in the household and the risk of asthma and other respiratory outcomes (such as chronic coughs) in children aged 6–12 [36]. More than 20% of US asthma cases are attributable to mold in the home, according to one study [38].

Recent research also has documented associations between a variety of adverse health effects and indoor emissions of volatile chemicals from modern building materials [39–41]. Among the studied chemicals, evidence is strongest for formaldehyde [39, 40]. Formaldehyde has long been known to irritate the eyes and nasal passages in children and adults [40]. Multiple studies have linked development of childhood asthma and asthma exacerbations among those with previously diagnosed asthma to formaldehyde [39, 42]. Although some authors have questioned the strength of this evidence [40], a meta-analysis published in 2010 concluded that "results indicate a significant positive association between formaldehyde exposure and childhood asthma" [42]. Toxicologic research using rats and mice has linked formaldehyde exposure to increased risks of nasopharyngeal cancer, but recent research using molecular methods, in combination with epidemiologic evidence, suggests that these risks are much smaller than suggested by the animal studies of the early 1980s [41, 43].

The main indoor source of formaldehyde is emissions from composite wood products such as fiberboard, particleboard, and plywood [40]. Current guidelines suggest that formaldehyde exposure at concentrations less than 0.1 mg/m^3 are unlikely to cause adverse health effects. Measured mean indoor concentrations are generally lower than this level, but in some circumstances indoor concentrations can exceed this value. For example, in 2006, formaldehyde exposures in trailers distributed to hurricane Katrina victims by the US Federal Emergency Management Agency received a great deal of media attention. An independent scientific investigation found that the median formaldehyde concentration measured in four such trailers was 0.54 mg/m^3, and the highest level was 1.1 mg/m^3—more than 5 and 11 times the recommended exposure limit, respectively [44].

The Global Burden of Disease Project estimated that in 2013, 3.3 million deaths (6.2% of total deaths) and 92 million DALYs (3.8% of total DALYs) were attributable to indoor air pollution [7]. Most of this burden occurred in the developing world and was associated with indoor use of solid fuels for cooking and heating. The IHME study attributed 25,000 deaths (0.94% of total US deaths) to indoor air pollution: 9900 due to radon and 15,200 due to secondhand smoke [12]. Estimates of deaths and DALYs from mold and formaldehyde were not included in either the global or US studies. However, burden of disease studies elsewhere indicate that these two health determinants—especially mold—may pose a substantial disease burden. For example, a study in the United Arab Emirates attributed 12% of adult asthma and 8.6% of child asthma to exposure to mold indoors [11]. In addition, the study attributed 1.4% of children's visits to medical facilities for asthma to formaldehyde exposure [11].

Occupational Exposure to Environmental Pollutants

Although accidents, such as trips and falls, and ergonomic problems contribute substantially to the occupational disease burden, this review focuses on exposure to chemicals and airborne particulate matter in workplace environments. Physicians have recognized occupational pollutants as an important health determinant since at least the eighteenth century, when Percival Pott attributed scrotal cancer among young chimney sweeps to their exposure to soot [45]. Previous estimates of the disease burden from occupational pollutants have divided these exposures into three categories: [1] occupational asthmagens; [2] occupational particulate matter, gases, and fumes; [3] and occupational carcinogens [46, 47]. For all three categories, the most common resulting diseases overall are respiratory illnesses, including asthma, COPD, and lung cancer [48, 49].

Globally, estimates have suggested that 11% of asthma is associated with occupational exposures [46]. The American Thoracic Society has estimated that approximately 15% of asthma is attributable to occupational exposure [50]. Hundreds of biological and chemical agents in workplaces can trigger asthma. Biological agents include grains, flours, plants, wood dusts, and furs and other animal parts. Chemical agents include welding fumes, chlorofluorocarbons, alcohols, and metals and their salts [46]. Prior studies have found that occupational risks for asthma are highest among those employed in mining, manufacturing, service work, agriculture, and transportation. A recent study found that workers most at risk for exposure to airborne contaminants causing new-onset asthma, when compared to exacerbation of preexisting asthma, include nurses, cleaners, bakers, spray painters, and agricultural workers [51]. In addition to increasing the risk of asthma, exposure to occupational particulate

matter can contribute to COPD, silicosis, asbestosis, and coal workers' pneumoconiosis, the latter two of which are essentially exclusively occupational illnesses [46].

Among the hundreds of potential occupational carcinogens, those with the strongest evidence linking occupational exposures to health outcomes and contributing the most to occupational cancers are asbestos, diesel engine exhaust, secondhand smoke, and silica [8]. A survey of occupational exposure to 139 carcinogens in European Union workplaces, which is used as the basis for current estimates of the disease burden associated with occupational carcinogens, found that the occupations with the highest risk of exposure to these substances are mining, construction, transportation, and manufacturing [46].

The Global Burden of Disease Project estimated that in 2013, 561,000 deaths (1.0% of total deaths) were attributable to occupational exposures: 304,000 (0.56%) from carcinogens; 205,000 (0.38%) from particulate matter, gases, and fumes; and 52,000 (0.10%) from asthmagens [7]. In addition, 17.4 million DALYs (0.71% of the global total) were attributable to these occupational exposures: 5.80 million (0.24%) to carcinogens; 8.80 million (0.36%) to particulate matter, gases, and fumes; and 2.77 million (0.11%) to asthmagens.

In the United States, the occupational disease burden is lower than that globally, due to stronger occupational health and safety regulations than in developing countries. In total in 2010, 9000 US deaths (0.34% of total deaths) were attributable to occupational exposures—about one-third of the global attributable fraction. Of these deaths, 5900 (0.22%), 2900 (0.11%), and 200 (0.0075%) were attributable to carcinogens, particulate matter, and asthmagens, respectively. Of total US DALYs, 362,000 (0.44%) were attributed to occupational exposures, which is about 38% lower than the global attributable fraction. Of these, 120,000 (0.15%) were attributable to carcinogens, 167,000 (0.20%) to particulate matter, and 75,200 (0.092%) to asthmagens.

While burden of disease analyses are useful indicators of the potential magnitude of risks from environmental exposures, research suggests that the occupational disease burden may be substantially underestimated. Causes of underestimation include the long latency periods between occupational exposures and the onset of some diseases, the multiple potential causative factors for any given disease, and the lack of recognition by primary healthcare providers that workplace pollutants could have contributed to a patient's health status [52]. A US study designed to assess the impacts of underreporting of occupational illnesses found that 39% of patients in general medical clinics believed their illness could be "possibly caused by work," and 66% thought it could be "possibly worsened by work," even if not caused by work [53].

Drinking Water Pollution

Control of microbial contaminants in drinking water has been heralded as the greatest public health advance of the twentieth century in the United States. Between 1900 and 1940, US mortality rates declined by 40%, and life expectancy at birth increased from 47 to 63 years. Nearly half of these gains have been attributed to the reduction in population exposure to waterborne pathogens brought about by installation of drinking water chlorination and filtration systems in major US cities [54]. Nonetheless, waterborne disease outbreaks—albeit sporadic—continue to occur in the United States, and some populations are at increased risk, as compared to others.

The vast majority of waterborne disease outbreaks are unreported [55, 56]. Nonetheless, a CDC database including all outbreaks reported since 1971 provides some insights into the nature of waterborne illnesses (Fig. 37.2) and etiologic agents (Fig. 37.3) that continue to pose risks to US population health [58]. Among 762 reported outbreaks attributed to contamination of drinking water from public water supplies or individual wells, 88% resulted in acute gastrointestinal illnesses (AGI) caused by a range of intestinal pathogens (Fig. 37.2). Next most common were hepatitis A (4% of outbreaks) and acute respiratory illness caused by *Legionella* (3% of outbreaks).

Outbreak data indicate that the rate of *Legionella* outbreaks is increasing; during the period 2001–2006, *Legionella* caused 29% of reported outbreaks, all from growth and dissemination in premise plumbing, pipes, and storage infrastructure (including two outbreaks in healthcare settings). In addition to outbreaks of AGI, hepatitis A, and *Legionella*, one outbreak of primary amebic meningoencephalitis (caused by *Naegleria fowleri*) occurred, along with several outbreaks of skin rashes. About 11% of outbreaks were caused by chemicals, most commonly copper but also including fluoride, nitrate, arsenic, and other chemicals.

Although AGI arising from waterborne pathogens is usually self-limited, in rare cases these infections can lead to serious chronic or even fatal conditions. For example, *Campylobacter* is associated with Guillain-Barre syndrome; *Salmonella* and *Shigella* with reactive arthritis; *Giardia* with failure to thrive, lactose intolerance, and chronic joint pain; and *E. coli* O157:H7 with hemolytic uremic syndrome [56]. Furthermore, waterborne contaminants associated with self-limiting AGI in healthy populations may lead to severe complications and mortality among sensitive populations, such as the elderly, immunocompromised, pregnant women, and young children. For example, the largest US waterborne disease outbreak in recent history occurred due to contamination of the Milwaukee, Wisconsin, water supply with

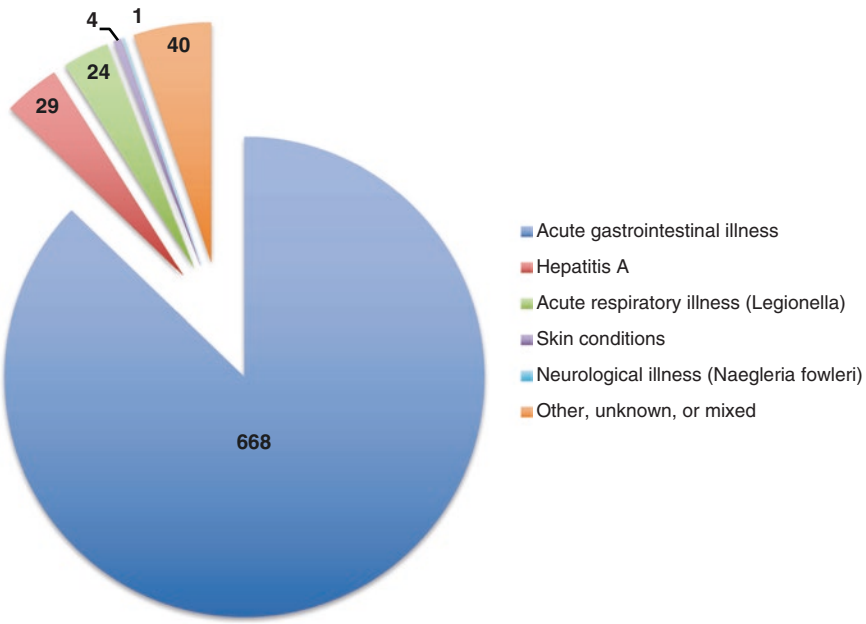

Fig. 37.2 Illnesses in reported US waterborne disease outbreaks, 1971–2006 (developed from data in [57])

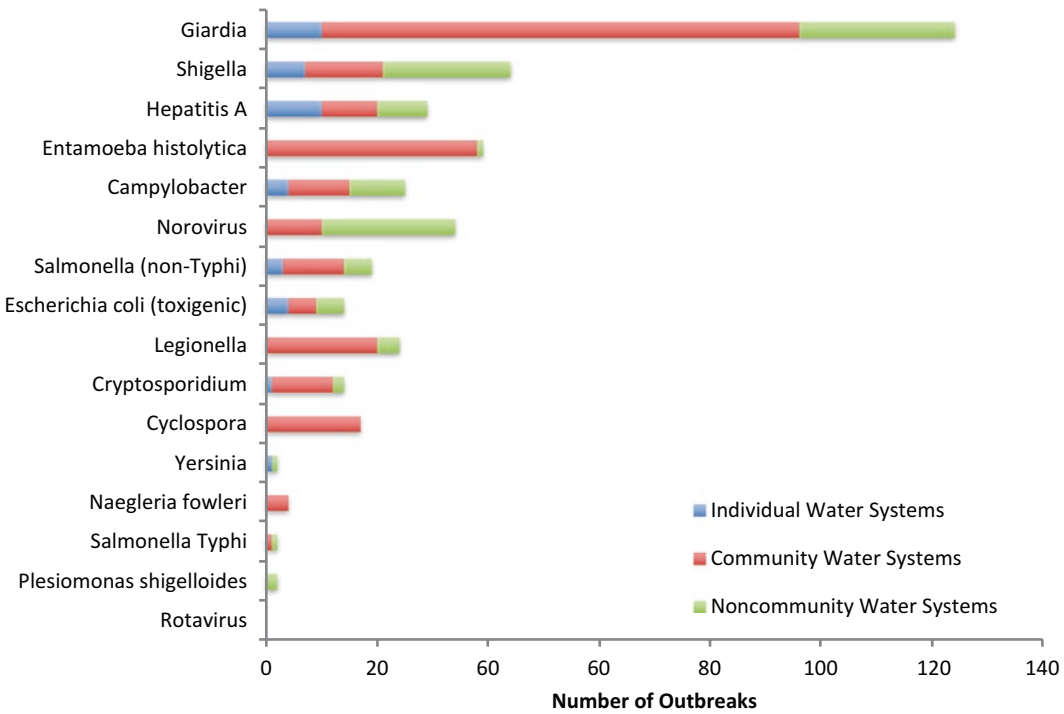

Fig. 37.3 Etiologic agents associated with reported US waterborne disease outbreaks, 1971–2006 (of 456 infectious disease outbreaks with known etiologies; developed from data in [57])

Cryptosporidium for 2 weeks in 1993 [59, 60]. This outbreak sickened more than 400,000 people and caused 50 premature deaths, 85% of them among AIDS patients. Recent evidence suggests that repeated infections with *Cryptosporidium* among infants aged 0–2 can lead to malnutrition, impaired growth, and decreased educational performance during later childhood [61].

While waterborne disease outbreaks are generally rare in large municipal water systems, breakdowns in these systems occur. In addition to the Milwaukee example, one recent highly publicized example of the failure of a municipal system was the case in Flint, Michigan, where city residents were exposed to elevated levels of lead in their drinking water. The increase in lead exposure was caused by a switch in the city's water supply, from Lake Huron water treated by the City of Detroit to the corrosive water of the Flint River, as part of an effort to save money for the bankrupt city. Recent research has found that the incidence of elevated blood lead levels in children more than doubled (from 2.4% to 4.9%) during this time period [62], placing the exposed children at increased risk of neurocognitive impacts such as reduced IQ and overall life achievement.

About 14% of the US population obtains their drinking water from private wells [63]. These wells are not regulated by the Safe Drinking Water Act, which covers only public water systems—those serving more than 25 people or 15 service connections year-round (community systems) or those regularly serving the public (non-community systems, such as campgrounds, gas stations, and schools, factories, or hospitals with their own water systems). Recent research has shown that those relying on private wells for their drinking water are at increased risk of AGI from waterborne pathogens. For example, a study in North Carolina found that 7.3% of emergency department visits for AGI could be attributed to microbial contaminants in drinking water; of these visits, 99% were associated with contamination of private wells [17].

Also at higher risk of exposure to contamination are those relying on small or very small water systems—those serving fewer than 3300 or 500 people, respectively. These systems lack the economies of scale of larger systems and are more likely to be financially stressed, causing difficulties with appropriate monitoring and maintenance of treatment systems. In a typical year, nearly 90% of violations of the Safe Drinking Water Act occur in small and very small water systems [64–66].

In addition to illnesses tracked in the CDC's waterborne disease surveillance system, contamination of drinking water is associated with other illnesses not easily recognized as waterborne due to multiple etiologies and a lag between exposure and disease onset. These other illnesses include lead poisoning, such as in the Flint, Michigan, case, and cancers. Among carcinogens in drinking water, disinfection byproducts formed by the reaction of disinfectants (such as chlorine) with natural organic compounds in the water (from decayed vegetation and other sources) appear to pose the biggest health impact, followed very distantly by arsenic, which is naturally occurring. Despite the increased cancer risks that may be caused by disinfection byproducts, studies have shown that the benefits of reduced infectious disease risks far outweigh the cancer risks [67].

Arsenic is a naturally occurring chemical concentrated in selected geologic regions. Acute exposure to high levels of arsenic in drinking water causes skin lesions, including blackfoot disease. However, such acute exposures are generally not observed in the United States. At lower exposure levels such as those that could occur in US groundwater in some geologic regions, chronic exposure to arsenic in drinking water is associated with skin, bladder, kidney, and lung cancer; heart disease; neurological abnormalities; and diabetes [68, 69]. In the United States, health risks from arsenic exposure are likely to be highest in private wells, due to the lack of regulation [70]. Public water systems, in contrast, are required to monitor for arsenic and remove it to very low levels if detected.

The Global Burden of Disease Project attributed 1.25 million deaths (2.3% of the total) and 75.1 million DALYs (3.1% of the total) to unsafe water sources. These estimates are based on the fraction of the population in each country with access to improved water and sanitation facilities, as defined by the WHO/UNICEF Joint Monitoring Programme for Water Supply and Sanitation (Table 37.3). The fraction without access to improved water sources is assumed to have a 35% increased risk of AGI and typhoid, in comparison to those with improved water access.

Because the vast majority of US residents have access to improved drinking water sources, the IHME estimation approach may not provide the most accurate information for US policymaking. The approach is not based on US-specific water quality data, and it does not include noninfectious disease risks, such as cancer, that may be of concern.

For this chapter we estimated separately the burden of disease in the United States from waterborne pathogens and carcinogens based on water quality and health outcome data. To develop these estimates, we applied AF estimates from

Table 37.3 WHO/UNICEF definitions of unimproved and improved water sources

Unimproved	Improved
Unprotected spring	Piped water into dwelling
Unprotected dug well	Piped water to yard/plot
Cart with small tank/drum	Public tap or standpipe
Tanker truck	Tube well or borehole
Surface water	Protected dug well
Bottled water	Protected spring
	Rainwater

recent comprehensive studies in North Carolina that are based on measured concentrations of microbial and chemical contaminants in public water supplies and private wells [17–19]. These studies estimated that 7.3% of acute gastrointestinal illnesses and 0.30% of cancers are attributable to microbial and chemical contaminants in drinking water, respectively. We multiplied these fractions by IHME data on deaths and DALYs from AGI and all cancers in the United States in 2010, in order to estimate the US burden of disease from drinking water pollution [12]. Using this approach, we attribute 2600 deaths (0.097% of total deaths) and 66,000 DALYs (0.081% of the total) to waterborne contaminants. Among the deaths, 1900 (0.071%) are attributable to carcinogens and 710 (0.027%) to pathogens. Among DALYs, 37,000 (0.045%) are attributable to carcinogens and 29,000 (0.036%) to pathogens. By contrast, the IHME estimate attributed 300 deaths and 10,700 DALYs to unsafe drinking water in the US, considering only effects on AGI and typhoid due to lack of access to an improved water source.

Lead Exposure

Lead toxicity has been recognized for more than 2000 years. For example, during the first century AD, Roman scholar and naval commander Pliny, in his *Naturalis Historia*, described poisoning among shipbuilders along with pallor among miners exposed to lead [71, 72]. Nonetheless, until the first cases of childhood lead poisoning were documented in the late nineteenth and early twentieth centuries, lead exposure was thought to occur only in certain high-risk occupations [73]. Recent events in Flint, Michigan, in which lead concentrations in the municipal water supply peaked due to the switch to a corrosive water that leached lead from water pipes, has refocused national attention on health risks of lead exposure [74, 75].

Exposure to lead may occur though ingestion of lead-contaminated dust, water, soil, or food or from inhalation of contaminated air. Until lead was banned from gasoline in progressive stages beginning in 1980, the major source of exposure was ingestion of soil and dust contaminated with airborne lead released by motor vehicles [76]. Dust from lead in household paint is another major source. Lead was banned from household paint in 1978 [77], but homes built before then remain at risk. Even if covered with additional paint layers, household residents (especially children) are at risk of exposure via dust from flaking paint, for example, in window casings where friction can erode upper layers and leave a dust residue on window sills. Consumer products, such as glazed ceramics from certain countries, also can be sources of lead exposure.

Lead solder in food cans is a dietary source, although the food industry has collaborated with the Food and Drug Administration over the past three decades to virtually eliminate the use of lead-containing materials in food storage containers manufactured in the United States [78]. As a result of bans on lead in gasoline, household paint, and food cans, blood lead levels in children and adults have declined progressively since the 1980s. For example, according to the CDC, the fraction of children with blood lead levels above 10 μg/dl decreased from nearly 8% to less than 0.5% during the time period 1997–2015 [79]. Nonetheless, each year an estimated 120,000 children under age 5 have blood lead levels above 10 μg/dl (the CDC's threshold for elevated blood lead before 2012, when the definition of elevated blood lead changed to 5 μg/dl).

Over the course of the twentieth century, concern about lead exposure increased as studies demonstrated risks at increasingly lower exposure levels. In the United States, the first documented case of childhood lead poisoning was recorded in 1914 [73]. At the time, the prevailing wisdom was that a child who survived acute poisoning would recover fully. However, in 1943, the first follow-up study of acutely lead poisoned children found that 19 of 20 subjects exhibited cognitive difficulties, including behavioral problems, learning difficulty, and failure in school many years later [73]. In the 1970s, researchers began to document cognitive effects of lead in children who had been exposed but showed no clinical signs of acute poisoning. As subsequent research has built on these findings [80–83], the CDC has progressively lowered its definition of elevated blood lead concentrations from 60 μg/dl in 1960 to the current 5 μg/dl. Recent research suggests that adverse impacts occur even below 5 μg/dl [73].

At high exposure concentrations, lead can cause acute clinical symptoms in children and adults. The concentration at which acute symptoms occur varies by individual but is generally in the range of 60 μg/dl. In adults, symptoms of acute lead poisoning include peripheral neuropathy with wrist or foot drop, slowed peripheral nerve conduction, colic, clumsiness, clouded thinking, weakness, and paralysis. In addition, acute lead poisoning increases the incidence of stillbirths and female and male infertility. In adults, lead toxicity should be considered in the differential diagnosis of abdominal pain, arthralgia, hypertension, severe headache, increased intracranial pressure, CNS dysfunction, anemia, and renal dysfunction. A blood lead level >10 μg/dl should be considered elevated, even though clinical symptoms are rarely seen below 60 μg/dl [73].

Children are more vulnerable to adverse health effects from lead exposure due to their still-developing central nervous systems, increased lead absorption, and more frequent hand-to-mouth behavior. Clinical symptoms of acute exposure, which usually manifest at blood lead levels above 60 μg/dl, may begin with abdominal pain and arthralgia, progress to clumsiness and staggering with headaches and behavioral problems, and in the worst cases lead to encepha-

lopathy (though the latter is rare in the United States). Beginning in the 1970s, researchers began to document associations between permanent IQ loss in children and exposure to lead, even at low exposure levels [80]. Recent meta-analyses have found a loss of about 1.3 IQ points for every 5 μg/dl increase in blood lead levels in children [84]. New research shows adverse impacts on social behavior and associated increases in aggression and delinquency later in life. One study of bone lead levels in a juvenile cohort found that 11–38% of delinquent behavior could be attributed to early lead exposure on the basis of bone lead measurements [85]. However any child with growth failure, abdominal pain, behavior change, hyperactivity, language delay, or anemia should be tested for lead toxicity [73].

When blood lead levels exceed 40 μg/dl, patients should receive chelation therapy, with a 5-day course of EDTA (sodium calcium edetate) or a 19-day course of dimercaptosuccinic acid (succimer). A repeated course may be required if blood lead levels do not stabilize. Critically, the source of exposure must be identified through a home inspection (or, for workers, work site investigation). Unfortunately, chelation therapy does not eliminate the cognitive damage in children, and the only remedy for low-level lead exposure is therefore primary prevention [73].

WHO and IHME estimates of the burden of disease attributable to lead exposure emphasize the risks of relatively low but widespread exposures, rather than acute exposures. On the basis of the strength of available evidence, they focus on IQ loss leading to mild mental retardation in some children, gastrointestinal effects in children, elevated blood pressure in adults, and anemia in children and adults. Globally, the IHME estimated that 853,000 deaths (1.6% of the total) and 17 million DALYs (0.69% of the total) could be attributed to lead exposure in 2013 [8]. In the United States, 17,900 deaths (0.67% of the total) and 306,700 DALYs (0.37% of the total) could be attributed to lead exposure in 2010 [12].

Automobile-Centric Urban Designs

Since World War II, Americans have become much less physically active due to declines in physically active transportation (e.g., walking and biking), occupations, and household activities [86]. Overall, only about 45% of Americans meet the CDC's recommendation of 150 min of moderate to vigorous physical activity per week [87]. While about 36% of Americans are aware of the CDC's physical activity guidelines, fewer than 1% could correctly identify the amount of activity the CDC recommends [88]. Failure to meet these guidelines is associated with increased risks of multiple chronic diseases, including breast and colorectal cancers, diabetes, ischemic heart disease, and stroke [12, 89–92].

The decline in physical activity and associated rise in chronic disease rates is in part attributable to automobile-centric urban designs of the post-World War II era, along with increases in automation reducing physical activity at work and home [93–96]. In the United States, highway construction projects and suburban sprawl of the twentieth century in effect eliminated physical activity as a means of transportation for many Americans. For example, only 3.4% of Americans reported walking or biking to work in 2012 [97]. Recent research has shown that US residents who walk to work spend an additional 19.8 min per day walking, when compared to those who drive, and bicycle commuters exercise 32 min a day (28 min due to cycling and 4 due to walking) more than automobile commuters [97].

These results suggest that at least some Americans could achieve most or all of the recommended physical activity by switching from driving to either walking or cycling to work. Similar benefits can be gained by switching from driving to using public transportation. For example, a study in Charlotte, NC, showed that residents who began using a new light rail stop to commute reduced their BMI by 1.18 kg/m^2, on average, over 1 year—equivalent to a weight loss of 6.45 lbs for someone who is 5′5″ tall [98]. Multiple simulation studies have also shown substantial health benefits of reduced chronic diseases, mediated through physical activity, of compact neighborhoods with accessible public transportation, infrastructure (such as sidewalks and bikeshare programs) to support walking and cycling, and mixed land uses, in comparison to sprawling suburban neighborhoods lacking in such infrastructure [99–102].

The Global Burden of Disease Project attributed 2.18 million deaths (4.1% of total deaths) and 45.1 million DALYs (1.8% of the total) in the year 2013 to physical inactivity [8]. Relative to other environmental determinants, the physical inactivity risks are much higher in the United States than globally. The IHME attributed 234,000 deaths (8.8% of the total) and 4.32 million DAYLs (5.3% of the total) to physical inactivity [12]. Thus, the proportion of disease attributable to physical inactivity is three times as large in the United States as globally when measured as DALYs and more than twice as high when measured as deaths.

Addressing Environmental Risk Factors in Chronic Illness Care

This chapter highlights that all of the most common chronic diseases in the United States can be triggered or exacerbated by exposure to pollutants in the ambient, home, or workplace environment. In addition, modern urban designs that discourage physically active transportation (e.g., walking and cycling) in favor of reliance on personal automobiles are now widely recognized as an environmental risk factor affecting

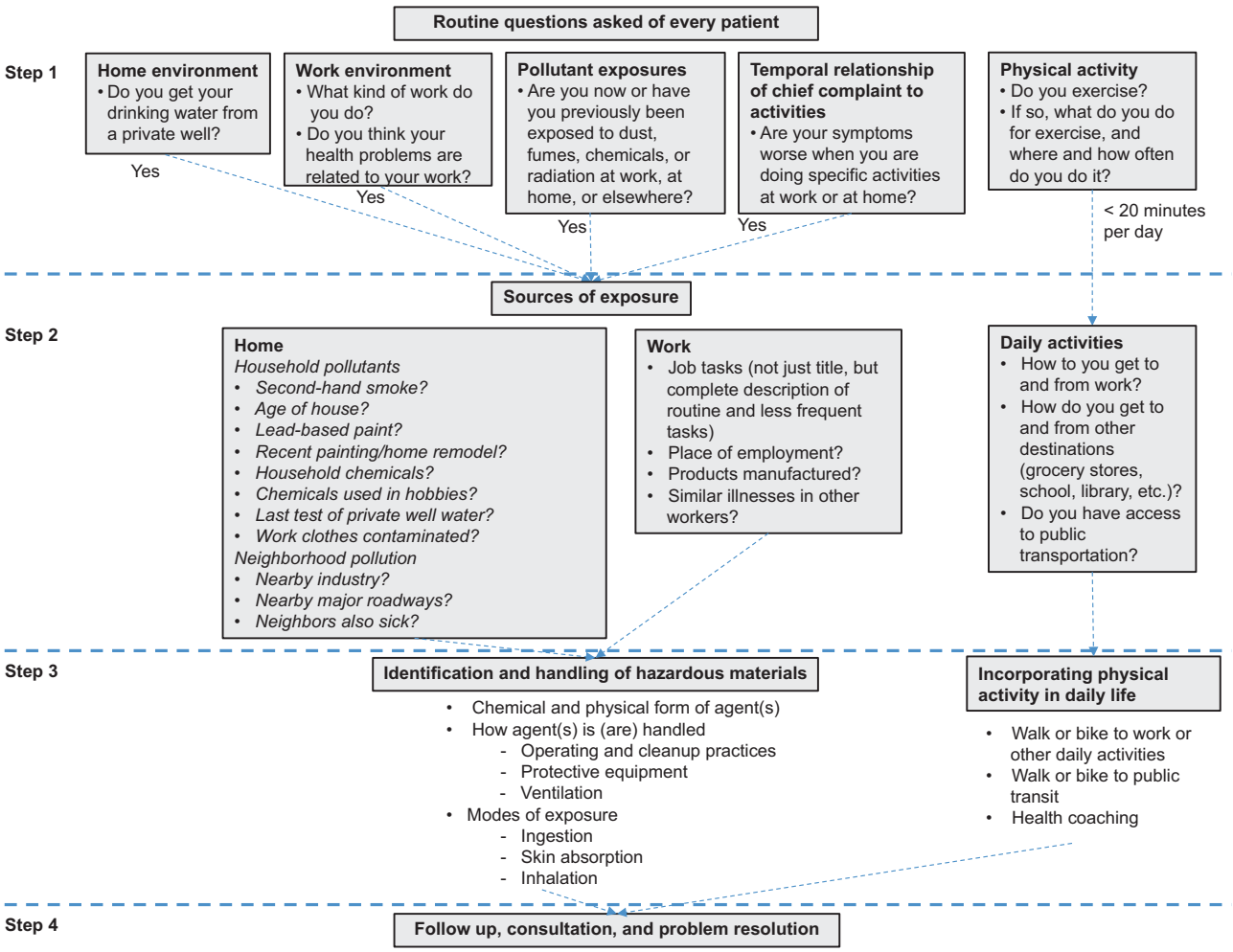

Fig. 37.4 Systematic approach to diagnosing potential environmental contributors to patient health

chronic disease prevalence. Given the multitude of environmental factors influencing health, untangling the potential role of any one of these factors—or combinations of them—in illnesses presenting to a physician or other healthcare provider may be daunting. Nonetheless, identifying underlying environmental factors may be critical to effective treatment or management of a patient's disease.

To help physicians uncover whether environmental factors may be contributing to a patient's disease, specialists in environmental and occupational medicine have developed systematic approaches to eliciting patient histories and diagnosing environmental or occupational illnesses. Fig. 37.4 provides an example, which is adapted from previous questionnaires by physicians at the Harvard School of Public Health and Yale University School of Medicine to include questions about risk factors related to the built environment [103, 104].

The approach for eliciting environmental health histories from patients shown in Fig. 37.4 occurs in three stages, proceeding from the general to the specific. The first stage includes several broad screening questions. The first few questions elicit information to help the physician determine whether the patient may have been exposed to pollutants at home or at work. In addition, these screening questions ask whether the patient has observed a temporal relationship between symptoms and exposures (e.g., decreased symptoms during vacations). If such relationships exist, then the suspicion that an underlying environmental risk factor may have triggered or exacerbated health symptoms increases. In addition, due to the mounting evidence of the deleterious effects of modern environments on physical activity, the screening stage includes two questions about whether and how much the patient exercises. Based on the answers to the screening questions, the physician may or may not proceed to a second, more detailed line of questioning. In this stage, the physician should ask not only about job titles or home locations but also about detailed job tasks, hobbies, and other infrequent activities that could lead to exposure. For example, there is a case of a retired executive who experienced myocardial infarction as a result of using methylene chloride to strip varnish from a wooden chest in an unvented basement; methylene chloride is rapidly metabolized to carbon

Table 37.4 Occupational and environmental health organizations in the United States

Organization	Mission	Contact information
Agency for Toxic Substances and Disease Registry	Federal public health agency that provides health information to prevent harmful exposures and diseases related to toxic substances	Telephone: 800-232-4636
		Website: http://www.atsdr.cdc.gov/
American College of Occupational and Environmental Medicine	Organization representing physicians and other healthcare professionals specializing in the field of occupational and environmental medicine	Telephone: 847-818-1800
		Website: http://www.acoem.org/
Association of Occupational and Environmental Clinics	A nationwide network of more than 60 multidisciplinary clinics and more than 250 occupational and environmental medicine professionals	Telephone: 888-347-2632
		Website: http://www.aoec.org/
National Institute for Occupational Safety and Health	Federal agency responsible for conducting research and making recommendations for the prevention of work-related illness and injury	Telephone: 800-232-4636
		Website: http://www.cdc.gov/niosh/
Occupational Safety and Health Administration (OSHA)	Federal agency responsible for enforcing safety and health legislation. OSHA also offers free on-site consulting to small- and medium-sized businesses. Consultations are separate from enforcement and do not result in penalties	Telephone: 800-321-6742
		Website: http://www.osha.gov/

Source: Re-created from Taiwo et al. [104]

monoxide, which can place substantial stress on the cardiovascular system. For patients whose health symptoms could result in part from physical inactivity, in this stage the physician can also inquire about potential opportunities to incorporate walking and cycling into the patient's daily routine.

The third step is to characterize health effects of exposures uncovered during the first and second stages. Table 37.1 lists health outcomes associated with risk factors discussed in this chapter. For additional information about specific hazardous chemicals, physicians can consult material safety data sheets, which employers are required to provide to workers or their physicians, reference manuals, occupational safety and health organizations (see Table 37.4), or poison control centers. Other references include [103] *Dreisbach's Handbook of Poisoning* [105] and *Clinical Toxicology of Commercial Products* [106], available in medical libraries.

The last stage involves identifying options for treating or managing the patient's condition, along with developing a follow-up plan. In some cases, eliminating exposure to the risk factor can treat the illness. Examples include installing a home water treatment system where water contamination is a source of illness or wearing personal protective equipment to guard against occupational exposures. In some cases, such as for chronic beryllium disease, a change of jobs may be essential. Medical treatment (e.g., chelation therapy for lead exposure) is available for some environmental exposures. In other cases, a physician can refer patients to specialists in occupational medicine or other related fields. Physicians can also report suspected environmental and occupational illnesses to public health officials, trade union health specialists, and workplace managers, thus potentially leading to protections for others. In the case of exposures in the workplace, physicians can help patients to apply for workers' compensation to help cover their medical expenses. In some states, workers can claim these benefits even if occupational exposure was not the primary cause if the work environment "precipitated, hastened, aggravated, or contributed to the ... illness" [103].

When illness is associated with lifestyle choices that may be impacted by the modern built environment, one option is to prescribe health coaching. Over the past decade, health coaching has emerged as a complimentary approach to combating chronic disease [107, 108]. While the definition of health coaching continues to evolve, commonly it includes one-on-one, telephone, or web-based consultations to help patients set and achieve goals for health-promoting behavior changes. Coaching methods are drawn from research in behavioral psychology. Several universities now offer certificate programs in integrated health coaching. Additional information about health coaching can be found at http://guides.mclibrary.duke.edu/integrativecoachingpatients.

Reporting Requirements for Environmental Diseases

When a physician suspects an environmental or occupational factor may have contributed to clinical symptoms in a patient, in some cases those illnesses must be reported to the health department. These reportable illnesses are in two categories: infectious and occupational. The lists of reportable illnesses vary greatly by state, as illustrated in Table 37.5, which compares reportable infectious diseases in California and North

Table 37.5 Comparison of reportable conditions in North Carolina and California (as of December 2016)

Condition	State	Condition	State
Acquired immune deficiency syndrome (AIDS)	North Carolina	Lymphogranuloma venereum	North Carolina
Amebiasis	California	Malaria	Both
Anaplasmosis	California	Measles (rubeola)	Both
Anthrax	Both	Meningitis, pneumococcal	North Carolina
Babesiosis	California	Meningitis, specify etiology: viral, bacterial, fungal, parasitic	California
Botulism	Both	Meningococcal infections	Both
Brucellosis	Both	Middle East respiratory syndrome	North Carolina
Campylobacteriosis	Both	Monkeypox	North Carolina
Chancroid	Both	Mumps	Both
Chickenpox (varicella) (outbreaks, hospitalizations and deaths)	California	Nongonococcal urethritis	North Carolina
Chikungunya virus infection	Both	Novel influenza virus infection	North Carolina
Chlamydia trachomatis	Both	Novel virus infection with pandemic potential	California
Cholera	Both	Paralytic poliomyelitis	North Carolina
Ciguatera fish poisoning	California	Paralytic shellfish poisoning	California
Coccidioidomycosis	California	Pelvic inflammatory disease	North Carolina
Creutzfeldt-Jakob disease	Both	Pertussis (whooping cough)	California
Cryptosporidiosis	Both	Plague	Both
Cyclosporiasis	Both	Poliovirus infection	California
Cysticercosis or taeniasis	California	Psittacosis	Both
Dengue	Both	Q fever	Both
Diphtheria	Both	Rabies, human	North Carolina
Domoic acid poisoning (amnesic shellfish poisoning)	California	Rabies, human or animal	California
Ehrlichiosis	Both	Relapsing fever	California
Encephalitis, arboviral	North Carolina	Respiratory syncytial virus (only report a death in a patient <5 years of age)	California
Encephalitis, specify etiology: viral, bacterial, fungal, parasitic	California	Rickettsial diseases (non-rocky Mountain spotted fever), including typhus and typhus-like illnesses	California
Escherichia coli, Shiga toxin-producing	Both	Rocky Mountain spotted fever	Both
Flavivirus infection of undetermined species	California	Rubella (German measles)	Both
Foodborne disease	Both	Rubella congenital syndrome	North Carolina
Giardiasis	California	Salmonellosis	Both
Gonococcal infections	California	Scombroid fish poisoning	California
Gonorrhea	North Carolina	Severe acute respiratory syndrome (SARS)	North Carolina
Granuloma inguinale	North Carolina	Shiga toxin (detected in feces)	California
Haemophilus influenzae, invasive disease	Both	Shigellosis	Both
Hantavirus infection	Both	Smallpox	Both
Hemolytic uremic syndrome	Both	*Staphylococcus aureus* with reduced susceptibility to vancomycin	North Carolina
Hemorrhagic fever virus infection	North Carolina	Streptococcal infection, group A, invasive disease	North Carolina
Hepatitis A, acute infection	Both	Streptococcal infections (outbreaks of any type and individual cases in food handlers and dairy workers only)	California
Hepatitis B	Both	Syphilis	Both
Hepatitis C	Both	Tetanus	Both
Hepatitis D	California	Toxic shock syndrome	North Carolina
Hepatitis E	California	Trichinosis	Both

(continued)

Table 37.5 (continued)

Condition	State	Condition	State
Human immunodeficiency virus (HIV) infection confirmed	North Carolina	Tuberculosis	Both
Human immunodeficiency virus (HIV) infection, stage 3 (AIDS)	California	Tularemia	Both
Human immunodeficiency virus (HIV), acute infection	California	Typhoid (cases and carriers)	Both
Influenza virus infection causing death	North Carolina	Typhus, epidemic (louse-borne)	North Carolina
Influenza, deaths in laboratory-confirmed cases for age 0–64 years	California	Vaccinia	North Carolina
Influenza, novel strains (human)	California	Vibrio infections	Both
Legionellosis	Both	Viral hemorrhagic fevers, human or animal (e.g., Crimean-Congo, Ebola, Lassa, and Marburg viruses)	California
Leprosy (Hansen disease)	Both	West Nile virus infection	California
Leptospirosis	Both	Whooping cough	North Carolina
Listeriosis	Both	Yellow fever	Both
Lyme disease	Both	Yersiniosis	California
		Zika virus infection	Both

Carolina as of December 2016. In general, the lists of reportable occupational conditions are much shorter than those for infectious disease. For example, North Carolina requires reporting of only three occupational diseases: silicosis, asbestosis, and elevated blood lead levels. The Council of State and Territorial Epidemiologists (CSTE) maintains web sites where physicians can look up infectious (http://www.cste.org/?StateReportable) and occupational illness reporting requirements (http://www.cste.org/group/OHWebsites) for their state. Reporting of suspected environmental or occupational causes of illness to federal agencies is not required. Nonetheless, state health departments routinely report selected infectious diseases specified by CSTE and CDC as "notifiable" to CDC in order to support monitoring of national disease trends and to inform national public health policies.

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