Housing and Health

Intersection of Poverty and Environmental Exposures

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The importance of adequate housing for the maintenance of health and well-being has long been a topic of scientific and public health policy discussion, but the links remain elusive. Here we explore the role of the residential environment in the etiology of illness (specifically asthma) and the persistence of socioeconomic health disparities. Housing conditions, shaped by social forces, affect exposure to physical and chemical "toxicants," thereby translating social adversities into individual illness and population health disparities. We discuss the mediating role of housing in determining health outcomes at multiple levels (social–structural, neighborhood, and individual family). To date, little attention has been paid by most environmental health scientists to the social–structural conditions underlying gross inequities in the distribution of toxic exposures, with even less attention to the processes whereby these social conditions may directly affect susceptibility to the toxic exposures themselves. This chapter goes beyond traditional medical and environmental science models to incorporate a range of social and physical determinants of environmental pollutions, illustrating how these conditions result in health and illness. We focus here on childhood asthma as an example of a serious public health problem that has been associated with low income, minority status, and characteristics of the home environment. We end the chapter with a discussion of the environmental justice movement and the role of housing as a potential agent of change and focus of interventions aimed to reduce the harmful effects of environmental pollutants.

Key words: **housing; poverty; environmental toxicants; health**

The world we build for ourselves has profound effects on our health, so that prevention of disease may depend as much on architecture as immunization. —Kellet[93]

Introduction

Poverty, poor housing, and degraded environments are linked to increased risks of many diseases, but apart from a few well-studied examples, the links among poverty, environment, and disease remain elusive.[1] In this chapter we explore the role of the residential environment in the etiology of illness and in the persistence of disparities in health. In particular we explore the concept that variations in housing, shaped by social forces, affect exposure to physical and chemical "toxicants," thereby translating social adversities into health disparities.

The places we live are held together or torn apart by both physical and social infrastructures, and the two cannot be understood independently. The physical infrastructure of the communities in which we live consists of buildings, roads, transportation, water, air, light—each component having its own ability both to support health and to transmit toxic exposures. The "housing" in which we live embodies many elements, including physical/material (e.g., location, density, building height, maintenance, air quality, sanitation, pests, hazardous exposures), social (e.g., threats to safety, noise, social networks, cost), and psychological components (e.g., interpersonal conflict, sense of permanence)—any one of which can affect health. We experience housing at multiple levels (social–structural,

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neighborhood, and individual family), and conditions at each level can affect conditions at other levels. For example, housing quality (neighborhood level) is partly determined by public policy (social–structural level), as for urban renewal. Inadequate city code enforcement and repair of city properties can lead to the systematic deterioration of the housing stock and, consequently, of entire neighborhoods. Neighborhood deterioration affects the social and physical conditions of individual units and hence the well-being of families. This process may permit or lead to environmental pollution. The physical conditions that affect us are not always extreme. They include the chronic stresses of overcrowding, inadequate garbage removal, location near busy transportation routes, and poor ventilation—conditions that are part of the everyday lives of the residents of many urban communities. As a result, both exposure and susceptibility to harmful exposures are unevenly distributed in our society, and the residential environment plays a large role in such inequities.

The social infrastructure that surrounds the places in which we live, consisting of services, social networks, organizational structures, political forces, and human values, can bind communities together or tear them apart, again with consequences for human health and well-being. Disturbances and hazards in the physical system can disrupt the social system and vice versa.[2] The history of urban renewal efforts in this country reflects, for the most part, the failure to take the social consequences of these physical changes into consideration, with the result that whole communities have suffered displacement,[3] fragmentation, and the loss of social cohesion.[4] One of the negative consequences of urban renewal efforts has been the concentration of the most disadvantaged segments of the urban black population in a few areas (as opposed to dispersal)—hypersegregation.[5] Interneighborhood variations in housing costs and crowding are among the clearest legacies of segregation in many urban areas and have been associated with adverse health outcomes, such as high rates of low birth weight.[6] As a result of these historical processes, African Americans tend to live in areas that differ markedly from those of whites, namely, areas characterized by a concentration of low-income housing projects with high levels of crime and social dislocation. Yet Sampson et al. point out that rates of violent offenses, even in low-income neighborhoods, are strongly influenced by variations in family structure (independent of income, region, size, density, age, and racial composition)—specifically, family disruption. In public housing projects, for example, high rates of crime derive from high levels of family disruption as much as from poverty and unemployment.[4]

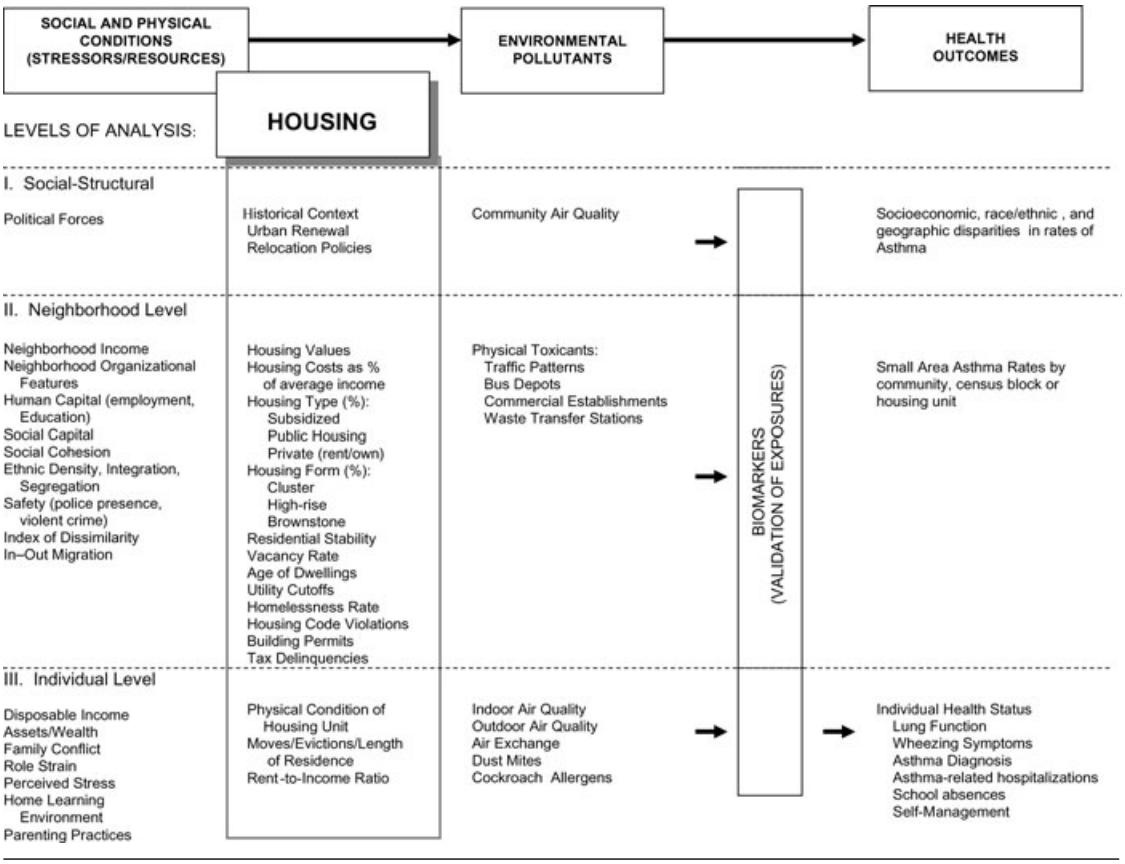
Residential stability has been identified as one of the most important predictors of community health—even more important than standard sociological variables, such as poverty and racial composition. Residential mobility often constitutes a barrier to the development of informal local friendship networks, kinship bonds, and local associational ties. Housing stability is often measured by indices of mobility: (1) the number of times that an individual or family has moved or the length of time in the present dwelling (individual family level), (2) the percentage of individuals or families in the community who have lived in their present dwelling for a specified period (neighborhood level), and (3) patterns of in–out migration in the larger region as a result of immigration policies (social–structural level).

The focus of most research in environmental health science is to identify and quantify associations between toxic physical or chemical exposures and disease outcomes and to do so in relation to one toxic exposure at a time. Consistent with a medical model, the introduction of biomarkers has moved this agenda forward by validating the degree of individual exposure and improving the precision of effect size estimates. Doing so has in turn led to increased technical and programmatic responses to the reduction of environmental pollution, including some positive changes in public health policy.

However, little attention has been paid to date by environmental health scientists to the social–structural conditions underlying gross inequities in the distribution of such toxic exposures in the first place, with even less attention to the processes whereby these social conditions may directly affect susceptibility to the toxic exposures themselves. The continued reliance on a limited medical model, coupled with a focus on singular risk associations, may further refine our ability to identify factors that increase an individual child's chances of having a particular adverse health outcome in response to a toxic exposure but will do nothing to reduce social and racial disparities in disease prevalence.

TABLE 1 lays out the framework for discussing links between housing and health. Of primary interest is the role of toxic residential exposures in translating physical and social conditions into health problems. TABLE 2 provides examples of residential conditions at multiple levels of analysis. We focus here on childhood asthma as an example of a serious public health problem that has been associated with low income, minority status, and characteristics of the home environment.[7–10] We end the chapter with a discussion of the

TABLE 1. Links between environmental exposures and health outcomes mediated by housing



environmental justice movement and the role of housing as a potential agent of change and focus of interventions aimed to reduce the harmful effects of environmental pollutants.

Asthma: Illness in Social Context

Asthma is the most common chronic disease of American children and is the leading cause of pediatric emergency department visits, pediatric hospitalizations, and school absenteeism. Asthma prevalence in the United States increased by 75% between 1980 and 1994, and increases were observed in all races, ethnicities, sexes, and age groups, as well as in all regions of the country. From 1980 to 1996, 12-month asthma prevalence increased both in counts and rates, but no discernible change was identified in asthma attack estimates since 1997 or in current asthma prevalence from 2001 to 2004. During 2001–2003, current asthma prevalence was higher in children (8.5%) than adults (6.7%), females (8.1%) than males (6.2%), blacks (9.2%) than whites (6.9%), those of Puerto Rican descent (14.5%) than those of Mexican descent (3.9%), those below the federal poverty level (10.3%) than those at or above the federal poverty level (6.4%–7.9%), and those residing in the Northeast (8.1%) than those residing in other regions (6.7%–7.5%).[11]

Although the rapid increase in prevalence seems to have leveled off in recent years, there were in 2001 approximately 14 million adults and 6 million children living with asthma in the United States, costing in excess of $12 billion dollars. Similar increases in asthma incidence and prevalence have been seen in other industrialized nations worldwide. The causes of these increases are not well understood and are much debated. Suggested explanations include children's decreased exposure to parasites and other infectious agents in early life (the "hygiene hypothesis"), increased levels of fine particulate air pollution from motor vehicles, and the construction of ever tighter buildings with less and less indoor circulation of fresh air.

TABLE 2. Environmental health exposures at different levels of analysis

Housing parameters	Environmental health factors	
	Physical	Social
Social–structural level		
Historical context	Environmental justice issues	Social justice issues
Urban renewal efforts	State/local environmental regulations	Political equality
Relocation policies	Zoning codes	Human rights
Community level		
Housing values	Outdoor air quality (PAHs, DEP, PM)	Social cohesion
Housing costs (% of income)	Traffic patterns	Community organizations
Housing type (public, private, subsidized)	Location of bus depots and waste transfer stations	Residential stability Safety/violent crime
Housing form (cluster, high-rise, etc.)	Toxic emissions from businesses	Racial segregation
Residential stability	Neighborhood trash removal	Social capital
Vacancy rate	Coal-burning furnaces in schools	Human capital
Age of dwellings	Lead level (soil, built environment)	Crowding
Utility cutoffs	Safety of neighborhood playgrounds	Information channels
Homelessness rate	Cleanup efforts	Community health status
Housing code violations	Pest control (community-wide)	Indicators
New building permits	High-quality food sources	Employment rate
Tax delinquencies	Public transportation	
Individual family level		
Physical condition of housing unit (roof, leaks, holes)	Indoor air quality (NO_2, PAHs, $PM_{2.5}$) Air exchange	Family relationships Sense of permanence
Moves/evictions/length of residence	Home allergens (cockroach, dust mite) Endotoxins	Self-esteem Physical/psychological
Rent-to-income ratio	Lead, mercury	Well-being
Adequacy of utilities	ETS Use of pesticides	Adequate diet Job satisfaction

Asthma incidence, prevalence, and hospitalization rates in the United States are disproportionately high in poor communities, and the highest rates are seen among poor minority children living in inner-city communities, where asthma continues to be epidemic.[8,11,12] In New York City alone, an estimated 300,000 children and 700,000 adults have been diagnosed with asthma during their lifetime,[13] and approximately 260,000 residents have had an asthma episode during the past year. Research has shown childhood asthma prevalence in certain low-income, minority neighborhoods to be as high as 23%, approximately four times the national average.[14] Diminished environmental quality (specifically, disproportionately severe ambient air pollution) has been associated with elevated rates of asthma in poor communities. One of the earliest studies showing that air contaminants were being released more often in areas in the United States where people of low socioeconomic status lived was published in 1970.[15]

Urban residence has been suggested as a possible explanation for the high asthma prevalence in minorities and the poor. It is suggested that because minorities and low-income children are more likely to live in urban areas, they are exposed to environmental factors that contribute to higher asthma prevalence. However, a study in Los Angeles found that racial/ethnic disparities persisted within an urban center, even after adjusting for income and measures of healthcare access.[16] Therefore, it is possible that other measures of low socioeconomic status are more important than individual household income in influencing asthma rates at the population level. Despite the generally high rates of asthma among low-income minority children, most disadvantaged African American and Hispanic children do not develop asthma, suggesting marked variability in either exposures or vulnerabilities, or both, of individuals within these populations. The search for other sources of variability in low-income urban populations must include the contribution of more proximal risk factors, both social and physical, that are potentially modifiable.

Public housing may be a particular risk factor for asthma.[17–20] In New York City, for example, within communities with high asthma rates, asthmatics were five times more likely than nonasthmatics to live in public housing.[8] Also, short-term housing used by transient individuals is likely to be characterized by

poorer-quality management and upkeep, with conditions that can trigger asthma symptoms in those without permanent homes.[21]

An association of asthma prevalence with low-income, inner-city residence has been shown nationwide,[22] yet confounding of socioeconomic disadvantage with ethnicity and place of residence has plagued the literature.[23–25] Several studies have suggested that more severe forms of asthma are related to poverty,[26,27] and these findings are generally consistent with the hospitalization and mortality studies cited earlier. Data from the National Hispanic Health and Nutrition Examination Survey suggest variability in asthma rates among Hispanic groups: 5.2% in Cuban American children, 2.7% in Mexican American children, and 11.2% in Puerto Rican children.[28] A recent study of geographic variations in pediatric asthma rates, sampled by ethnicity and socioeconomic level, showed considerable variation in prevalence of severe asthma (persistent wheeze) by ethnicity, socioeconomic level, and geography.[29] After adjustment for level of community disadvantage (percentage values for overcrowding, being unemployed, being without a car, and being over 65), ethnic differences in prevalence of severe asthma disappeared, but some geographic variation remained. Although the persistent geographic variation in severe asthma rates may be due, in part, to the poor management of asthma in poorer areas, the authors[29] suggest that material and behavioral characteristics associated with poverty, such as parental smoking, air pollution, housing conditions, and allergens, may contribute to the disparities. To date, virtually no studies have applied state-of-the-art geographic methods to study the structural aspects of the asthma problem.

Disproportionate Exposure to Physical Environmental Toxicants and Home Allergens

Inequities in exposure to toxicants have undoubtedly contributed to the disturbing picture of disease and disability among disadvantaged populations, although the precise role of each exposure is not known. It is generally agreed that the residents of economically disadvantaged communities are more likely to be exposed to pollutants than those who live in more advantaged communities, but not all disadvantaged communities are alike. Across the United States, Latinos and African Americans are disproportionately exposed to many environmental hazards, including air pollutants.[30] In the United States, 60% of Hispanics and 50% of African Americans, compared with 33% of Caucasians, live in areas failing to meet two or more of the national ambient air quality standards. More than 30% of Hispanics and 16% of African Americans live in areas that do not meet the standards for particulate matter (PM).[31] Exposures to polycyclic aromatic hydrocarbons (PAHs) in particular are high among low-income, urban, and minority populations, largely because of the uneven distribution of outdoor pollution sources. These same minority populations are also more likely than others to experience poverty and a range of adversities that accompany poverty, including substandard housing, poor nutrition, and inadequate health care.

Next we review common air pollutants, allergens, and mold, and we show how disproportionate exposure to these toxicants, mediated by residential characteristics, can result in adverse health outcomes (childhood asthma) at the individual level and health disparities at the population level.

Particulate Matter, Polycyclic Aromatic Hydrocarbons, Diesel Exhaust Particles

Ambient PM levels result from regionwide pollution emissions as well as local sources of combustion. Foremost among the local sources of PM are cars, trucks, and buses using the network of highways, commercial truck, and bus routes. Also, diesel bus depots, waste incinerators, and industrial operations release substantial amounts of airborne particulates in these areas. Diesel engines emit 30–100 times more particles than are emitted by gasoline engines with contemporary emission-control devices. Sources of diesel particulate, often located in the poorest sections of town, include diesel bus garages, marine transfer stations, and commercial bus terminals. Such sources produce high concentrations of fine elemental carbon particles in the surrounding area, posing a hazard for individuals who live nearby.[32]

There is substantial individual variation in exposure to PM by location, in part because total individual exposure to respirable particles (PM of diameter $< 2.5\ \mu m$ [$PM_{2.5}$]) depends on particles encountered both indoors and outdoors. Because nearly all our time is spent indoors, exposures that occur indoors, especially at home, are of prime importance. The single largest indoor source of $PM_{2.5}$ is cigarette smoking. Other important indoor sources include cooking fumes; dust from carpets, furniture, and clothes; and emissions from stoves and kerosene heaters.[33] Not surprisingly, emissions are higher from appliances that are faulty or in need of repair. Local outdoor PM concentrations depend both on the $PM_{2.5}$ background as well as on the proximity of local combustion sources, such as

diesel vehicles.[32] Physical characteristics of dwellings, such as interior volume, window draftiness, and air temperature, affect both the buildup of particles generated indoors and the penetration of outdoor particles.[34] Again, poor quality or substandard housing tends to contain higher exposures to these pollutants.

PAHs are widespread pollutants commonly found in air, food, and drinking water.[35] Incomplete combustion of organic material is the major source of PAHs. PAHs are listed among the 189 hazardous air pollutants covered under the Clean Air Act. They are present in airborne PM mainly from combustion of gasoline and diesel fuels, coal and oil for residential heating, tobacco products, and other organic materials. In general, emissions from motor vehicles and residential heating are the major sources of PAHs in outdoor air, whereas secondhand smoke is usually the major indoor source. Individual airborne PAH exposure varies and depends on lifestyle; season; and proximity of residence to roadways, incinerators, and industrial sources, as well as indoor sources, such as secondhand smoke and indoor heating. As for PM and diesel exhaust particulates (DEP), concentrations of PAHs tend to be higher in deteriorated housing stock and lower-income areas.

There is strong epidemiological evidence that levels of PM of diameter $< 10\,\mu m$ (PM_{10}), and especially $PM_{2.5}$, exacerbate asthma in children.[36–38] Children's exposure to PM_{10} has been associated with restricted activity, school absences, increased hospital admission for acute respiratory disease, increased respiratory symptoms, and decreased lung function.[36,39,40] Also, epidemiological and experimental data indicate that DEP and fine particles in general can increase the allergic response to antigens, hence risk of asthma. Associations have also been observed between respirable particulates and reduced birth weight and other developmental deficits.[36,41,42]

Environmental Tobacco Smoke

Recent research indicates that exposure to secondhand or environmental tobacco smoke (ETS) is more prevalent in low-income households and among African Americans and Hispanics than in Caucasians.[43–45] Furthermore, higher levels of cotinine and a tobacco-specific carcinogenic compound, nitrosamine, have been reported in black smokers than in Caucasian smokers, after controlling for self-reported amount of smoking.[45,46] African American children have been reported to have twofold higher cotinine levels than Caucasian children as a result of exposure to one cigarette per day.[47] Similarly, after adjustment for cigarette dose, cotinine levels in pregnant women were higher in African Americans than in Caucasians, whereas the rate of decrease in infant birth weight per nanogram of maternal cotinine was similar in the two groups.[48] These findings point to the possibility that cigarette smoking has a more deleterious effect on fetal development among African Americans than among Caucasians.[48]

ETS is a known risk factor for asthma.[49] ETS may also increase the ease of allergic sensitization by altering the integrity of the epithelial barrier.[50] Prenatal exposure and/or infant postnatal exposure to ETS have been associated with childhood asthma.[51–53] However, the effects of pre- and postnatal ETS on asthma have not been well differentiated., nor is it clear if the effect of prenatal exposure to EST on childhood asthma is mediated by low birth weight, which is itself a risk factor for childhood asthma.[51]

Nitrogen Dioxide

Nitrogen dioxide (NO_2) from gas stoves and other sources has been implicated as a contributor to asthma in some studies, but the epidemiological evidence is equivocal.[36] Indoor combustion appliances, especially gas-burning stoves and ovens, are major sources of NO_2, and indoor concentrations can reach levels well above the outdoor air quality standard when gas appliances are being used. One of the few studies to examine indoor residential NO_2 levels in underprivileged neighborhoods found concentrations that were much higher than those previously measured in middle-class homes.[52] The authors speculated that this effect may have been due to inadequate maintenance of burners or to the use of gas ovens as supplemental apartment heating sources. Again, low-income families with faulty appliances and inadequate heating as a result of substandard housing are disproportionately exposed to potentially toxic conditions, and NO_2 has been implicated in asthma symptoms.[53]

Allergens

Goldstein *et al.*[52] have reported levels of airborne cockroach allergen in low-income New York City apartments that were orders of magnitude higher than those seen in other cities. Sarpong *et al.*[54] found that African American race was a predictor of higher allergen exposures. Eighty-five percent of the homes of inner-city asthmatic children had detectable cockroach allergen levels. Although little is known about the role of rodent allergens as triggers of asthma among inner-city children, preliminary evidence suggests that sensitivity to rodent allergens may be more prevalent among low-income children who are frequent emergency department users than among a matched control group.

The contribution of indoor allergens to childhood asthma has been well documented.[55–59] Early childhood exposure appears to be a risk factor for development of allergy and asthma symptoms in people who are genetically predisposed; and in sensitized individuals, continued exposure appears to promote ongoing airway inflammation and hypersensitivity to other irritants. Allergic sensitization to cockroaches has been related to the level of bedroom allergen exposure in children, with higher exposures among African American[54] and other low-income urban populations.[60]

Various airborne triggers in the home environment play a large role in the causation and exacerbation of asthma.[8,17] These triggers include secondhand cigarette smoke; pesticides; and a variety of allergens, most notably dust mites, cats, mice, and cockroach antigens.[20,61,62] Indoor levels of all these triggers are disproportionately high in poor communities.[63] For example, deteriorated housing has been associated with high cockroach allergen levels in urban homes.[21]

There is some evidence that the distribution of cockroach allergens is influenced by characteristics of the built environment, such as building design and management,[64] type of foundation,[65] and type of dwelling (apartments versus houses).[66] Goldstein *et al.* reported extremely high levels of airborne cockroach allergen in Harlem apartments, with 85% of the homes of inner-city asthmatic children having detectable cockroach allergen levels.[52] Although several studies have investigated associations between type of housing and cockroach allergen levels,[59,67] none have used a measure of housing deterioration.

The significant contribution of indoor allergens to asthma has been documented in several studies.[55–59] The emerging evidence suggests that indoor aeroallergens may play at least two roles in allergy and asthma. Early childhood exposure appears to be a risk factor for development of allergy and asthma symptoms in people who are genetically predisposed, and in sensitized individuals, continued exposure appears to promote ongoing airway inflammation and hypersensitivity to other irritants.

The importance of cockroach allergens and their relationship with urban housing has emerged over many years.[52,59,68,69] The rate of allergic sensitization to cockroaches was directly related to the level of bedroom allergen exposure in a group of children studied in Maryland.[54] Also, African American race was a predictor of higher allergen exposures. The National Cooperative Inner-city Asthma Study found that among children who were both exposed and sensitized to cockroach allergen, there were statistically significant elevations of asthma symptoms, doctor visits, missed days of school, and other adverse outcomes. These results lend weight to the growing concern about indoor allergen exposures to disadvantaged urban residents. Many studies have documented the association between increased incidence of asthma and specific levels of dust mites in the home[70] and specific levels of mite allergen.[56,71] Immunoglobulin E antibodies to common inhalant allergens, including dust mite, cockroach, and cat allergen, are a risk factor for emergency department visits, and dust mite and cockroach allergens are the most common allergens in low-income, inner-city populations.[60]

Mold

Mold is an important home-related trigger for asthma to which the poor are disproportionately exposed. Mold (usually *Aspergillus* species) tends to grow where there is moisture, and therefore the two prime locations for mold formation in urban apartments are the kitchen and the bathroom.[72] Housing conditions that predispose to mold formation, especially in older and poorly maintained buildings, are plumbing leaks, roof leaks, and inadequate ventilation.[10] Mold triggers asthma by releasing mycotoxin-containing spores into the indoor atmosphere. Once inhaled, this mycotoxin can inflame the pulmonary tissues, triggering asthma.[10,73]

Lead

Lead is probably one of the best-known developmental toxicants, with a long and troubled regulatory history.[74] Although this contaminant is not implicated in respiratory health outcomes, such as asthma, we include it here because of its close association with housing and its important regulatory history. An estimated 4.6% of African American children have blood lead concentrations $>25\ \mu g/dL$, compared with 1.2% of white children.[75] In New York City, lead poisoning is found almost exclusively among African American and Hispanic children.[75,76] The major source of lead exposure for American children is lead-based paint in housing built before 1978, when lead-based paint was banned in the United States.[77] The most lead-painted homes are found in the northeastern and midwestern states.[78,79] The major route of children's exposure to lead from paint is via ingestion of lead-contaminated dust that forms inside homes from the abrasion, flaking, and chipping of lead-based paint. Children between the ages of 1 and 6 years are at highest risk of lead exposure because the oral exploratory, hand-to-mouth behavior that is so normal in this age group facilitates transfer of lead dust from the environment into children's bodies.

Even exceedingly low levels of exposure to lead are associated with an increased risk of disability and disease.[79] In children and adolescents, diminished intelligence, shortening of attention span, reading problems, attention deficit–hyperactivity disorder, school failure, delinquency, and criminal behavior are the consequences of exposure to lead. There is no evidence of a threshold below which lead does not cause these effects. Moreover the decrements in intellectual function per unit increase in blood lead concentration are greater at blood lead levels $<100\,\mu g/L$ ($10\,\mu g/dL$), the level considered acceptable by the World Health Organization. On average, there is an estimated decline of two to three IQ points for children whose blood lead levels rise from 100 to $200\,\mu g/L$ ($10–20\,\mu g/dL$) but a decline of four to seven IQ points for children whose blood lead levels rise from $10\,\mu g/L$ to $100\,\mu g/L$ ($1–10\,\mu g/dL$).

Much progress against lead poisoning has been made in the United States in the past 25 years. The incidence of childhood lead poisoning has been reduced by more than 90%, and the mean blood lead level has declined by more than 90%. These gains have resulted principally from the removal of lead from gasoline. Nonetheless, more than 300,000 U.S. children still have blood lead levels of $10\,\mu g/dL$ or more. Moreover, these elevated blood lead levels occur disproportionately in poor children, especially poor African American children, living in inner-city communities. The consequences of this continuing, disproportionately high prevalence of elevated lead levels in low-income communities include cognitive deficits and delays, leading to poor learning, diminished lifetime accomplishment, and perpetuation of poverty.

For families who are poor, the likelihood is high that they will be forced to live in substandard, frequently older housing,[80] despite often paying more than 30% of their income for rent.[81] The housing in which poor people live often has structural damage, which if not corrected can further increase risk of exposure to home-related environmental hazards.[81,82]

Burden of Social and Physical Toxicants: Environmental Injustice

The chronic nature and co-occurrence of exposures to multiple chemical toxicants as well as socially adverse conditions pose methodological challenges for risk assessment. In practice, few toxic exposures occur in isolation, and the unfavorable social conditions that underlie pollution typically generate many different kinds of environmental hazards, which tend to accumulate over time. Also, exposures can occur at multiple levels of experience, including the individual and community level. For example, exposure to poverty or substandard housing may be experienced at the individual level (personal income, number of homeless episodes) and the community level (average income in the neighborhood, amount of concentrated poverty, proportion of imminently dangerous buildings in the neighborhood). Furthermore, mechanisms operating at the individual level cannot be adequately understood without reference to group-level data. For example, the effect of individual exposure to toxicants may depend on or be conditioned by community-level conditions and social processes.

A continuing source of debate has been whether it is income and market forces or minority status and racism that drive the distribution of environmental pollution among populations. This question was the focus of a longitudinal study in Los Angeles, California, that examined the distribution of sources of environmental pollution in relation to changes in demography starting in the 1970s. The study showed that polluting industries purposely moved into disenfranchised minority communities. At the same time, minority communities did not move to polluted areas seeking less expensive housing.[83] Other studies have shown that minority ethnicity or race are stronger predictors of poor health disparities and environmental injustice than income level.[84] Although more research needs to be conducted to disentangle the role of minority status and economic level on environmental exposure, the two factors clearly interact in complex ways with social and political factors that lead to unfavorable health outcomes in the poor.

The co-occurrence of hazardous exposures and social adversities in this society represents a type of environmental injustice, in which the greatest toxic burden is carried by those who can least afford the adverse health consequences—a unifying theme in the examples that we describe in this chapter. The term *environmental injustice* describes the disproportionate and inequitable exposure of poor and minority populations to hazards in the environment. The concept of environmental injustice emerged from community-based research undertaken in the American South in the 1980s that documented that a disproportionate number of waste sites housing hazardous materials were being placed in African American neighborhoods.[85,86] This concept has become an important tool for analyzing connections among poverty, environmental degradation, and poor health.

Examples of environmental injustice that have been well studied in the United States include

the disproportionate exposure of poor and minority communities to lead paint, hazardous waste sites, air pollution, substandard housing, dangerous jobs, and polluting industries. As illustrated in this chapter, such environmental risk factors are disproportionately concentrated in poor communities and in communities inhabited by people of color.[87,88] Disproportionate exposures of persons in these communities, especially children, to toxic environmental hazards appear to contribute significantly to well-documented disparities in the incidence and prevalence of asthma, lead poisoning, neurodevelopmental disability, and other chronic health problems. Beyond childhood, the disproportionate exposure in early life of children in poor communities to environmental toxins sets the stage for a lifetime of suboptimal health and thus helps to perpetuate the intergenerational cycle of underachievement and poverty. The disproportionate employment of poor and minority workers in hazardous jobs is a major factor that underlies observed disparities in occupational disease and injury. Environmental injustice is highly correlated with other factors that link poverty with poor health, including inadequate access to medical and preventive care, lack of availability of healthful foods, lack of safe play spaces for children, absence of good jobs, crime, and violence.

Environmental Justice Movement

To this day, the environmental justice movement continues to work out of grass-roots organizations, sparking important policy actions at the community level. Many of the poor communities involved in this movement partner with institutions of higher learning to gather data documenting environmental injustice. Before 1990, there was little potential for legal or regulatory redress for these communities. However, the movement spurred the U.S. Environmental Protection Agency to produce a report and recommendations for addressing the issue of environmental injustices against poor and minority communities.[89] This report formed the basis for the Environmental Justice Executive Order 12898 signed by President Clinton in 1994, in which it is mandated that federal agencies shall aim to achieve environmental justice as part of their mission and shall address, as appropriate, the disproportionately high environmental health effects of policies and regulations on minority and low-income populations. The Environmental Protection Agency remains the federal agency charged with addressing most environmental justice issues. In the executive order, environmental justice is defined as follows:

> The fair treatment and meaningful involvement of all people regardless of race, ethnicity, income, national origin or educational level with respect to the development, implementation and enforcement of environmental laws, regulations, and policies. Fair treatment means that no population, due to policy or economic disempowerment, is forced to bear a disproportionate burden of the negative human health or environmental impacts of pollution or other environmental consequences resulting from industrial, municipal, and commercial operations or the execution of federal, state, local and tribal programs and policies.

This concept of environmental justice entails that no one community should be targeted to bear the consequences of environmental pollution on the basis of their socioeconomic or demographic characteristics.

Housing as an Instrument of Change

Can interventions and policies aimed at redressing environmental injustices reduce health disparities? We know that many of the factors that influence housing quality can be changed by changes in policy, such as decisions about where to build a housing project, what materials to use in construction, enforcement of municipal codes, rehabilitation of existing residential units, and dispersal of the disadvantaged, yet we do not know if such interventions will improve health. For example, residential management of public housing may increase housing stability, tenant buyouts may increase home ownership and commitment, and rehabilitation of existing residential units and strict code enforcement may prevent physical deterioration. However, the links among such neighborhood-level interventions, reduction in exposure to toxicants, and real health improvements at both the individual and the population levels remain to be studied. As advocated by former U.S. Surgeon General David Satcher, one of the best community-level strategies to reduce the effect of hazardous pollutants is the prevention of inappropriate and environmentally unjust siting of pollution sources (conference sponsored by the Columbia Center for Children's Environmental Health, "The Health of Our Children in the Urban Environment," March 27, 2000).

It is possible to reduce the health disparities associated with living in poverty and in environmentally dangerous housing. However, it is difficult for the poor alone to implement the changes needed. Many low-income tenants usually live in rental properties.[72,90,91] Tenants have little or no decision-making power to fix structural problems (such as chipped lead-based paint or leaking pipes) that increase exposure to environmental hazards. Also, relocating from a home with a hazardous indoor environment to a place with a healthier one is economically difficult for the poor.[91]

Community groups and government can play a key role in helping to reduce home-related environmental exposures among the poor. One strategy is to raise awareness about the dangers of environmental hazards among low-income tenants. Rothman *et al.*[78] described an educational campaign initiative to educate residents of North Philadelphia, Pennsylvania, about the dangers of lead. At the end of the 3-year intervention, the authors described a 27% reduction in venous blood lead levels greater than 14 μg/dL among the intervention group. In New York City, mold has become a widespread problem. This concern prompted the Public Advocate of the City of New York to produce a report, *Unhealthy Exposure: Mold in New York City Homes*. The government report, in addition to raising awareness, also makes recommendations on how to reduce exposure among residents through legislation.[92]

Another example of how community-based organizations and government can help the poor reduce their exposure is assisting in offsetting the high expense of relocating once a home-related environmental hazard is discovered. McLaine *et al.*[91] described an initiative in Baltimore, Maryland, that provided financial, housing, and social work assistance to low-income families affected by lead to help them move to safer housing. Lead-poisoned children relocated through the program had a significantly lower blood lead level than the children who did not move. On the legislative side, the state of Maryland passed a law, *Lead Risk Reduction in Housing*, which provides rent subsidies to families with lead-poisoned children paid for by the property owner, who is also responsible for relocation expenses.

Conflicts of Interest

The authors declare no conflicts of interest.

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