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Social Determinants of Health: Implications for Environmental Health Promotion

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In this article, the authors draw on the disciplines of sociology and environmental and social epidemiology to further understanding of mechanisms through which social factors contribute to disparate environmental exposures and health inequalities. They propose a conceptual framework for environmental health promotion that considers dynamic social processes through which social and environmental inequalities—and associated health disparities—are produced, reproduced, and potentially transformed. Using empirical evidence from the published literature, as well as their own practical experiences in conducting community-based participatory research in Detroit and Harlem, the authors examine health promotion interventions at various levels (community-wide, regional, and national) that aim to improve population health by addressing various aspects of social processes and/or physical environments. Finally, they recommend moving beyond environmental remediation strategies toward environmental health promotion efforts that are sustainable and explicitly designed to reduce social, environmental, and health inequalities.

Keywords: *social determinants of health; health disparities; environmental health promotion; health promotion interventions; population health*

The proximate goal of this article is to contribute to the conceptualization of social determinants of health, with a particular focus on environmental health issues; the more ambitious long-term goal is to better ensure that environmental health promotion programs are explicitly designed—and therefore more effective—in decreasing disparities in health within and across population groups. Our particular interests lie in understanding how social and environmental inequalities contribute to health disparities. For instance, inequalities resulting from racism and the distribution of material resources lead to unequal *burdens* of physical, chemical, and biological exposures (i.e., disparities in environmental exposures) within and across communities (both geographically and socially defined). Social inequalities also contribute to unequal *impacts* of environmental exposures as communities experience differential access to resources that help to

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mitigate the negative effects of unequal burdens (e.g., nutritious foods and quality medical care).

Drawing on our respective disciplines of sociology and epidemiology, we delineate here various mechanisms through which social factors contribute to disparate environmental exposures and thus contribute to health inequalities. We first propose a conceptual framework for understanding environmental health disparities that builds on and integrates other frameworks. Next, we examine the interface between social inequalities and four environmental health concerns in order to illustrate diverse mechanisms through which socioeconomic inequalities and aspects of the physical environment together shape health disparities by race/ethnicity, social class, gender, sexuality, and other socially defined categories. We end by recommending strategies for moving environmental health promotion efforts beyond environmental remediation and toward sustainable health promotion that may ultimately reduce social inequalities, redress the disproportionate burdens of environmental toxins, and help in eliminating health disparities.

A CONCEPTUAL FRAMEWORK FOR UNDERSTANDING SOCIAL AND ENVIRONMENTAL INEQUALITIES

The conceptual framework we propose here for understanding the implications of social inequalities for environmental health emphasizes the interplay of social processes with features of the physical environment. In constructing this framework, we have built on and synthesized earlier models (notably ours) that examine relationships between social inequalities, the physical spaces within which people reside, and population health. Specifically, we adapted a conceptual model for understanding racial disparities in health that was developed by Schulz and colleagues,¹ drawing on the joint urban planning and public health framework of Northridge and Sclar.² Our joint model specifically outlines the multiple and dynamic pathways through which underlying social, political, and economic conditions influence aspects of the environment, thereby affecting individual and population health and well-being.

In discussing environmental exposures that have the potential to harm (or promote) health, an inclusive model would allow for examination of any physical, chemical, or biological pollutant (or promoter) of the air, water, soil, or biota. Because this environmental scope is too broad to cover adequately in one article, we have elected to focus attention here on a narrower range of exposures, namely, those that centrally involve the *physical* environment, consisting of the *built* environment and the *natural* environment. We consider the *built* environment to encompass all of the buildings, spaces, and products that are created or significantly modified by people and the *natural* environment to comprise everything else. The model we developed and present below, titled *Social Determinants of Health and Environmental Health Promotion*, helps to delineate how social, political, and economic processes—operating singly or in combination—interface with features of the built environment (land use, transportation systems, and buildings) to influence population health (Figure 1).

Here we posit that, at the macro level, social relationships are produced and reproduced through social and cultural institutions and legal codes that institutionalize systems of inequality. These systems include but are not limited to inequalities based on race/ethnicity and racism, social class and class bias, gender and sexism, sexuality and heterosexism, and age and ageism. In the United States, economic and political orders, legal codes and human rights doctrines, and social ideologies have historically played leading

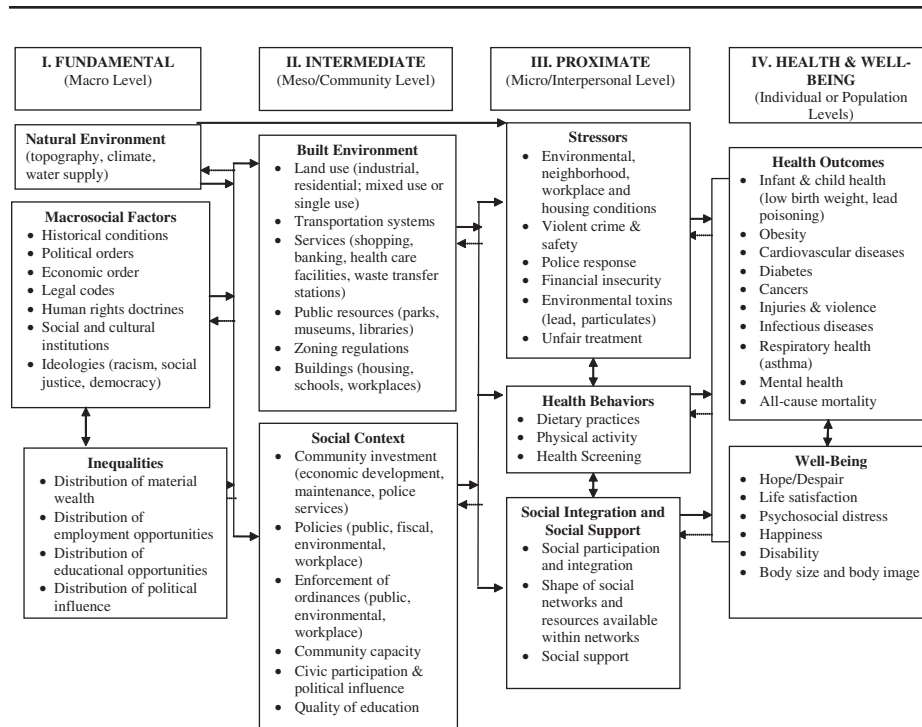


Figure 1. Social determinants of health and environmental health promotion.

NOTE: The model presented in Figure 1 is adapted from a conceptual model for understanding racial disparities in health that appears in Schulz et al.¹ In keeping with our emphasis here on social determinants of environmental health disparities, the model has been modified to specifically examine relationships between social inequalities, the built environment and social context, and environmental health disparities, drawing on Northridge and Sclar.²

roles in creating spatial concentrations of poverty and wealth, and in many regions, spatial concentrations of African Americans and other racial/ethnic groups in areas with circumscribed access to social, political, and material resources.^{1,3-5} These political, economic, and legal processes, and the unequal distribution of material resources that they produce, are included in our model as *fundamental* factors, that is, factors that affect health by influencing access to multiple resources that are necessary to maintain health.⁶⁻⁸

The spatial concentration of poverty and wealth influence both the built environment and *social context*, which we include in our model as *intermediate* factors (also called mesolevel) in pathways that eventually influence health and well-being. For example, within areas of greater concentrations of wealth live individuals and groups with the social and economic resources to influence political decisions; this is less likely to be the case in areas of greater concentrations of poverty. In the contemporary United States, such resources might translate into greater power to influence public, fiscal, environmental, and workplace policies regarding infrastructure investments and economic development (denoted here as *social context*). These, in turn, influence features of the built environment, including where to site services such as waste transfer stations, transportation systems such as highways, and buildings such as affordable housing for working-class

and impoverished families. Communities with fewer social, economic, and political resources may consequently experience greater environmental exposure to noxious land uses, along with their associated physical and psychosocial *stressors* (considered here as *proximate* factors), and exposure to environmental toxins, including lead and particulates.^{9,10}

Similarly, spatial concentrations of poverty and wealth influence the resources available to support—and therefore the adequacy of—community infrastructure, for example, public resources such as parks, museums, and libraries (i.e., the built environment). The distribution of poverty and wealth also influences, and is influenced by, community investment (economic development, maintenance, and police services) and the enforcement of ordinances (public, environmental, and workplace), all of which are aspects of the social context within which individuals reside.

The *intermediate* factors shown in our conceptual model are of particular interest as we consider environmental health disparities. It is here that we believe societies and communities may intervene to both “push up” against the fundamental factors that contribute to health disparities through multiple pathways and at the same time directly influence *proximate* factors that lead to both individual and population health and well-being.

Proximate factors are observable at the *personal* or *interpersonal* level. Neighborhoods with higher concentrations of poverty may experience increased exposure to *stressors* that are detrimental to health, for example, violent crime, financial insecurity, and unfair treatment such as discrimination, even as they experience reduced access to resources that may promote *health behaviors*, for example, grocery stores with fresh produce and fruit that influence dietary practices, well-maintained public spaces such as parks and waterfronts that facilitate physical activity, and affordable public transportation that improves access to health screenings. Communities with inadequate resources may be unable to maintain sidewalks or walkways (an intermediate-level factor), contributing to decreased physical activity (a proximate factor) and increased risk of obesity-related conditions as well as rates of falls and unintentional injuries (*health outcomes*). In areas where financial resources are inadequate to maintain a culturally competent police force (see police response under *stressors*), residents may experience increased rates of anxiety (a *mental health outcome*) and police brutality (injuries and violence). *Social integration and social support* are also influenced by intermediate-level factors, for example, the availability of safe and well-maintained public spaces for social interaction, and violent crime and resulting concerns about safety that lead citizens to curb outdoor activities and informal interactions among neighbors. Proximate factors such as exposure to stressors, health behaviors, and social integration and social support have been shown empirically to be related to health and well-being.¹¹⁻¹⁴

In summary, the emphasis in our conceptual model of Social Determinants of Health and Environmental Health Promotion (Figure 1) is on the implications of social and economic inequalities (fundamental factors) for the built environment and social context (intermediate factors) that influence stressors, health behaviors, and social relationships (proximate factors) that ultimately result in individual and population *health and well-being*. We recognize the limitations of presenting social processes and environmental effects as a series of boxes and arrows as they are far more complex than what is visually represented in Figure 1 (e.g., direct effects of the natural environment or the built environment on health outcomes are not represented in the model, although clearly such effects occur, as with natural variations in the amount of fluoride in the water supply that influence oral health). Nonetheless, there is a need for clarity in thinking through the pathways whereby social processes and physical environments interface to affect health. Thus, our

hope is that this “simplified” visual tool may aid explicit consideration of the linkages between *macro*, *meso*, and *micro* phenomena that affect individual and population health.

Clarification Regarding the Term *Fundamental*

A potential misinterpretation of our conceptual model requires public comment. We use the term *fundamental* to denote historically intransient factors such as racial/ethnic and socioeconomic inequalities that have resulted in egregious health disparities by race/ethnicity and social class among population groups. Our use of the term *fundamental* does not mean that racial/ethnic and socioeconomic disparities are static and unchangeable. Neither do we wish to suggest that it is futile to intervene at intermediate or proximate points in the model to counteract the effects of fundamental factors on health inequalities (note that the arrows in Figure 1 are bidirectional). Rather, as defined here and used in our model, fundamental factors are those phenomena that influence multiple health outcomes and that operate through multiple pathways and mechanisms to influence access to the resources necessary to maintain health. Although traditionally, health promotion has focused attention on more proximate factors to improve specific health outcomes, in the absence of explicit attention to intermediate and fundamental inequalities, the relationships between unequal social conditions and health—namely, health disparities—are likely to persist. Our model emphasizes the dynamic nature of the social processes that produce and maintain health disparities under conditions of social and economic inequalities.

Dynamic Systems and Their Implications

There are at least four important implications of the dynamic nature of our model for health promotion interventions. First, because the systems and processes that sustain inequalities are themselves dynamic, these systems and processes have the potential to eliminate inequalities in the same way that they have the potential to sustain them. That is, fundamental inequalities are produced and reproduced through social actions. They have the potential for change, although that potential must be measured against the weight of the historical forces that have sustained inequalities over time. Environmental health promotion interventions may be able to interrupt the dynamic processes that produce inequalities and move those systems toward greater equality. Such health promotion interventions may target systems or policies not commonly considered within the purview of the health sector, namely, intermediate factors.

An illustrative example is prevailing wage and other types of living wage policies that require employers to pay workers wages that are, for example, sufficient to raise a family of four above the poverty line.¹⁵ Living wage policies would be located under *social context* in the intermediate column in our model and have implications for other intermediate-level factors such as access to public resources, community investment, and the quality of education available to community residents. These intermediate-level factors would be expected in turn to influence more proximate factors associated with health and well-being, such as stressors (e.g., through reducing financial insecurity), health behaviors (e.g., through making more nutritious foods affordable), and social integration and social support (e.g., through making it possible to work fewer hours or fewer jobs to make ends meet, and thereby spending more time with friends and family). In addition, in the long term, living wage policies may influence more fundamental factors such as economic inequalities by redistributing wealth from employers to workers.

The second important implication of the dynamic nature of our model for health promotion interventions is that each aspect of the system is related to, and thus may influence or be influenced by, the others. As illustrated in the living wage example above, more proximate factors may be influenced by changes in fundamental or intermediate factors, whether those changes are a result of intentional interventions or are unintentional effects of other changes. Thus, efforts to reduce health disparities by intervening on intermediate or proximate factors must take into consideration the influence of more fundamental factors. An example would be an intervention that seeks to influence behavior by increasing knowledge about determinants of cardiovascular disease. The ability to act on this information will be influenced by access to produce that is rich in the nutrients that are currently thought to be heart healthy (e.g., financial resources necessary to purchase those foods, as well as spatial proximity to retail outlets and farmers' markets that sell fresh produce) and physical environments that are conducive to physical activity (e.g., safe from violence and crumbling sidewalks, "walkable" communities with interesting street scenes and bike lanes, and accessible parks and other recreational facilities). Thus, if health promotion campaigns fail to attend to disparities in the ability to act on available knowledge, they may improve the health of populations with greater access to resources more than the health of populations with reduced access to resources; in other words, they may actually exacerbate, rather than reduce, health disparities.

Third, our model emphasizes the influence of fundamental factors on intermediate and proximate factors (i.e., from left to right in Figure 1), but the dynamic nature of the systems represented by this model also allows for potential influence within levels (represented by bidirectional arrows within columns). And finally, although there are historic moments when shifts occur in fundamental factors, such as the creation of the United Nations in 1948 that acknowledged international responsibility for health and human rights,¹⁶ more often it is the slow forces of change that push up against these underlying factors, for example, the buildup of carbon dioxide from anthropogenic sources, especially automobile use, that influence global climate change.¹⁷ Therefore, we allowed for this possibility by using hatched arrows that are directed from right to left—that is, from intermediate to fundamental factors—in our model.

ILLUSTRATING THE UTILITY OF OUR CONCEPTUAL FRAMEWORK

Next, we use our developed model to trace several pathways through which social inequalities intersect with aspects of the physical environment to influence health and well-being. We have selected four case studies to illustrate key points, two that deal with unequal environmental exposures (diesel exhaust and lead) and two that deal with social disparities in health and well-being (unintentional injury and psychosocial stress). It is of particular interest here to both identify the intervention points (levels) for each case study and to determine whether the interventions implemented resulted in more equitable distribution of environmental exposures and/or reductions in health disparities among social groups.

Case Study 1: Diesel Exhaust

Very fine particulate matter formed from combustion such as that found in diesel exhaust has been linked to cancers, cardiovascular diseases, exacerbation of asthma

symptoms, other respiratory diseases, hospital admissions, and deaths.¹⁸⁻²¹ Populations at greatest risk of suffering adverse health effects from diesel exhaust include infants and children, older adults, persons with preexisting respiratory disease and other acute and chronic conditions, and communities near environmental sources, such as major truck and bus routes, truck stops and diesel bus stations, and industrial sites (e.g., petroleum refineries), distribution, and storage sites.^{18,22-29} Pearson and colleagues²⁶ found that lower levels of education are associated with increased health effects from particulate matter, potentially mediated by poorer nutrition, lower access to and use of health care, and higher exposures to diesel exhaust and other air pollutants resulting from closer residence to sources such as highways and industrial facilities. Although the benefits of diesel fuels for the transportation of goods and people are distributed among most of the population, the adverse health effects from diesel exhaust are distributed unequally according to characteristics of the built environment (land use and transportation systems) and social context (civic participation and political influence), both of which are linked in our model to the unequal distribution of material wealth.

In many urban communities, the most important sources of particulate matter are diesel buses and trucks. Efforts to reduce the negative environmental and health impacts of the combustion of diesel fuels include reducing their sulfur content,³⁰ retrofitting older diesel engines to reduce emissions,³¹ tightening emissions standards for new vehicles,³¹ and converting to alternative fuels such as natural gas that produce 1/10th to 1/20th the particulate matter produced by diesel fuels.³² The conversion of bus and truck fleets to natural gas is more cost-intensive than other available options,³¹ potentially making this strategy more available to communities with greater economic resources. Furthermore, communities with greater financial and political resources may be better able to prevent the siting of diesel bus stations in their neighborhoods than communities with less political influence.

Six of the seven diesel bus depots in Manhattan are located in Harlem and Washington Heights, two largely disadvantaged African American and Latino communities in northern Manhattan.³³ The former “Dump Dirty Diesel” campaign provides a cogent example of a health promotion campaign aimed at the intermediate level. The Metropolitan Transit Authority (MTA) in New York City originally planned to convert its bus fleet from diesel fuel to cleaner burning natural compressed gas in Long Island (home to more affluent communities), but not in northern Manhattan (home to largely impoverished communities). The “Dump Dirty Diesel” campaign—spearheaded by the Natural Resources Defense Council, which worked in coalition with a host of local environmental organizations and agencies—was successful in bringing needed political attention to this issue. The MTA subsequently decided to integrate cleaner-burning “hybrid” buses into its northern Manhattan fleet.

The political mobilization of communities with fewer economic and political resources, and the emergence of strong environmental justice groups, illustrates the potential for change in intermediate factors related to environmental health promotion. These groups have successfully advocated for both local and federal policy changes, the most important of which was Executive Order 12898, signed by President Bill Clinton in 1994.³⁴ This order requires federal agencies to “identify and address disproportionately high and adverse human or environmental effects of their programs, policies, and activities on people of color and impoverished communities in the United States and its territories and possessions” (p. 209).⁹ Mobilization of, and sustained action by, local environmental groups, with the support of the larger public, may be successful in reducing environmental health disparities by, for example, influencing local transportation poli-

cies and encouraging enforcement of existing environmental regulations. Such interventions at the intermediate level have implications for more proximate factors (noise, exposure to toxic substances) linked to health outcomes. In addition, local political mobilization may provide the infrastructure for continued social movements directed toward social and economic equality, thus pushing up against fundamental factors.

Case Study 2: Lead

Lead exposure is one of the most common preventable poisonings of childhood. Ingested and inhaled lead can contribute to a lack of attentiveness, hyperactivity, and irritability in children. High levels of lead exposure may result in problems with learning, delayed growth, and hearing loss, whereas very high levels of lead exposure may lead to brain damage and death.³⁵⁻³⁸

Lead exposure may come from a variety of sources, including lead-based paint, most often found in houses built before 1950 in the United States. Homes and cities with older water systems may also have lead in pipes, which results in elevated concentrations of lead in drinking water. The sources of lead contamination in a given community are associated with the distribution of elevated blood lead levels among population groups, with infants and young children at special risk of lead poisoning. A study of blood lead levels among White and Native American children in a former mining community found no differences in blood lead levels by ethnicity but significant differences in blood lead levels by socioeconomic position.³⁹ In communities where the primary source of lead contamination is lead-based paint, poor children are disproportionately exposed to lead compared with wealthier children, due to their greater likelihood of living in older homes, rental properties, and poorly maintained housing.⁴⁰

For the U.S. population overall, blood lead levels have decreased dramatically during the past several decades, from a mean blood lead level of 12.8 $\mu\text{g}/\text{dL}$ in 1976-1980 (National Health and Nutrition Examination Surveys, or NHANES II) to only 2.8 $\mu\text{g}/\text{dL}$ in 1988-1991 (NHANES III).⁴¹ Regulations passed in the 1970s and 1980s to reduce lead in paint and gasoline demonstrate the effectiveness of this intermediate-level intervention (policy and enforcement) in improving *infant and child health*.⁴¹ Remediation programs encouraging residents to apply fresh lead-free paint over older layers of leaded paint have been unevenly successful in controlling the inhalation of dust and the ingestion of lead chips by children living in older housing. Brody and colleagues,⁴² working with NHANES data, found that a greater proportion of non-Hispanic Black children in all income categories continue to have blood lead levels equal to or greater than 10 $\mu\text{g}/\text{dL}$ (the current "safety" level) than do non-Hispanic White or Mexican American children of comparable incomes. Results from this survey demonstrated that among children aged 1 to 5 in the lowest income group, 28.4% of non-Hispanic Black children had blood lead levels equal to or greater than 10 $\mu\text{g}/\text{dL}$, compared with only 9.8% of non-Hispanic White children and 8.8% of Mexican American children. Blood lead levels equal to or greater than 10 $\mu\text{g}/\text{dL}$ were most prevalent for non-Hispanic Black children living in central cities with populations of 1 million or more: Fully 36.7% of children in this group had elevated blood lead levels, compared with 5.2% of non-Hispanic White children living outside of central cities. Thus, while efforts to reduce childhood lead exposure through policies designed to regulate the use of lead in paint and gasoline have been effective for the population as a whole, grave disparities remain in lead exposure, with poor, African American, central-city children at elevated risk for lead poisoning.

Disadvantaged socioeconomic position may contribute to increased susceptibility as well as increased exposure to the detrimental effects of lead. Iron deficiency anemia and inadequate calcium intake may lead to increased absorption and retention of lead in the body. Children who live in households with incomes below the poverty level and/or who reside in poor communities are more likely than economically privileged children to have inadequate dietary intakes of iron and calcium.⁴³

Therefore, to reduce health *disparities* from differential lead exposure, health promotion interventions will need to move beyond policies regulating the sale of lead-based paint and general remediation efforts toward targeted efforts to reduce social disparities in lead exposure. Examples of such targeted efforts are available at both the proximate and the intermediate levels in our model. At the proximate level, the American Academy of Pediatrics Committee on Environmental Health has developed guidelines for universal screening of children living in areas where 27% or more of housing was built prior to 1950 and in populations in which the percentage of 1- to 2-year-olds with elevated blood lead levels is 12% or higher.³⁸ Such prioritized screening programs may identify children with elevated blood lead levels and allow for timely medical intervention but are less likely to contribute to primary prevention of lead exposure.

At the intermediate level, the effectiveness of housing policies in reducing children's blood lead levels was evaluated by Brown and colleagues.⁴⁴ The proportion of children who subsequently resided at addresses where children with lead poisoning had lived was compared in two northeastern states with differing levels of enforcement of their blood-poisoning prevention statutes. Results of this study indicated that children living at addresses where there was limited enforcement of blood-poisoning prevention statutes were four times more likely to be lead poisoned than children living at addresses with strong enforcement of statutes. Therefore, interventions at the intermediate level—such as effective enforcement of existing housing policies designed to prevent lead poisoning, building low- and moderately priced housing for families with limited incomes, and economic development intended to increase the educational and employment opportunities of poor families—may be effective not only in reducing childhood lead poisoning rates overall but in eliminating racial and socioeconomic differences in lead poisoning rates.^{9,44} Without such targeted prevention strategies, remediation efforts may contribute to continued disparities in infant and child health.

Case Study 3: Unintentional Injury

Of all the health outcomes listed in our conceptual model, perhaps the one with the most accumulated evidence regarding links to the built environment is unintentional injuries. Building safe transportation systems (e.g., ensuring that the width of streets and the spacing of traffic signals are such that disabled persons and older adults have sufficient time to cross busy intersections)⁴⁵ and providing safe and affordable housing (e.g., through community-building strategies and enforcement of New York City ordinances that require landlords to keep all properties in “good repair”)²⁵ have strong historical precedent. So, too, does designing and maintaining parks and playgrounds within central cities.⁴⁶

In 1975, a pediatric trauma registry was established at Harlem Hospital Center. Surveillance data from this system showed that 300 children (nearly 1% of the children in the community) were admitted to Harlem Hospital Center annually with severe injuries, many of which resulted from falls through open windows. Working with the New York City Department of Health and the public schools in Central Harlem, an educational cam-

paign titled “Children Can’t Fly” was started to warn parents of the dangers of open windows. Harlem Hospital Center physicians and their supporters successfully lobbied for a New York City law to require window guards in apartment buildings, which went into effect in 1979. During the next 2 years, the number of childhood injuries from falls out of windows dropped by 96% in Harlem.⁴⁷ The community-building strategies employed by this initiative engaged health care professionals, landlords, and community members in a successful, sustained intervention with dramatic improvements in childhood injury rates. This is an example of an intermediate-level intervention (civic participation and political influence) influencing a fundamental determinant of health (i.e., the legal code).

In 1988, in recognition that injuries were the second leading cause of childhood death in Central Harlem, the Department of Pediatrics officially started the Harlem Hospital Injury Prevention Program.⁴⁸⁻⁵⁰ The high childhood death rates from unintentional injuries in Central Harlem were considered to be directly related to a lack of safe places to play (intermediate factors, as part of the built environment), combined with too few organized activities, sports, and after-school programs (proximate factors, as part of social integration and social support). As a result, a spectrum of health promotion programs were created and implemented to intervene at the proximate level, including supervised after-school activities such as art, dance, baseball, soccer, and cycling; a bicycle shop and infant car seat fitting station; a mock city block called Safety City in which third graders are taught pedestrian safety; and the distribution of home safety kits and infant car seats for parents with infants discharged from Harlem Hospital Center. The most intensive program, however, was aimed at the intermediate level: a sustained community mobilization effort to “take back” parks and playgrounds from drug dealers, clean up the discarded needles and crack vials, and install safe surfaces and new playground equipment. To date, 35 new playgrounds have been established for schools and day care centers, and seven new public park play areas have been built in Central Harlem.

In its first decade of existence, the activities of the Harlem Hospital Injury Prevention Program directly involved nearly 20% of children in Central Harlem. This contributed to a reduction of childhood injuries of all types by 50% or more in the community. Through the Injury Free Coalition for Kids, this program has been replicated in 17 cities across the United States, thus better ensuring that this local model has wider ramifications for the prevention of childhood injuries in other communities.

Case Study 4: Chronic Stress

Inequalities in the distribution of material wealth and political power characterize many large metropolitan areas, with residents of older urban areas disproportionately likely to have incomes that are below the poverty line, to live in neighborhoods in which a large proportion of others have similarly low incomes, and to self-identify as non-Hispanic Black or Hispanic.^{3,4,51} Residents of areas in which there are high concentrations of poverty are likely to encounter built environments that are aging and poorly maintained, inadequate enforcement of public and environmental regulations, and reduced opportunities for employment. Schools in resource-poor areas are often in worse physical condition and have more students per classroom than schools in more resource-rich areas. The combination of aspects of the built environment and the social context can contribute to exposure to multiple stressors, including noxious odors, emissions from local industries, illegal dumping, occupational hazards, financial stressors, and concerns about crime and safety.

The physiological response to stressors—allostasis—is an important mechanism that allows the body to respond effectively in the short term. Chronic exposure to environmental stressors, however, can set in motion a complex of physiological responses referred to as “allostatic load,” thereby contributing to decreased cognitive function, abdominal obesity, hypertension and other cardiovascular diseases, insulin resistance, diabetes, and decreased immune response.⁵²⁻⁵⁴

As early as the 1970s, social epidemiologists began to document heightened blood pressure and other chronic health conditions among residents of urban census tracts characterized by economic divestment, lack of trust in police response, and longer firefighting response times (indicators of the adequacy of municipal supports for community life), compared with residents of census tracts with lower rates of poverty and shorter police and firefighter response times.⁵⁵ Documentation of the disproportionate risk of chronic disease morbidity and mortality from multiple causes for residents of high-poverty urban neighborhoods has continued, with a substantial literature now supporting these disparities.^{1,4,56-59}

Many mechanisms have been proposed to account for the disproportionate risk experienced by residents of economically underdeveloped urban communities, including decreased access to nutritious food and an oversupply of fast foods that are high in fat;⁶⁰ high crime and poorly maintained public spaces that reduce opportunities for recreational activity,⁶¹ informal socializing among neighbors,⁶² and the ability of neighbors to enforce collective social norms;⁶³ reduced educational and employment opportunities and quality;⁶⁴⁻⁶⁶ and increased exposure to environmental hazards such as airborne particulate matter and noise.⁶⁷⁻⁶⁹ In other words, residents of high-poverty neighborhoods may experience increased risk of multiple chronic stressors associated with the built environment and social context, which contribute, in turn, to allostatic load. This physiological response to chronic stress, in turn, has subsequent implications for the risk of chronic conditions such as diabetes and cardiovascular disease as well as symptoms of depression and despair, contributing to disparities in these health indicators between residents of more and less advantaged neighborhoods.

There are a number of potential health promotion interventions that may be undertaken at the intermediate level to address disparate exposures to stressful physical and social environments. Examples include housing interventions designed to reduce the density of poverty and improve the quality of housing available to residents,^{40,70} urban planning interventions to increase safety and create safe and attractive spaces for informal socializing,⁴⁵ living wage laws,¹⁵ and efforts to promote racial and socioeconomic diversity as well as meaningful resident representation in local planning and renewal efforts.⁷¹⁻⁷³

To examine one of these potential points of intervention in somewhat more depth, living wage laws passed at the municipal level not only ensure that workers are compensated fairly for their work but prevent local employers from undercutting wages, thus driving down locally available wages.* Living wage laws have also been linked to increased employer investment in training and other employee development efforts, greater racial and ethnic diversity in the workforce, and reduced occupational injuries. Furthermore, Pollin and Luce¹⁵ argue that living wage laws passed at the municipal level are a crucial

*The authors thank Nancy Krieger for giving us permission to draw on her background materials on the living wage case example that she prepared for the conference, “Health Impact Assessment: Perspectives on the Promise and Pitfalls of Measuring Effects of Policy and Politics on Public Health,” held at the Harvard School of Public Health, Boston, August 16-17, 2002. One of the authors (Mary Northridge) was a member of the core conference planning group and contributed to the living wage case example.

component of a broader “policy framework” for reversing the economic decline of cities. They argue that by addressing the contribution of low-wage employment to increasing rates of urban poverty, making a clear statement that the goal of urban economic policies should be to provide a living wage for workers, and providing a positive benchmark against which to assess the type of business firms that the city is trying to attract and retain, living wage laws can contribute to building a network of employers that contribute to the economic stability of the community, rather than exploiting a population with few employment options. Pollin and Luce also suggest that the political mobilization and citizen participation required to establish a local living wage policy may translate to enhanced community effectiveness in mobilizing around other urban development concerns. Although there have been relatively few efforts to assess the health effects of living wage interventions, the accumulated evidence documenting the negative effects on health of residence in high-poverty neighborhoods with older housing stock, deteriorating infrastructure, and limited political influence underscores the importance of continued efforts to intervene on these aspects of the built environment and social context and to document their contributions to health outcomes.

IMPLICATIONS FOR HEALTH PROMOTION INTERVENTIONS TO REDUCE ENVIRONMENTAL HEALTH DISPARITIES

Working through the four case studies using our conceptual model in the previous section helped us to better elucidate possible mechanisms through which social inequalities contribute to health disparities. Many of the intermediate-level interventions (involving the built environment and/or the social context) employ the tools of urban planners who seek to supersede market forces in the development of healthy and sustainable built environments, that is, built environments that attend to the social and material well-being of their residents, as well as the natural systems on which they depend.^{2,74,75} Intermediate-level interventions, carefully applied, can act to reduce health disparities through their influence on proximate factors that are related to disparities in health and well-being. Furthermore, in some cases, they may also “push back” against fundamental social inequalities and macro-level factors, as well as the natural environment, for example, through anthropogenic production of greenhouse gases that contributes to global climate change.

Zoning strategies designed to reestablish more mixed-use communities (i.e., both commercial and residential),¹⁰ as well as incentives to developers for building housing where a subset of homes are designated for families with limited incomes, are two strategies for promoting urban areas with more economic diversity. These intermediate-level interventions can also serve to reduce the concentrations of wealth and poverty that appear as fundamental factors in our model. Similarly, intermediate-level interventions that actively engage current residents and neighborhood organizations in redevelopment planning can contribute to sustainable and vibrant mixed-income communities, at the same time that they encourage political engagement and civic participation.⁷²⁻⁷³ Furthermore, community reinvestment strategies can increase the safety and affordability of homes in urban, suburban, and rural areas, helping to move families currently limited to dilapidated housing stock into homes that are more conducive to health.⁴⁰

Current inequalities in the siting of noxious facilities, for example, bus depots, waste transfer stations, water pollution control plants, and refineries, which disproportionately expose residents of poor communities to toxic substances, must be redressed if the U.S. society is to move toward equality in environmental health exposures. This will require

the allocation of additional public and private funds for the remediation of currently polluted locations, as well as development of proactive social and environmental policies designed to regulate the siting of new facilities (an intermediate-level strategy). Remediation efforts must also be monitored to ensure that sites located near poor communities, communities of color, and rural communities receive additional funds for remediation compared with communities with greater access to economic resources.

Intermediate-level interventions that create safe parks and pathways that encourage play, relaxation, exercise, and social interaction across social class and racial groups can encourage the development of stronger social networks within urban, suburban, and rural communities; foster a greater sense of trust and connectedness among residents; and provide opportunities for physical activity and recreation. Finally, attention is needed to encourage civic engagement and political equity within urban areas, and between urban, suburban, and rural areas. Collaboration among local governments for regional planning is essential if difficult. Care must be exercised to ensure that environmental health promotion interventions at the regional level do not end up sacrificing the health and well-being of less powerful communities for the benefit of more powerful communities.⁷⁶⁻⁷⁹

CONCLUDING COMMENTS: PROMOTING ENVIRONMENTAL HEALTH EQUALITY

The framework for understanding social inequalities as fundamental determinants of health sketched earlier in this article has multiple implications for understanding and promoting environmental health. Our model emphasizes the dynamic and interrelated nature of social processes that both influence and are influenced by aspects of the physical environment, and the implications of these interrelated social and physical environments for health. Although continued environmental remediation efforts are an important component of environmental health promotion efforts and are necessary to redress the effects of processes that have contributed to greater exposure to environmental toxins for some social groups compared with others, it is clear that environmental health promotion must not stop with remediation. The development of sustainable health promotion efforts that eliminate disparities in health requires proactive planning to create equitable access to the resources that are necessary to sustain health as well as to reduce inequalities in exposures to environmental hazards. These efforts must actively resist the reproduction and exacerbation of current health disparities, as well as the creation of new disparities, by addressing the *fundamental processes* that drive inequalities in hazardous exposures and protective resources available in the face of hazards. In other words, environmental health promotion efforts must include the creation of structures and processes that actively work to dismantle existing inequalities and to create economic, political, and social equality.

Unequal social relationships shape health disparities through differences in environmental exposures and access to resources, including access to quality health care. These disparities are created and sustained through dynamic processes that are implemented through legal, political, and economic systems. While health disparities may not be intended consequences of these systems, they are nonetheless important consequences of the inequalities that are reproduced by those systems. Health promotion initiatives designed to change existing patterns of health inequalities need to take into account unequal social relationships that shape health disparities through unequal environmental exposures and unequal access to protective resources. Sustainable change in the existing patterns of disparate environmental exposures and health outcomes within and across

population groups will require building more equality into social relationships at the fundamental level, by intervening decisively at the intermediate level, rather than limiting health promotion efforts to more proximate pathways to individual and population health and well-being.

Efforts to address intermediate and fundamental factors must anticipate both political and economic challenges as well as unintended consequences. For example, efforts to enforce local land use or environmental policies may be met with resistance from those who benefit from less active enforcement. Collaborative efforts that actively engage those outside of the health sector—for example, landlords; transportation officers; urban planners; and local, state, and regional government officials—may be effective in devising effective and sustainable built environment and policy interventions that improve population health. Meanwhile, by being involved in urban planning, housing, transportation, and social welfare initiatives, public health practitioners and researchers may better determine the health effects of public and private initiatives at the population level. Such efforts are essential if we are to carefully elucidate and effectively eliminate the underlying inequalities that drive racial and socioeconomic disparities in health.

References

1. Schulz AJ, Williams DR, Israel BA, Lempert LB: Racial and spatial relations as fundamental determinants of health in Detroit. *Milbank Q* 80(4):677-707, 2002.
2. Northridge ME, Sclar E: A joint urban planning and public health framework: Contributions to health impact assessment. *Am J Public Health* 93(1):118-121, 2003.
3. Jargowsky PA: *Poverty and Place: Ghettos, Barrios, and the American City*. New York, Russell Sage Foundation, 1997.
4. Williams DR, Collins C: Racial residential segregation: A fundamental cause of racial disparities in health. *Public Health Rep* 116:404-416, 2001.
5. Wilson WJ: *The Truly Disadvantaged: The Inner City, the Underclass and Public Policy*. Chicago, University of Chicago Press, 1987.
6. Cassel J: The contribution of the social environment to host resistance: The Fourth Wade Hampton Frost Lecture. *Am J Epidemiol* 104(2):107-123, 1976.
7. House JS, Lepkowski JM, Kinney AM, Mero RP, Kessler RC, Herzog AR: The social stratification of aging and health. *J Health Soc Behav* 35(3):213-234, 1994.
8. Link BG, Phelan J: Social conditions as fundamental causes of disease. *J Health Social Behav* 36(Extra issue):80-94, 1995.
9. Northridge ME, Stover GN, Rosenthal JE, Sherard D: Environmental equity and health: Understanding complexity and moving forward. *Am J Public Health* 93(2):209-214, 2003.
10. Maantay J: Zoning, equality, and public health. *Am J Public Health* 91(7):1033-1041, 2001.
11. Heaney CA, Israel BA: Social networks and social support in health education, in Glanz K, Lewis FM, Rimer BK (eds.): *Health Behavior and Health Education*. San Francisco, Jossey-Bass, 1997, pp. 179-205.
12. Israel BA, Farquhar SA, Schulz AJ, James SA, Parker EA: The relationship between social support, stress, and health among women on Detroit's east side. *Health Educ Behav* 29(3):342-360, 2002.
13. Lantz PM, House JS, Lepkowski JM, Williams DR, Mero RP, Chen J: Socioeconomic factors, health behaviors, and mortality. *JAMA* 279(21):1703-1708, 1998.
14. Yen IH, Syme SL: The social environment and health: A discussion of the epidemiologic literature. *Annu Rev Public Health* 20:287-308, 1999.
15. Pollin R, Luce S: *The Living Wage: Building a Fair Economy*. New York, New Press, 1998.

16. Gruskin S, Tarantola D: Health and human rights, in Detels R, McEwen J, Beaglehole R, Tanaka K (eds.): *The Oxford Textbook of Public Health*. New York, Oxford University Press, 2001, pp. 311-335.
17. McNeil JR: *Something New Under the Sun: An Environmental History of the 20th Century World*. New York, Norton, 2000.
18. Dockery DW, Pope AC, Xu X, Spengler JD, Ware JH, et al: An association between air pollution and mortality in six US cities. *N Engl J Med* 329(24):1753-1759, 1993.
19. Li Y, Roth HD: Daily mortality analysis by using different regression models in Philadelphia count, 1973-1990. *Inhal Toxicol* 7:45-58, 1995.
20. Linn WS, Szlachcic Y, Gong H Jr, Kinney PL, Berhane KT: Air pollution and daily hospital admissions in metropolitan Los Angeles. *Environ Health Perspect* 108(5):427-434, 2000.
21. Pope CA, Dockery DW, Schwartz J: Review of the epidemiological evidence of health effects of particulate air pollution. *Inhal Toxicol* 7:1-18, 1995.
22. Ashe WF: Acute effects of air pollution in Donora, Pennsylvania, in McCabe LC (ed.): *Air Pollution*. New York, McGraw-Hill, 1952, pp. 455-458.
23. American Lung Association: *Diesel Exhaust and Air Pollution*. Retrieved January 3, 2003, from http://www.lungusa.com/air/airout00_diesel.html
24. Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Ferris BGJ: Effects of inhalable particles on respiratory health of children. *Am Rev Respir Dis* 139(3):587-594, 1989.
25. Kinney PL, Aggarwal M, Northridge ME, Janssen NAH, Shepard P: Airborne concentrations of PM_{2.5} and diesel exhaust particles on Harlem sidewalks: A community-based pilot study. *Environ Health Perspect* 108(3):213-218, 2000.
26. Pearson RL, Wachtel H, Ebi KL: Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. *J Air Waste Manag Assoc* 50(2):175-180, 2000.
27. Phalen RF, Oldham MJ, Kleinman MT, Crocker TT: Traceobronchial deposition predictions for infants, children and adolescents. *Ann Occup Hygiene* 32:11-21, 1988.
28. Phalen RF: *The Particulate Air Pollution Controversy: A Case Study and Lessons Learned*. Norwell, MA, Kluwer, 2002.
29. Samet JM, Bishop Y, Speizer FE, Spengler JD, Ferris BG Jr: The relationship between air pollution and emergency room visits in an industrial community. *J Air Pollution Control Assoc* 31(3):236-240, 1981.
30. Wahlin P, Palmgren F, Van Dingenen R, Raes F: Pronounced decrease of ambient particle number emissions from diesel traffic in Denmark after reduction of the sulphur content in diesel fuel. *Atmos Environ* 35(21):3549-3552, 2001.
31. Schimek P: Reducing emissions from transit buses. *Regional Sci Urban Economics* 31(4):433-451, 2001.
32. American Lung Association of California: *Reducing Public Exposure to Diesel Emissions From Heavy Duty Trucks*. Retrieved January 3, 2003, from http://www.californialung.org/advocacy/diesel_position.html
33. Northridge ME, Yankura J, Kinney PL, Santella S, Shepard P, Riojas Y, Aggarwal M, Strickland P, The Earth Crew: Diesel exhaust exposure among adolescents in Harlem: A community-driven study. *Am J Public Health* 89:998-1002, 1999.
34. Lui F: Dynamics and causation of environmental equity, locally unwanted land uses, and neighborhood changes. *Environ Manage* 21:643-656, 1997.
35. National Research Council: *Measuring Lead Exposures in Infants, Children and Other Sensitive Populations*. Washington, DC, National Academy Press, 1993.
36. Needleman HL, Gastonis CA: Low level lead exposure and the IQ of children: A meta-analysis of modern studies. *JAMA* 263:673-678, 1990.
37. Robinson GS, Keith RW, Bornschein RL, Otto DA: Effects of environmental lead exposure on the developing auditory system as indexed by the brainstem auditory evoked potential and pure tone hearing evaluations in young children, in Lindberg SE, Hutchinson TC (eds.): *Heavy Metals in the Environment*. New Orleans, LA, CEP Consultants, Ltd., 1987, pp. 223-225.

38. American Academy of Pediatrics Committee on Environmental Health. Policy statement: Screening for elevated blood lead levels. *Pediatrics* 101(6):1072-1078, 1998.
39. Malcoe LH, Lynch RA, Keger MC, Skaggs VJ: Lead sources, behaviors, and socioeconomic factors in relation to blood lead of Native American and white children: A community-based assessment of a former mining area. *Environ Health Perspect* 110(suppl 2):221-231, 2002.
40. Krieger JW, Higgins DL: Housing and health: Time again for public health action. *Am J Public Health* 92(5):758-768, 2002.
41. Pirkle JL, Brody DJ, Gunter EW, Kramer RA, Paschal DC, Flegal KM, Matte TD: The decline in blood lead levels in the United States. The National Health and Nutrition Examination Surveys (NHANES). *JAMA* 272(4):284-291, 1994.
42. Brody DJ, Pirkle JL, Kramer RA, Flegal KM, Matte TD, Gunter EW, Paschal DC: Blood lead levels in the US population. Phase I of the Third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). *JAMA* 272(4):277-283, 1994.
43. Pearse A, Mitchell MC: Nutrition and childhood lead poisoning. Retrieved January 3, 2003, from <http://ohioline.osu.edu/hyg-fact/5000/5536.html>
44. Brown MJ, Gardner J, Sargent JD, Swartz K, Hu H, Timperi R: The effectiveness of housing policies in reducing children's lead exposure. *Am J Public Health* 91(4):621-624, 2001.
45. Northridge ME, Levick N: Preventing falls at home: Transforming unsafe spaces into healthy places for older people. *Generations* 26(4):42-47, 2003.
46. Olmstead FL: Public parks and the enlargement of towns, in LeGates RT, Stout F (eds.): *The City Reader*. New York, Routledge, 1996, pp. 314-320.
47. Barlow B, Miemirska M, Gandhi R, Leblanc W: Ten years of experience with falls from a height in children. *J Pediatr Surg* 18(4):509-511, 1983.
48. Davidson LL, Durkin MS, O'Connor P, Barlow B, Heagarty MC: The epidemiology of severe injuries to children in northern Manhattan: Methods and incidence rates. *Paediatr Perinat Epidemiol* 6(2):153-165, 1992.
49. Laraque D, Barlow B, Durkin M, Heagarty M: Injury prevention in an urban setting: Challenges and successes. *Bull NY Acad Med* 72(1):16-30, 1995.
50. Krotowski T (ed.): *Special Report—The Injury Free Coalition for Kids: A Passion for Prevention*. Princeton, NJ, Robert Wood Johnson Foundation, 2000.
51. Sugrue TJ: *The Origins of the Urban Crisis: Race and Inequality in Postwar Detroit*. Princeton, NJ, Princeton University Press, 1996.
52. McEwen BS: *The End of Stress as We Know It*. Washington, DC, National Academies Press, 2002.
53. McEwen BS, Seeman T: Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci* 896:30-47, 1999.
54. McEwen BS, Stellar E: Stress and the individual mechanisms leading to disease. *Arch Int Med* 153(18):2093-2101, 1993.
55. Harburg E, Schull WJ, Erfurt JC, Schork MA: A family set method for estimating heredity and stress. I. A pilot survey of blood pressure among Negroes in high and low stress areas, Detroit, 1966-1967. *J Chronic Dis* 23(2):69-81, 1970.
56. Diez-Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ, Sorlie P, Szklo M, Tyroler HA, Watson RL: Neighborhood of residence and incidence of coronary heart disease. *N Engl J Med* 345(2):99-106, 2001.
57. O'Campo P, Xue X, Wang M-C, O'Brien-Caughy M: Neighborhood risk factors for low birth weight in Baltimore: A multi-level analysis. *Am J Public Health* 87(7):1113-1118, 1997.
58. Pickett KE, Pearl M: Multilevel analyses of neighborhood socioeconomic context and health outcomes: A critical review. *J Epidemiol Community Health* 55(2):111-122, 2001.
59. Yen IH, Kaplan GA: Neighborhood social environment and risk of death: Multilevel evidence from the Alameda County Study. *Am J Epidemiol* 149(10):898-907, 1999.
60. Morland K, Steve W, Diez-Roux A, Poole C: Neighborhood characteristics associated with the location of food stores and food service places. *Am J Prev Med* 22:23-29, 2002.

61. [Brownson RC, Baker EA, Housemann RA, Brennan LK, Bacak SJ: Environmental and policy determinants of physical activity in the United States. *Am J Public Health* 91\(12\):1995-2003, 2001.](#)
62. [Fullilove MT: Injury and anomie: Effect of violence on an inner-city community. *Am J Public Health* 88\(6\):924-927, 1998.](#)
63. [Sampson RJ, Raudenbush SW, Earls F: Neighborhoods and violent crime: A multilevel study of collective efficacy. *Science* 277\(5328\):918-924, 1997.](#)
64. [Orfield G: *The Growth of Segregation in American Schools: Changing Patterns of Separation and Poverty Since 1968*. Cambridge, MA, Harvard Project on School Desegregation, 1993.](#)
65. [Orfield G: *Schools More Separate: Consequences of a Decade of Resegregation—New Research Findings From the Civil Rights Project at Harvard University*. Cambridge, MA, Harvard University, 2001.](#)
66. [Wacquant LJD, Wilson WJ: The cost of racial and class exclusion in the inner city. *Ann Am Acad Pol Soc Sci* 501:8-25, 1989.](#)
67. [Lopez R: Segregation and black/white differences in exposure to air toxics in 1990. *Environ Health Perspect* 2:289-295, 2002.](#)
68. [Perlin SA, Wong D, Sexton K: Residential proximity to industrial sources of air pollution: Interrelationships among race, poverty, and age. *J Air Waste Manag Assoc* 51\(3\):406-421, 2001.](#)
69. [Stansfeld S, Haines M, Brown B: Noise and health in the urban environment. *Rev Environ Health* 15\(1-2\):43-82, 2000.](#)
70. [Wambem DB, Piland NF: Effects of improved housing on health in South Dos Palos, California. *Health Serv Rep* 88\(1\):47-58, 1973.](#)
71. [Downs A: Urban realities. *Brookings Rev* 12:26-31, 1994.](#)
72. [Henson DP: Crafting a new community. *Places* 12\(3\):66-67, 1999.](#)
73. [Keating L: Redeveloping public housing: Relearning urban renewal's immutable lessons. *J Am Plann Assoc* 66\(4\):384-397, 2000.](#)
74. [Schuman T, Sclar E: The impact of ideology on American town planning, in Corbin-Sies M, Silver C \(eds.\): *Planning the Twentieth-Century American City*. Baltimore, Johns Hopkins University Press, 1996, pp. 428-448.](#)
75. [Wheeler S: Planning sustainable and livable cities, in LeGates RT, Stout F \(eds.\): *The City Reader*. London: Routledge, 2000, pp. 435-445.](#)
76. [Keeler GJ, Dvonch JT, Yip F, Parker EA, Israel BA, Marsik FJ, Morishita M, Barres JA, Robins TG, Brakefield-Caldwell W, Sam M: Assessment of personal and community-level exposures to particulate matter among children with asthma in Detroit, Michigan, as part of Community Action Against Asthma \(CAAA\). *Environ Health Perspect* 110\(suppl 2\):173-181, 2002.](#)
77. [Sclar E, Northridge ME: Property, politics and public health. *Am J Public Health* 91\(7\):1013-1015, 2001.](#)
78. [Syme SL: *Community Participation, Empowerment and Health: Development of a Wellness Guide for California*. Berkeley, University of California at Berkeley School of Public Health, 1997.](#)
79. [Shepard P, Northridge ME, Prakash S, Stover GN: Advancing environmental justice through community-based participatory research. *Environ Health Perspect* 110\(suppl 2\):139-140, 2002.](#)