Urban Air Pollution and Health Inequities: A Workshop Report

The American Lung Association*

Over the past three decades, an array of legislation with attendant regulations has been implemented to enhance the quality of the environment and thereby improve the public's health. Despite the many beneficial changes that have followed, there remains a disproportionately higher prevalence of harmful environmental exposures, particularly air pollution, for certain populations. These populations most often reside in urban settings, have low socioeconomic status, and include a large proportion of ethnic minorities. The disparities between racial/ethnic minority and/or low-income populations in cities and the general population in terms of environmental exposures and related health risks have prompted the "environmental justice" or "environmental equity" movement, which strives to create cleaner environments for the most polluted communities. Achieving cleaner environments will require interventions based on scientific data specific to the populations at risk; however, research in this area has been relatively limited. To assess the current scientific information on urban air pollution and its health impacts and to help set the agenda for immediate intervention and future research, the American Lung Association organized an invited workshop on Urban Air Pollution and Health Inequities held 22–24 October 1999 in Washington, DC. This report builds on literature reviews and summarizes the discussions of working groups charged with addressing key areas relevant to air pollution and health effects in urban environments. An overview was provided of the state of the science for health impacts of air pollution and technologies available for air quality monitoring and exposure assessment. The working groups then prioritized research needs to address the knowledge gaps and developed recommendations for community interventions and public policy to begin to remedy the exposure and health inequities. *Key words:* air pollution, community intervention, environmental justice, environmental monitoring, genetic susceptibility, health status susceptibility, population surveillance, public policy, urban health. — *Environ Health Perspect* 109(suppl 3):357–374 (2001).
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Observations of geographic differences in the distribution of environmental pollution have long been noted but received little governmental recognition until the 1970s, with mention in the second annual report to the President by the Council on Environmental Quality in 1971 (*1*). During that decade the environmental justice movement was seeded by the joint concerns of civil rights activists and environmentalists. The quality of the environment in which minorities lived was becoming a civil rights issue. In 1982 the environmental justice movement took root when the term "environmental racism" was coined by Dr. Benjamin Chavis Jr., who was then Executive Director of the United Church of Christ Commission for Racial Justice (*2*) and later became Executive Director and CEO of the National Association for the Advancement of Colored People.

The catalyst for the environmental justice movement was a plan by the State of North Carolina to build a toxic waste landfill for polychlorinated biphenyl–contaminated soil in Warren County, a community almost wholly comprising low-income and racial/ethnic minority residents (*1,2*). Civil rights activists and environmental advocates joined forces to stage large demonstrations, and Congress called for the U.S. General Accounting Office to investigate siting practices in Southeastern States (*3,4*). A number of other key studies and reports followed that fueled the environmental justice movement during the last two decades (*2*).

According to the U.S. Environmental Protection Agency (U.S. EPA), environmental justice means, in part, that

> no group of people, including a racial, ethnic, or socioeconomic group, should bear a disproportionate share of the negative environmental consequences resulting from industrial, municipal, and commercial operations or the execution of federal, state, local and tribal programs and policies. (*5*)

Although environmental justice activists have brought inequities of urban air pollution to the forefront of the federal government (*6*), urban communities, where residents are mainly minorities and have low income, continue to experience disproportionately higher air pollution exposure levels and higher risks for diseases than nonurban communities. A recent report of the National Research Council provides a perspective on environmental justice and the related needs for scientific research (*7*).

In response to the environmental and health problems of racial/ethnic minority groups and low-income communities, an invited workshop on Urban Air Pollution and Health Inequities was organized by the American Lung Association to identify disproportionate exposures to outdoor air pollution in urban communities, develop a set of priorities based on community health needs, and recommend a coordinated research agenda to address environmental inequities.

Participants were placed into working groups charged with addressing key issues pertaining to urban air pollution and health inequities. The first part of the workshop focused on assessing the available scientific information and identifying the knowledge gaps in modeling and databases; personal exposure assessment in urban environments; health impacts of urban ambient air pollution; and factors determining susceptibility in urban populations. The agenda for the second part of the workshop included setting priorities and recommendations for monitoring ambient air pollution concentrations and exposures; understanding health impacts and susceptibility; and reducing inequities through community-based intervention and public policy. This article summarizes the background scientific information and presents the recommendations of the working groups.

Background Scientific Information

Health Effects of Ambient Air Pollution

General overview of health effects research. The evidence on the health effects of air pollution has been summarized in a state-of-the-art review (*8,9*) and a recent monograph (*10*), as well as in two recent U.S. EPA criteria

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*See Appendix for list of participants.

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documents (*11,12*). The identified adverse health effects and biologic markers of response, listed in Table 1 and taken from the American Thoracic Society's review (*8*), are diverse in scope, severity, duration, and clinical significance. This diversity reflects the multiple pathways of injury by air pollution and the sweeping nature of the research evidence, which comes from epidemiologic studies, human clinical exposures, animal toxicologic studies and *in vitro* experiments. Moreover, clinical, biologic, and societal perspectives of what constitutes an adverse health effect of air pollution have been evolving (*13–15*). With biologic markers providing early indications of injury, sensitive research approaches offer evidence of subtle population effects, and quality-of-life has been added as an outcome of interest. A growing concern about a wider range of pulmonary and extrapulmonary effects may expand this list to include neoplasms, airway sensitization, neurologic abnormalities, and developmental toxicities, as well as other possible health effects (*15*). The collective database still contains significant gaps, however, on the health effects of single and multiple pollutant exposures (*16*), including exposures to diesel exhaust and other complex mixtures. The specific mechanisms responsible for the morbidity and mortality associated with increases in levels of particulate matter [particulate matter with an aerodynamic diameter less than 10 µm (PM_{10})] are currently ill-defined and poorly understood (*17,18*).

Research specific to urban populations. The health effects experienced by urban populations exposed to air pollutants are expected to be similar to those listed in Table 1. The severity of the effects, however, may be greater in urban and racial/ethnic minority or special population groups in comparison with the overall population or the white majority. Although evidence is lacking, several factors point to this possibility (*19,20*). Exposures to some outdoor pollutants may be generally higher in the urban community than in the general population. Routine ambient monitoring may not accurately capture local or microenvironmental, high-level exposures in urban settings (e.g., near diesel bus depots). Such high-level exposures would, of course, be expected to produce proportionately larger risks to health. Populations in urban locations may disproportionately include subpopulations more susceptible to ambient exposures. Factors contributing to the heightened susceptibility may include preexisting disease, insufficient access to optimal medical care, poor nutrition, socioeconomic stresses, and coexposure to other pollutants such as bioaerosols.

Table 1. Health effects and biologic markers of response associated with air pollution.[a,b]

- Excess cardiorespiratory mortality
  - Deaths from heart or lung disease in excess of number expected
- Increased healthcare utilization
  - Increased hospitalizations, physician visits, emergency department visits
- Asthma exacerbations
  - Increased physician visits, medication use
  - Decreased peak flow measurements
- Increased respiratory illness
  - Increased respiratory infections, physician visits, episodic symptoms
- Increased respiratory symptoms
- Decreased lung function
  - Spirometry, peak flow rates, airways resistance
- Increased airways reactivity
  - Altered response to challenge with methacholine, carbachol, histamine, cold air
- Lung inflammation
  - Influx of inflammatory cells, mediators, proteins
- Altered host defense
  - Altered mucociliary clearance, macrophage function, immune response

[a] Clinical or public health significance of some effects are unknown. [b] Data from the American Thoracic Society (*8*).

Numerous epidemiologic studies have addressed health effects across large-scale urban areas and have provided a sizable baseline database of health status and health effects. These studies have included both open cohort (time–series) (*21–26*) and closed cohort investigations (*27–33*) of the consequences of exposure to ambient PM_{10}, ozone, nitrogen dioxide, and other criteria pollutants, which must meet the National Ambient Air Quality Standards (NAAQS) for outdoor concentrations. Several studies have shown that background levels of fine particulate matter [particulate matter with an aerodynamic diameter less than 2.5 µm ($PM_{2.5}$)] and ozone vary little across large U.S. urban areas of 20–50 km in diameter (*34,35*). Nevertheless, significant spatial and temporal gradients in the sources, composition, and concentrations of ambient gases and particles may result in varying health effects of air pollution within the same city. Large-scale studies, however, have not focused on intraurban or neighborhood differences in the distribution of health effects associated with urban air pollutants.

Since 1993 epidemiologic studies have reported health effects across small-scale, intraurban areas, particularly in Europe and Japan. Many of these studies have used traffic congestion or density as a surrogate for exposure to mobile source pollutants, including nitrogen dioxide, carbon monoxide, ultrafine particles, and air toxics (*36–45*). By combining comprehensive traffic flow information with health data from ongoing geographic information systems, insights can be gained into the effects of local traffic (exhaust) on respiratory symptoms, lung function, and asthma. Many studies have shown that heavy traffic flow (particularly truck traffic) is associated with increased risks of childhood respiratory symptoms, decreased lung function, and increased doctor visits or hospital admissions for asthmatic children, for example (*36–45*).

Industrial sources may release volatile organic compounds (VOCs) and other toxic chemicals into urban areas. The health effects of exposure to VOCs or hazardous air pollutants have been evaluated in a few studies (*46–49*). In the Kanawha County Health Study (*47*), concentrations of petroleum-related VOCs measured at local elementary schools in West Virginia were associated with the prevalence of persistent lower respiratory symptoms, including asthma. In Taiwan, residents in a petrochemical-polluted area complained about more acute irritative symptoms (i.e., eye irritation, nausea, throat irritation, chemical odors) than residents in a less-polluted comparison area (*49*).

Research on subpopulations. A few studies have explicitly targeted children and urban racial/ethnic minority populations or focused on interracial differences in susceptibility to air pollution. Recent work suggests that photochemical pollution and other forms of air pollution have long-term effects on children's respiratory health (*50,51*). Lung function is lower in children who breathe more polluted air. These effects may leave children more vulnerable to respiratory disease and may result in a lower level of lung function as they enter adulthood. Moreover, higher air pollution levels have also been directly linked with increased prevalence of asthma in children (*52*).

In the United States most morbidity and most deaths from asthma occur in urban areas. For example, when compared to national asthma mortality rates in the 1990s, Chicago, Illinois, had higher rates for all age groups (*53*). The greatest disparity, however, was among non-Hispanic blacks. In the United States the asthma mortality rate among blacks was 2.5 times higher than for whites; in Chicago it was 4.7 times higher. In 1992 the rate of asthma hospitalizations in Boston, Massachusetts, was twice as high as the Massachusetts rate after adjusting for age and gender (*54*). Further small-area ecologic analysis showed positive correlations between asthma hospitalizations and higher poverty rate and proportion of nonwhite residents. Small-area analysis of California by ZIP code resulted in similar findings on asthma hospitalizations (*55*). In New York City the highest asthma mortality rate was in the neighborhood of East Harlem and was four times the citywide rate in the 1980s (*56*). African Americans and Hispanics in Philadelphia, Pennsylvania, have higher rates of death from asthma, but only in areas with higher poverty rates (*57*). Another small-area study in Chicago also points to poverty rather than race as being a much stronger risk factor for asthma mortality (*58*). When data for blacks and whites were analyzed together, asthma mortality was significantly associated with lower socioeconomic status; this association was not significant when blacks were analyzed separately.

Clinical studies (i.e., experimental exposures of volunteers to air pollution) do not show differential responses to air pollution by race. Seal and colleagues (*59*) exposed healthy African–American and Caucasian adults to different ozone exposure concentrations and found no significant racial group differences in respiratory symptoms and pulmonary function, although there was a trend toward greater effects on lung function among African–American males. Similarly, a small clinical study (*60*) found no significant group differences in lung function or nasal lavage cells between African–American and Caucasian males with asthma after exposure to sulfur dioxide.

Findings of observational studies are similar. In a time–series substudy of the National Cooperative Inner-City Asthma Study (NCICAS), Mortimer et al. (*61*) found that ambient ozone concentrations were associated with similar lung function decrements in children with asthma who were African American and Puerto Rican living in New York City. Greater responses to ozone, however, were seen in Chicago in Mexican children with asthma compared to African Americans. In a panel study of African–American children with asthma in Los Angeles, California, Ostro et al. (*62*) reported that both ozone and PM_{10} were associated with respiratory symptoms. On the basis of emergency clinic visit data, White et al. (*21*) found that inner-city African–American children with asthma may have their disease exacerbated by high ambient ozone pollution.

Individuals with atopy constitute another subpopulation that may be highly susceptible to the effects of air pollutants (*63–69*). Allergen sensitization is highly specific and reflects prior allergen exposure. Subsequent short exposures to allergens may cause prolonged increases in airway hyperresponsiveness, which indirectly affects susceptibility to other airborne pollutants such as nitrogen oxides and ozone. For example, allergen and air pollutant exposures are both independently associated with exacerbation of asthma, but they can also interact to intensify the severity of response (*70*). Rates of sensitization to allergens have been studied in both urban African–American and Hispanic populations and do not differ from rates seen in other population groups. In the NCICAS, Kattan et al. (*71*) found sensitization rates around 78% among urban, school-aged asthmatic populations without differences among African Americans, Hispanics, and other ethnic groups. Overall, this rate did not differ from the 88% rate of sensitization in the more broadly based asthmatic population in the Childhood Asthma Management Program (*72*). At the same time, specific sensitization patterns may vary considerably among different populations. For example, urban populations may be less frequently sensitized to pollens but more frequently sensitized to cockroach and other indoor allergens.

Factors Determining Susceptibility in Urban Populations

In a broad sense the term “susceptibility” refers to a greater likelihood of an adverse outcome given a specific exposure, in comparison with the population generally. For example, when a given population is exposed to air pollution at a given concentration and for a given period of time, some proportion of the population may experience adverse health outcomes, whereas the rest of the population does not. Identification of the determinants and contribution of susceptibility to the health effects of air pollution in urban communities is a key element of the environmental justice agenda.

Susceptibility is the result of both host and environmental factors. Genetic susceptibility may be the most commonly studied host factor, but other host factors to be considered include diet, physiologic state, and psychologic status. Some determinants of susceptibility (e.g., age) represent normal developmental phases for all human beings, or they may represent normal biologic attributes (e.g., gender) whose meaning is shaped, to a large degree, by the social context. Although consideration of individual and group differences in genetic susceptibility to the effects of air pollutants is relevant, the nonrandom distribution of population-level determinants of susceptibility appears to play a dominant role in influencing the observed pattern of health outcomes. For example, poverty areas (defined by social, economic, and geographic attributes) manifest excess rates of morbidity and mortality due to certain chronic conditions, including asthma, diabetes, and hypertension. We need to better understand these population-level factors, including the loss of social networks (*73*), as determinants of susceptibility in affected communities.

Population data are thus important for identifying specific subpopulations that may exhibit factors associated with increased susceptibility, and for defining the geographic or personal characteristics that determine risk for disease outcomes in exposed populations. Such information may be useful in contributing to rational planning of public health policies and specific intervention programs to prevent diseases attributable to urban air pollution. Before deciding if a particular subpopulation (e.g., the very young) shows increased susceptibility to the health effects of an environmental exposure when compared to the general population (as assessed by a specific indicator of outcome), researchers need to consider the following questions:

- Has the indicator been causally linked to the exposure of interest?
- Is the risk for the health indicator associated with the exposure different between the subpopulation and a comparison population?

For example, children with asthma are considered as likely to be at greater risk for respiratory morbidity (e.g., respiratory symptoms) than children not having asthma. To characterize children with asthma as susceptible to air pollution, researchers should show that the risk in this group is greater than in nonasthmatic children. The needed evidence might compare the exposure–response relationships for the two groups with the anticipation that susceptibility would be manifest as a steeper curve or perhaps a different form than found for nonasthmatic children.

Age-related windows of vulnerability. For many disease conditions the occurrence of disease is a reflection of the interaction between host and environmental factors, with both sets of factors possibly varying over the life span. Fetuses and infants are considered to be more susceptible to a variety of environmental toxicants than adults because of exposure patterns, physiologic immaturity, and the longer life span over which disease initiated in early life can develop (*74*). For example, children have higher breathing rates and therefore higher intake of air pollutants per unit of body weight (*75*). Developing organs may be more susceptible to toxicants due to higher rates of cell proliferation or changing metabolic capabilities (*76*). The first symptoms of asthma are generally manifest before 6 years of age in most individuals developing the disease, leading to the hypothesis that much of asthma originates in childhood or infancy and may even reflect *in utero* exposures. Studies of maternal exposure to PM during pregnancy have shown a negative impact on fetal development (*74,77*), but it is unclear whether prenatal exposures may translate into risk for the development of diseases such as asthma. Observational and biomarker studies in children and models of infant airway injury and repair would be helpful for testing hypotheses on the inception, pathogenesis, and outcomes of lung diseases occurring in early life, including asthma and bronchopulmonary dysplasia. Low birth weight is a risk factor for asthma (*78*). Low birth weight, particularly common among mothers in poor urban neighborhoods (*79*), is strongly associated with the mother’s psychosocial status (*80*).

Health status as a determinant of susceptibility. Epidemiologic studies suggest that individuals with chronic cardiopulmonary conditions, including asthma, chronic obstructive pulmonary disease (COPD), and cardiovascular diseases, are at increased risk for developing adverse health outcomes from exposure to urban air pollution.

Asthma. Even when mild in severity, asthma is associated with increased airways responsiveness and a tendency for increased ventilation and more central deposition of inhaled pollutants. Although it is known that air pollutants can cause asthma exacerbations, much remains to be learned about how environmental factors have contributed to the rise of asthma-related morbidity. Of particular interest is the interaction between exposure patterns over time and increased asthma prevalence (i.e., susceptibility) in some urban populations of color. Little is known about the relative contribution of local sources of air toxics to asthma morbidity or how such local sources interact with regional air pollution as determinants of asthma morbidity. Research on affected persons during the asthma epidemics in Barcelona, Spain, suggests that certain subgroups, namely atopic individuals, are at increased risk for manifesting the asthmatic syndrome following exposure to soybean dust (*81*). Such susceptible subgroups need to be characterized further in relation to specific exposures to inform preventive strategies.

Chronic obstructive pulmonary disease. The health status of persons with COPD is affected by ambient air pollutants, including ozone and PM_{10}. Effects range from unscheduled hospital visits to mortality, but little is known about the mechanisms by which these ambient air pollutants increase morbidity and mortality in people with COPD (*10*).

Cardiovascular diseases. Exposure to sufficiently high levels of carbon monoxide causes asphyxia and may increase cardiovascular morbidity. Recent epidemiologic studies indicate increased cardiovascular morbidity and mortality in association with ambient PM. Mechanisms that may increase cardiovascular mortality with exposure to particulate air pollution, particularly PM_{10} and $PM_{2.5}$, remain uncertain. Moreover, individuals with a variety of conditions, including central abdominal obesity, smoking, hypercholesterolemia, and hypertension, are at increased risk for adverse cardiovascular events (*9*). Potential mechanisms underlying interactions between exposure to air pollutants and these risk factors, as determinants for cardiovascular morbidity and mortality, have not yet been elucidated.

The role of genetic factors. Genetic factors are likely important in the development, persistence, and exacerbation of chronic respiratory diseases, although research in this area is only beginning (*82,83*). For example, the risk for developing asthma increases with having a first-degree relative with asthma, indicating a role for heritability, but the etiology is undoubtedly complex and the mode of transmission remains undefined. Specific genes for asthma have yet to be identified definitively. The sharp rise in asthma prevalence in just the past two decades implies an environmental cause, as its time course is not that of a genetic change in the population. Polymorphic genes for a vast array of physiologic functions are suspect as determinants of susceptibility to COPD and asthma (*84–88*). In addition, some hazardous air pollutants (HAPs) are allergenic and have caused airway hyperreactivity and asthma in occupational settings (*89–91*). It is unclear, however, whether the low-level exposures to environmental (ambient) air pollution can effect similar responses (*16*). Additional clinical and epidemiologic studies and informative animal models for studying the interactions of genes and ambient air pollution are needed.

Gender-related differential. Statistics on healthcare utilization suggest that women may be more vulnerable to the chronic effects of air pollutants. Scant comparative data are available, however, on women's time activity patterns, smoking habits, behavioral attributes, occupational exposures, or hormonal levels that may contribute to gender-related differential susceptibility.

Socioeconomic factors. There is ample evidence that socioeconomic status (SES) is a strong determinant of susceptibility to the adverse effects of air pollution. Not only are conditions such as asthma more prevalent in low-income communities, but there is evidence that increased prevalence of asthma is quantifiably related to the extent of social deprivation in low-income communities (*92*) and the experience of stress (*93*). Thus, an important direction of public health research is to identify factors common in the context of poverty that may increase exposure to environmental toxicants, increase susceptibility to their health effects, or both.

Social exposures. The morbidity and mortality experience of a population is more likely to be adverse in the context of lower SES and an unfavorable political climate (*73,94*). The community history provides an approach to characterizing existing resources within a community. Facets of such community history include information flow, existing networks, strengths of networks, extent of resource sharing, and the degree of the community's political empowerment or lack thereof, as well as cultural factors. Little data are available, however, on the mechanisms by which the social experience of a community may contribute to increased susceptibility to ambient air pollutants.

Stress. Strong evidence that psychosocial stress increases susceptibility to adverse effects of air pollution comes from the NCICAS. Children's mental health status was significantly associated with asthma morbidity, specifically wheezing and functional status (*95*). Several mechanisms have been proposed to explain the physiologic response to chronic stressors. Such mechanisms include cytokine immunomodulation and disruption of the hypothalamus–pituitary axis (*96*). Immunomodulation has been implicated in the mechanisms for several conditions associated with exposure to chronic stressors, including low macrophage concentrations (*97–99*). Similarly, immunomodulatory effects of chronic stressors are implicated in the propensity to asthma exacerbations. Although the mechanisms for such effects of stress remain to be elucidated, the new body of work on leptin and the adrenal hormones may prove valuable in showing mechanisms via stress (*100,101*). There are no data on potential interactions between exposure to ambient air pollution and the response to stressors in individuals with chronic respiratory diseases. Coping behaviors and locus of control may modify the impact of stressors in the chronically exposed individual. We do not understand how these behavioral characteristics may modify the consequences of exposure to stressors and how mechanisms underlying such effect modification.

Quantifying Urban Air Quality: Available Technologies

Monitoring and air quality modeling are critical tools for characterizing population exposure variables and tracking the consequences of air quality management programs. Monitoring is routinely carried out in urban areas, but geographic detail may be insufficient to establish differential exposures across communities. Air quality models that incorporate emissions and meteorologic data may be useful for this purpose.

Air quality monitoring and data collection. Data applicable to tracking air quality are collected routinely and fall into three classes: emissions inventories, ambient air quality monitoring, and meteorologic data. Each of these three types of databases provides information on a different dimension of pollutant dynamics that directly affects quantitative air quality assessments. Accuracy of the data depends on the methods of collection and the number of samples or frequency of measurements.

Emissions inventories. Following the approach of the U.S. EPA, emissions inventory databases can be divided into two pollutant categories: criteria pollutants and HAPs. Within each of these categories, the emissions are further subdivided as coming from point sources and area sources. Point sources are those large single facilities that are required to report emissions. In contrast, area sources are the myriad smaller sources such as motor vehicle emissions, and these inventories are generally estimated using emissions models. Similarly, emissions models have been used substantially for criteria pollutants, while emissions models are rapidly being developed and modified for HAPs.

Ambient air quality monitoring. As with the emissions inventory databases, ambient air quality databases can be divided into those for criteria pollutants and HAPs. The ambient criteria pollutant databases are generally more robust than the HAPs databases because of the larger number of monitoring stations and greater frequency of sampling. For example, in California alone there are over 250 ambient air–monitoring stations for criteria pollutants. The ambient HAPs monitoring network, on the other hand, consists of 22 ambient air–monitoring stations located statewide that monitor 58 substances with a sampling frequency of 1 day in 12. Excluding diesel exhaust, the more commonly monitored substances are benzene, 1,3-butadiene, hexavalent chromium, carbon tetrachloride, and formaldehyde.

Meteorologic data. Meteorologic data are among the simplest to obtain, as the instrumentation needed to collect the data is readily available with established protocols. The simplicity of data collection, however, is offset by the fact that complex physical terrain limits the representativeness of meteorologic data to localized regions. In addition, studies that require long-term estimates need at least 5 years of consecutive data; studies involving short-term, episodic events require more intensive data-gathering efforts.

Using air quality information in modeling. Air quality models can be used to estimate atmospheric concentrations of air pollution by simulating the physical atmospheric relationship between emission sources and receptors, the points for which pollutant concentrations are to be estimated. The construction of air quality models ranges from representing simple atmospheric processes to more complex situations.

Models can estimate downwind concentrations as a function of source and environmental parameters, as well as evaluate the effects of control measures. Modeling over a large geographic scale, such as nationally, may also provide information on a local scale, which can be used for prioritizing and targeting important pollutants and areas that are in need of further research. Models are also useful for estimating concentrations in circumstances where monitoring data are unavailable, such as when there is insufficient spatial or temporal ambient coverage. For a proposed new source, air quality models are useful in estimating expected concentrations.

There are four primary data inputs to an air quality model, as depicted in Figure 1. These inputs are source parameters, meteorologic conditions, physical terrain data, and ambient measurements.

Source parameters. Emissions are the primary data for the source parameters, and they should be characterized according to their spatial and temporal distribution. Physical variables constitute the secondary data for the source parameters. The physical variables include stack conditions (e.g., stack gas temperature, exit velocity, stack height, diameter) and building dimensions, which should be incorporated in a downwash analysis. Other types of source information that can be input for an air quality model are decay rates, chemical reaction rates, and deposition rates of various compounds.

Meteorologic conditions. Meteorologic data are needed for the models to simulate atmospheric transport, dispersion, and chemical transformations. Typical meteorologic data from aloft measurements include wind speed and direction and temperature at various heights above ground level. Additional surface data may include horizontal wind direction fluctuations, temperature lapse rate, and incoming solar radiation.

Physical terrain data. Elevation and type of land use constitute physical terrain data. The land use is important for placing receptors as well as estimating dispersion due to surface roughness and heat island effects. The more sophisticated models can use elevation to better describe the transport and dispersion of pollutants at the surface.

Ambient measurements. Ambient data are necessary for evaluating model performance. Although model performance is not part of every analysis, evaluation is necessary for establishing the credibility of the models. Once an air quality model has performed successfully under controlled conditions, it can then be used to evaluate emissions for different uncontrolled conditions. Photochemical grid-based models, which are generally used to evaluate widespread control strategies, almost always require performance evaluations using ambient data. On the other hand, a Gaussian-based model can be used for a single source under certain environmental conditions without a performance evaluation, such as when the emissions are from a proposed facility.

Types of air quality models. There are several types of air quality models available; the optimal model provides the most accurate representation of atmospheric transport, dispersion, and chemical transformations in the area of interest. The most commonly used models can be grouped into three categories, which are complementary: receptor-based models, regional/urban scale models, and microscale models.

Receptor-based models. Receptor-based models provide a predicted estimate of air pollutant concentrations at different geographic points, or receptors. They are observation based and dependent on measurements of specific ambient pollutants and detailed emissions inventories. In other words, they require profiles of the emission sources that are time- and pollutant-specific in order to describe concentrations at particular locations. Because the results of receptor-based models are dependent on ambient measurements, they are generally considered to be a good foundation for risk assessments. Limitations to receptor-based models are that *a*) concentrations can only be estimated at receptor locations, and *b*) locations of higher concentrations may be unobserved. The chemical mass balance model is a typical receptor-based model.

Regional/urban scale models. Regional or urban scale models provide projections of concentrations across geographic domains on the scale of large urban areas or regions. They typically use a gridded approach to simulate the three-dimensional transport, chemistry and dispersion of emissions. These grid-based models generally are used to evaluate control measures, and they require a gridded hourly emission inventory and meteorologic data as inputs, as well as ambient data to evaluate their performance. Grid-based models have a high computational demand; a 1-day simulation could use 1 day of computation time. Typical models in this category are the Urban Airshed Model, Models-3, and CALPUFF.

Microscale models. Microscale models generally are best suited for hot spot (i.e., near major stationary sources) analyses to estimate concentrations for receptors on a fine-scale resolution at distances from 50 m to a few kilometers from the source. These models are routinely used beyond these limits, however, at distances as close as 20 m and as far as 50 km. Microscale models are commonly used to evaluate the emissions of current or proposed facilities. Routine meteorologic data are input to establish the transport and diffusion of the emissions, but chemistry is usually not considered. Models in this category are the Industrial Source Complex 3 models, the American Meteorological Society/U.S. EPA

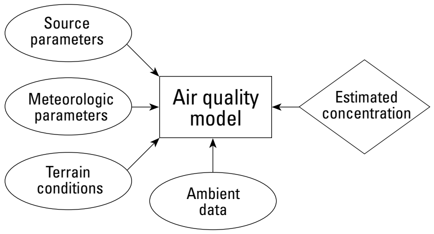

Figure 1. Data inputs to air quality model.

Regulatory Model (AERMOD) and CALPUFF. CALPUFF transcends both microscale and urban scale types of models.

Cumulative exposure modeling of hazardous air pollutants. Currently there are two models for cumulative exposure from multiple HAP emission sources: the Assessment System for Population Exposure Nationwide (ASPEN) model and the Multiple Air Toxics Exposure Study II (MATES II) model. Development of the ASPEN model was sponsored by the U.S. EPA as part of the Cumulative Exposure Project (CEP), which estimates long-term outdoor concentrations of 148 of the 188 HAPs listed in the Clean Air Act of 1970 (*102*) for every census tract in the contiguous United States in 1990 (totaling 60,803 census tracts) (*103,104*). The CEP uses ASPEN, a Gaussian dispersion model (*103*), to estimate outdoor concentrations of HAPs on the basis of emission rates of the HAPs, frequencies of various meteorologic conditions, and effects of atmospheric processes such as decay, secondary formation and deposition. Formulated around the Industrial Source Complex Long Term air dispersion model, ASPEN includes relatively simplified treatment of atmospheric chemistry, transport, and diffusion. Advantages of using these approaches include the ability to perform multiple runs of the model to evaluate sensitivity parameters with relatively short computing times, and analysis of a large domain—up to the entire nation.

By contrast, the MATES II approach uses a grid-based model with some limited chemistry and a microscale model for evaluation at a finer resolution. Although conceptually this is an ideal approach, the computational requirements are high for large domains, and this may limit sensitivity analysis on large scales. Developed by the South Coast Air Quality Management District in California as part of their study to assess toxic exposures in southern California, the MATES II approach uses the Urban Airshed Model with the Industrial Source Complex Short Term 3 (ISCST3) model for microscale analysis.

The California Air Resources Board is currently developing an exposure assessment model designed to evaluate health risk. This model, the Hot Spots Analysis and Reporting Program, comprises three modules: *a*) an emissions module, the California Emission Inventory Development and Reporting System; *b*) an air dispersion module, including ISCST3 and AERMOD; and *c*) a risk assessment module developed by the California Office of Environmental Health Hazard Assessment (Stochastic Approach). It may be possible to use the Hot Spots Analysis and Reporting Program to evaluate cumulative exposure on a neighborhood scale basis, providing an informative tool for analyses directed at environmental justice issues.

Although the models described can provide some information on relatively small geographic scales (e.g., neighborhoods), they are most appropriate for larger-scale areas. There is a need for a modeling approach that can couple the existing models to the neighborhood scale. There will, however, be substantial technical challenges to developing such approaches.

Exposure Assessment in Urban Communities

The environmental justice movement has raised concerns about the potential for disproportionate exposures to air pollution among disadvantaged or racial/ethnic minority populations in urban areas due to the proximity of polluting sources such as bus depots, trucking facilities, high-volume roadways, waste treatment and transfer stations, and industrial point sources. Although some data are available on personal air pollution exposures of urban residents, little is known about variations in personal exposures across communities in relation to local sources.

Impacts of local air pollution sources. Factors that can influence personal air pollution exposures of urban residents, as shown in Figure 2, include regional-scale polluted air masses, proximity to local ambient sources, indoor penetration of outdoor pollution, indoor pollution sources, time–activity patterns, and individual characteristics and behavior. Sorting out the relative importance of these diverse factors in driving differential urban air pollution exposures is a complex and daunting task, and one for which only limited data are yet available. Answers will likely differ for different urban air pollutants and locations.

Assessing exposure to outdoor sources of air pollution requires information on concentrations to which people are exposed as well as the frequency and duration of that exposure. Factors such as local meteorology, pollutant volatility, and the time residents spend proximate to sources (e.g., on the street) are significant determinants of personal exposures. Furthermore, the degree of air exchange from outdoors to indoors has a significant impact on the contribution of outdoor sources to exposures. For instance, residents who live in dwellings without air conditioning may be exposed to outdoor air most of the day during the summer months because windows remain open to increase ventilation or because more time is spent outdoors.

Local levels of air pollution are also influenced by the contribution of regional sources, especially for fine particles and ozone. For some people there are also significant contributions to total air pollution exposure from indoor sources such as kerosene heaters and smoking. It should be noted that these indoor pollution sources may contribute to the total exposure and health burden experienced by urban residents.

Given the model depicted in Figure 2, systematic differences in exposures across population subgroups could arise from disparities in either outdoor pollution concentrations or from time–activity patterns. Thus, to measure these systematic differences in exposure between urban and nonurban communities, information is needed both on where and how people spend their time as well as the pollution concentrations in each location.

Current knowledge of personal exposure assessment in urban environments. Although some data are available on personal exposures of urban residents to air pollutants, little is known about variations in personal exposures between or within communities in relation to local outdoor sources. The recently completed MATES I and II studies in Southern California examined spatial variations in outdoor air toxic concentrations related to sources and found local traffic to be an important determinant of local concentrations (*105*). The U.S. EPA Total Exposure Assessment Methodology studies of carbon monoxide and traffic in Denver, Colorado, and Washington, DC, addressed personal exposures, time–activity patterns, and housing characteristics, though not in low SES populations (*106*).

The Kanawha County Health Study reported associations between concentrations of VOCs and other pollutants and residential proximity to industrial sources in a working-class rural mining community in West Virginia (*47*). Studies have been completed and are in progress that address housing, traffic, VOC concentrations, and other factors in African–American neighborhoods

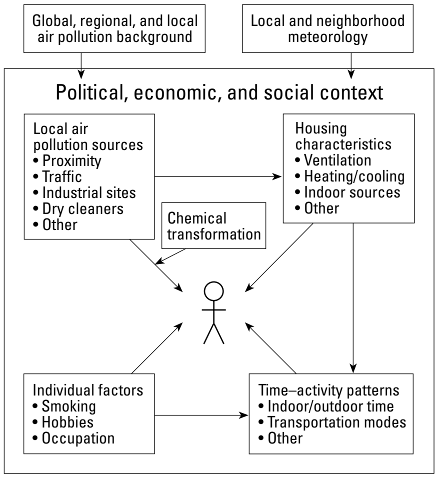

Figure 2. Factors influencing personal exposures to air pollution in urban environments.

of Baltimore, Maryland (*14,107–109*). The ongoing TEACH study (Toxic Exposures of High School Students: A Columbia/Harvard Study) is addressing $PM_{2.5}$ and air toxic exposures among racial/ethnic minority high school students in New York City and Los Angeles (*110*). The RIOPA (Relationships of Indoor, Outdoor and Personal Air) study is currently investigating indoor, outdoor and personal air concentrations of VOCs, aldehydes, and $PM_{2.5}$ in 100 residences in Los Angeles; Elizabeth, New Jersey; and Houston, Texas (*111*). Lioy et al. have reported data on toxic metal exposures in New Jersey urban areas (*112*). Although the data from these studies are not representative for the general population, they are providing needed information on personal exposures to air pollutants, particularly some of the HAPs. Except for the RIOPA study, these studies do not include individual-level data about SES, thus making it difficult to draw conclusions regarding differential exposures by SES.

Time, budget, and travel behavior studies consistently show that role-related characteristics, particularly employment status and gender, have strong influences on travel/activity patterns of individuals (*113*). Work status appears to explain most of the differences in traditional measures of activity patterns. Thus, factors contributing to exposure will likely differ for those who do and do not work outside the home. Nonworkers spend significantly more time at home (i.e., between 4 and 6 hr, on average), but only half as much time in travel as workers. Men spend about twice as much time traveling and being outdoors as women. Among adults, activity patterns do not differ based on age (over or above 60 or 65 years) once work status has been controlled (*113*). Clearly, children's outdoor exposure is determined by school schedules during much of the year.

To date, little of the time–activity data from air pollution exposure studies have been analyzed for differences by SES. Research shows, however, differences based on education and/or income in the uses of discretionary time and the amount of travel. Specifically, higher SES groups tend to spend more time in active and away-from-home activities than lower SES groups. The implications of this difference for current patterns of exposure to air pollutants are not clear. Lower SES groups also spend less time traveling and travel shorter distances. Much of the difference in travel behavior among income groups has been attributed to differential rates of automobile ownership.

Several researchers have investigated the potential for differences in in-home air pollution concentrations and personal exposures across groups defined by socioeconomic and demographic characteristics. Two different studies found that, despite differences in time at home and in travel, there are few statistically significant differences in levels of personal exposure to carbon monoxide (Washington, DC, area) and nitrogen dioxide (Los Angeles Basin) between workers and nonworkers (*113,114*). On the other hand, these analyses did show differences among gender and work-status groups in the contribution of different sources to the total exposure. In addition, another study found that being male, Hispanic, employed, and/or middle-aged were positively associated with personal exposures to various VOCs, even after proximity to sources was considered (*115*).

Geographic analyses suggest systematic differences in exposure by community. For instance, indoor levels of carbon monoxide in the Washington, DC, area and nitrogen dioxide in the Los Angeles Basin are higher in central-city areas than in the suburbs (*116,117*). Models of exposure based on both activity patterns (i.e., indoor vs outdoor time, exertion level, and mobility) and ambient monitoring show that low-income groups, racial minorities, and children are exposed to the highest levels of ozone and particulates in the South Coast Air Quality Management District of California (*118*). Kinney and colleagues recently reported results of a study of small-scale spatial variations in $PM_{2.5}$ and elemental carbon in the Harlem neighborhood of New York City (*119*). Another recent study reported a correlation between residential indoor polycyclic aromatic hydrocarbon concentrations and local traffic density (*120*). These limited data suggest that ambient concentration differentials can exist in urban areas due to traffic sources.

Using modeling to evaluate exposures in urban areas. Air-monitoring data can contribute to our understanding of community exposures. Because of financial, technologic, or logistical constraints, however, air-monitoring data may be unavailable or of limited scope geographically or temporally. Air quality modeling can help to fill in the gaps over both space and time. To have valid models, good data are needed on source locations and emission rates in the community, as well as meteorologic conditions such as wind direction and speed. Wind flow through urban street canyons is complex and difficult to model. Air pollution concentration gradients should be modeled in three dimensions, as vertical gradients may be important for personal exposures. Depending on the level of detail of the model and emissions inventory, estimated ambient concentrations of air pollutants from modeling can provide information on a large scale to screen for potential adverse impacts on health, or more precise estimates can be used to assess geographic variations in risk.

For example, the CEP estimates long-term outdoor concentrations of 148 of the 188 HAPs listed in the Clean Air Act for every census tract (60,803) in the contiguous United States for 1990. Concentration estimates were based on emissions rates of the HAPs, frequencies of various meteorologic conditions, and effects of atmospheric processes such as decay, secondary formation, and deposition. This assessment provides information for every community in the United States and can be used to assess priorities on important areas and pollutants in a community, but need to be supplemented with local information prior to making policy or community decisions.

An example of how large-scale modeling can be combined with local information is the Chicago Cumulative Risk Initiative for Cook County, Illinois, and Lake County, Indiana, an evaluation of potential environmental impacts of air pollution in the Chicago area. This is a joint project between U.S. EPA Region 5, local community activists, and researchers and was initiated by concerns from the community over high concentrations of HAPs in their neighborhoods (*121*). The project draws upon a variety of data sources, including the Toxics Release Inventory, local emission inventories, and ambient concentrations, which are used to describe local conditions in terms of air quality, health outcomes, and demographic makeup of the communities in the Chicago area.

Summary

The knowledge gaps in collective data on health effects of air pollution require additional research, particularly on the specific mechanisms for mortality and morbidity from increased PM exposure. The adverse health effects of air pollution among urban communities are even less well understood. Existing research specific to urban populations has clearly shown a disproportionate burden of exposure to air pollutants in low-income and racial/ethnic minority communities. These communities are also subject to higher risks of health problems associated with air pollution, at levels beyond those expected with the higher levels of exposure. Numerous host and environmental susceptibility factors in urban populations play roles in the observed increased burden of disease. Although the higher air pollutant exposure levels in urban areas can be mitigated through changes in policies and regulations, more data are needed on the health effects of air pollution in urban communities and the factors within these populations that lead to the observed inequities in health risks. In addition, exposure assessment, air quality monitoring, and air quality modeling are areas that should be addressed in future research and current actions aimed at resolving urban health inequities. Biomarkers are other potentially useful tools that could provide insights. Recommendations follow for air quality data collection and exposure assessment, health effects research, community-based intervention, and public policy.

Recommendations

Air Quality Data Collection and Exposure Assessment

Improved monitoring and modeling of urban air quality. In assessing the impact of ambient air pollution in urban areas, it is critical to characterize the pollutants, both in terms of species and concentrations, as well as exposures to both susceptible and nonsusceptible populations. The existing databases have been designed primarily for regulatory purposes and are not necessarily well suited for answering questions relating to exposure and health inequities. Pollutant levels can vary both horizontally and vertically, particularly in urban settings. Traffic patterns may result in pollutant levels very different in areas just blocks apart. In addition, building characteristics, which may be related to socioeconomic factors, can affect the movement of ambient air pollution into indoor spaces and resulting personal exposures. Along with the need to better characterize pollutant concentrations and exposures in urban environments, access to this information by communities, researchers, and decision makers needs to be ensured.

Equally as important as the types of data collected are the locations for monitoring urban pollution. Current monitoring sites are not always located to reveal urban inequities. For the most part there is insufficient spatial and temporal detail to answer exposure-related questions in urban environments. The current monitoring locations may or may not include hot spots and represent the entire exposure area. Dispersion modeling can be used to identify where these monitors may best be located, but a sufficient number of monitors is needed to assure that high-concentration areas are found.

Hot spots are currently defined by air quality data alone. More holistically, health and psychosocial stressors should be included in the modeling and hot spots redefined to include areas of significant health inequity. Siting of air pollution monitors should include site selection based on health hot spots and proximity to facilities and intersections of concern likely to increase exposures. Expansion and better targeting of existing air-monitoring systems would require a concerted effort of local, state, and federal agencies, along with participation by academia and community interests. Federal partners in monitoring would include the U.S. EPA, the National Institute for Environmental Health Sciences, the U.S. Department of Energy, the U.S. Department of the Interior, and the U.S. Centers for Disease Control and Prevention.

Although the State Implementation Plans (SIPs) required under the Clean Air Act often use dispersion modeling of criteria pollutants to identify high-concentration locations, analyses of toxic chemicals remain quite limited, particularly at the level of specificity needed to distinguish exposures in one community from those in another. The U.S. EPA made a first attempt to quantify and locate the highest concentrations of a series of toxic chemicals in the CEP. Because of the national scope of this study, it was not possible to develop the detailed local level data necessary to identify hot spots. The study was designed to estimate average concentrations for each census tract and not to identify peak concentrations that might occur inside a census tract. Urban areas need to be reevaluated, using more detailed information and knowledge of the communities, to identify high concentration areas in order to appropriately evaluate the relationship between hot spots and inequity issues.

Continued application of refined modeling to the cumulative impacts from various sources on a neighborhood scale would also contribute to more appropriate analyses of inequity issues. Modeling analysis could be refined by including additional parameters such as the refined deposition properties of pollutants and robust simulations of chemical reactions and the decay of hazardous air pollutants.

California is a leading state in obtaining ambient data for criteria and toxic pollutants and in modeling the emissions from urban air pollutants. As other states increase their abilities to obtain ambient data and improve their modeling capabilities, they could learn from the changes that California is making to its approach. A report of lessons learned from California that includes the perspectives of regulators and communities would be a very useful resource. Finally, modeling and monitoring need to be translated into public policy. Data, once gathered and analyzed, must be acted upon, not further debated.

Adoption of an interdisciplinary approach to data collection and analysis. Existing data sources from other sectors can be used to learn more about health variations in urban populations. Several potential sources of valuable data for secondary use are available from administrative record systems, including vital events registries, hospital admissions, emergency department visits, Health Care Finance Administration records, health maintenance organizations, and the Department of Veterans Affairs medical system. Supplemental analyses of previous epidemiologic studies with individual-level data, including the Harvard Six Cities Studies (*27*), the Harvard 24 Cities Study (*34,122*), and the NCICAS (*71,123*), could also add to our understanding of urban health inequities.

An interdisciplinary approach to analyses of the available data is also needed. Health departments should play a more central role in analyzing and using air quality data, and there should be local annual reports on air quality, with public meetings coordinated by the appropriate health and environmental agencies. Community environmental health advisory boards should be established that are trained and educated to interact with communities and to guide this ongoing effort.

Collection of air pollution data can be made much more informative through an interdisciplinary approach. One example is by expanding the use of geographic information systems technology in the construction of air-monitoring databases. Data on air pollutant levels and traffic patterns could be overlaid with data on other risk factors such as specific pollution sources, crowding, and poverty. It appears that psychosocial stressors, including but not limited to SES, may act as environmental toxicants independent from, but synergistic with, air pollutants. Current analytic methodologies and associated databases regarding air pollution and health in urban environments are incomplete, in that they do not include many health indicators such as psychosocial and related stressors, which are increasingly understood to affect individual biologic function on a number of levels.

The term “biopsychosocial model” characterizes the nested, interactive ecology of biology, mental function, and social status and relations in a range of human pathologies (*124–128*). The biopsychosocial model has not yet been widely applied to understanding the effects of air pollutants. According to this model, the effects of airborne or other toxic substances can be understood in the context of individual biology only when socioeconomy and personal and perhaps community history, which also profoundly affect that biology, are also considered. The approach implies that our understanding of the effects of toxic exposures must take place in the context of the multiplicative, synergistic impacts of the many other physiologic, psychologic, and social stressors that constrain, and indeed largely define, life in marginalized urban neighborhoods.

Existing pollution models can be expanded to incorporate the biopsychosocial model by including indices derived from the many administrative data sets available from government agencies, using standard tools available from population and community ecology. These data range from the decennial

U.S. census to annual school achievement indices, housing inspection information, monthly or even daily demand for law enforcement, fire extinguishment, and other emergency services. Standard ecosystem index methods can be applied to create local indices of psychosocial stress from such data. Incorporation of these indices with existing pollution models and data would permit tests of synergistic models against health status data available from various agencies on different scales.

Community access to information. Residents of low-income areas and communities of color recognize that air pollution exacerbates asthma and other respiratory diseases. In this context, databases and monitoring can be important tools for community education and for affecting public policy change. Making these data both accessible and understandable to community members, however, is a major challenge. The rapid development of technology and resources such as the Internet make accessibility possible, but only to the extent that computers are available. For example, the Internet is already being used for United States–Mexico border air quality information (*129*). In addition to accessibility, there is the need to develop ongoing relationships between governmental agencies and communities, perhaps through the development of regional centers or regional meetings, such that problems and issues can be approached from a long-term perspective as opposed to multiple ad hoc solutions. Communities have not been actively included in the development and use of databases. Reports on data should be prepared quarterly in lay language and explained in public meetings where communities can bring their own expert resource persons. Most low-income areas are widely served by local community- and church-based organizations. These organizations are important resources for community access to information and advice on appropriate local responses to the information.

Programs such as the Environmental Monitoring for Public Access and Community Tracking of the U.S. EPA should be widely implemented (*130*) and data made available for community-based research (CBR) and analysis, as in "Holding Our Breath" from the Communities for a Better Environment (*131*). Agencies should be certain that there are no barriers keeping data and analytic results from affected communities, so that they may respond at the health behavior level as well as political level. Resources and tools for community-based analyses should be expanded to increase participatory decision making and the likelihood of behavioral adaptation for better health outcomes.

Targeted exposure assessment. A more comprehensive description of actual exposures of urban populations would significantly enhance the ability to characterize risk from ambient concentrations. A potential step toward this end point is the use of broad-brush evaluations, such as the National Air Toxics Assessment of the U.S. EPA, to better delineate what chemicals may be present in different communities. Monitoring of emissions and exposures could then potentially be targeted toward specific chemicals. The use and further development of passive dosimeters, for example, would provide a noninvasive means of monitoring VOC and other HAP exposures that is both easy and practical. Because of their ease of use, passive monitors are an excellent tool for direct involvement of communities in monitoring ambient concentrations and personal exposures.

Along with a better description of urban exposures, additional research is needed on the following components of exposure assessment in urban settings:

- Time–activity patterns in specific community settings.
- Ventilation characteristics of urban apartment buildings. It is hypothesized that vertical flow may favor influx of traffic-polluted air at ground level.
- Microscale variations in ambient concentrations of HAPs as they relate to local sources. Studies are needed on vertical as well as horizontal gradients in concentrations over small spatial scales.
- Indoor/outdoor HAP relationships.
- The relationship between the political, social, and economic context on the one hand and pollution source-siting decisions on the other.
- Better air quality emissions inventories, to include both emissions factors as well as location information, or techniques for better specifying locations in the case of small dispersed sources. Other model improvements that may be important in specific urban areas include certain unique features of urban areas, such as terrain and building downwash. Models and methods for making better use of community-based source inventory data sets should be developed.

Community-level capabilities for carrying out exposure studies. The current capacity for community-based studies of personal air pollution exposures in urban areas is quite limited. Few communities possess the technical expertise to tackle such studies independently, and few scientists have yet become engaged. Addressing these gaps will require the formation of participatory CBR partnerships in the form of active collaborations between research and community groups in the conceptualization, design, staffing, execution, analysis, and reporting of community studies. This type of research has potential advantages over nonparticipatory community-based research or community-driven research, including enhanced enthusiasm and support for the research at the community level, greater study enrollment rates, availability of community interns for field work, increased credibility of findings, greater understanding and acceptance of research findings by the community, and improved tools for community advocacy.

One challenge to overcome is the tension that often exists between the goals of community advocates and those of research scientists. Community groups usually are in the position of advocating policy changes. Scientists are often uncomfortable working in such settings because of concerns about the effects on the scientific process and the possibility that media attention and overinterpretation will affect the ability to publish the data in peer-reviewed journals. Another challenge is that available technology for air monitoring is often limited in utility and accessibility for addressing personal exposures. Ideal technology would be affordable, portable, and provide real-time monitoring of criteria pollutant and air toxic concentrations. Most current technology, however, is expensive and blunt in terms of temporal resolution.

In spite of these limitations, air monitoring has an important role to play in community-based studies of air pollution exposures. Based on the experience gained in several existing research partnerships, it may be possible to develop a standardized, inexpensive research tool kit that could be adapted by community groups for their own studies. Aspects of the kit might include forms, protocols, and equipment for traffic counting and $PM_{2.5}$ and nitrogen dioxide sampling.

Health Effects Research

Characteristics that affect health can be at the individual, neighborhood, community, or even national level. Consequently, research approaches, as well as intervention strategies, differ across the spectrum from individual to national levels of organization. Recommendations were developed with consideration of the various levels of interaction.

Consensus on pertinent health indicators. Numerous health indicators have been studied, as summarized in Table 1, and require further analysis, but others need to be considered as well. Chronic and acute health effects need to be considered separately. Whereas the prevalence rates of some chronic health conditions such as asthma, atopy, cardiovascular disease, and cancer are well documented, there is a lack of sufficient baseline information on prevalence rates at local or state levels, as for conditions childhood asthma. Also lacking is experience with newer, more sensitive outcomes, including

- Average level and daily variation of physiologic measures such as bronchial hyperresponsiveness, heart rate, and heart rate variability.
- Biologic markers of exposure to ambient air pollutants such as DNA adducts to polycyclic aromatic hydrocarbons.
- Incidence of subclinical events such as respiratory symptoms and discharges of implanted automatic cardiac defibrillators.

New or novel pollutant indices are also needed, such as

- Estimates of spatial and temporal gradients or profiles of exposure on fine scales.
- Subjective impressions of exposure to traffic, as well as objective indicators such as measured proximity to major traffic arteries and automobile and truck counts.
- Direct, ongoing measurements of ambient air pollutants including nitrogen dioxide, carbon monoxide, ultrafine particle counts, particle acidity, and bioaerosols and their interactions with ambient and indoor air pollutants.

Indicators of health outcome events can be broadly grouped as mortality, medical morbidity measures, and physiologic parameters. Mortality, both total and cause-specific, is routinely available and well studied in relation to air pollution. Although mortality statistics have well-known limitations for specific diagnostic groupings, the overall mortality rate is a fundamental measure of public health. The rate of death from COPD is a useful indicator of the frequency of the disease in the population, and asthma deaths should be considered sentinel events.

A variety of medical morbidity indicators are available, although quality and completeness may vary depending on the types of health systems in place. The Health Care Financing Administration data on Medicare enrollees provide virtually complete coverage of significant health events in persons 65 years of age and older. The data files are cumbersome, but information on both hospitalizations and outpatient visits can be obtained. For example, Medicare hospital admissions in Chicago were analyzed to identify predisposing conditions for increased risk from air pollution (*132*). A nearly 2-fold increase in hospital admissions for cardiovascular diseases associated with air pollution was found among patients with concurrent respiratory infections. Similarly, individuals with asthma were twice as likely to be admitted for pneumonia associated with PM_{10} than those without asthma. Other potential indicators of events might be obtained from administrative databases associated with health systems, such as general hospitalization rates, emergency room visits, prescriptions filled, and medical devices sold. School or work absence rates, although rarely used, are an additional indicator of adverse events.

Mitigation of nongenetic susceptibility factors. Nongenetic susceptibility factors have not been well studied in the context of air pollution health effects. Community-level factors can be broadly classified as socioeconomic indicators; rates of disruptive and dangerous behaviors, including violent crime and drug use; and measures of the adequacy of the community's structure, including housing quality and adequacy of essential services. At the household or individual level, there are a variety of potential susceptibility indicators, some reflecting environmental exposures and others personal and lifestyle characteristics. Additionally, higher-level factors may also be relevant, such as medical care access and quality. Some of these nongenetic susceptibility factors are potentially amenable to intervention. Housing quality can be improved, for example, and overcrowding can be addressed. Unfortunately, income—a critical determinant—is not readily amenable to direct intervention.

Development of a national asthma surveillance system. With asthma morbidity being one of the most common and visible health effects from air pollution, a national asthma surveillance system is clearly needed (*133*). Any asthma surveillance system must address incidence, morbidity, and mortality. To date, an effective system for monitoring the incidence of asthma has not been developed. An understanding of asthma incidence is necessary to address changes in prevalence. A model approach could be developed in several representative locations, including the possibility of using preschool and schools to capture incidence from the responses to questionnaires completed by parents. Morbidity needs to be tracked to capture the face of the disease in the population. Again using schools as an example, it might be possible to track the number of inhalers maintained by the school nurse. Finally, mortality should be more fully utilized in an asthma surveillance system, as it represents a sentinel event that should always prompt investigation. Each asthma death provides an opportunity to learn more about potentially remediable exposures, and use of case-based investigations for all asthma deaths is strongly recommended.

Overall recommendations. Overall recommendations for the next steps in health research fall into three broad classes: further research on effects of specific factors, intervention research, and intervention programs.

Research on effects. Nongenetic susceptibility factors that require further research include diet, respiratory infections, and community stability and other characteristics. This is far from a comprehensive list, but serves as an illustration of the range of factors that needs to be considered. For diet, for example, studies might be carried out to assess modification of the health effects of air pollution by antioxidant supplementation or dietary change.

Intervention research. Intervention research is needed on how to best implement programs directed at exposures with characterized health effects, including exposures to allergens, combustion sources, and overcrowding. Intervention research should also be directed at such factors as access to medical care, level of health knowledge, and health behaviors. With allergen exposures, for example, clinical trials or other intervention research with evaluation would be warranted.

Intervention programs. Intervention programs need to be developed on the basis of evidence from research on the efficacy of interventions. For some factors, intervention programs are already warranted on the basis of available evidence, as with smoking and housing quality.

Community-Based Intervention

Community-based research. The compelling evidence that health inequities exist in urban environments raises important questions about why the economically disadvantaged are at disproportionate risk from asthma and other adverse effects of outdoor air pollution. CBR can be applied in a number of ways to identify and reduce health disparities arising from air pollutants in urban populations. By including an action component, CBR distinguishes itself from more traditional basic or clinical science, which does not have a direct and immediate impact on the community. Improvements in health and living conditions are a priority for most community-based organizations, as is the power to make their own decisions and to take actions to protect their health. For this reason many scientists who use traditional approaches are not familiar with the positive aspects of CBR. Among the attributes that community-based approaches offer are

- Research efforts are more cost-effective, with communities offering staff, facilities, and other resources that can leverage the traditional academic environment.
- Data collection is of better quality and quantity, provided the staff is properly trained and supervised.
- Recruitment and retention within studies are better when community members are more directly involved.
- Input from the community on what may or may not be effective can result in improved and novel hypotheses, study designs, and evaluation methods.
- Knowledge and findings can be applied more readily to intervention strategies.

- Data on community-identified concerns can be translated into action by the people affected.
- More successful translation of science to policy will lead to more informed public policy decisions.
- Health considerations can be inserted into economic development dialogues.
- Interventions are more likely to be sustainable with community participation in their development.
- Collaborations increase credibility, recognition, and trust for both community members and academic researchers.

Despite these advantages, CBR remains only a fraction of the total effort devoted to population-based research. A number of barriers, some of which are intuitive, account for the difficulties associated with developing and promoting CBR on a broader scale:

- Academic recognition for CBR is not always the same as that devoted to more traditional public health research.
- Credibility for community members is threatened when they risk becoming more closely associated with academic scientists rather than their grassroots colleagues.
- Credibility for scientists is questioned when they risk becoming more closely associated with advocacy groups.
- Expectations of the groups differ. Communities may expect to have their problems identified and solved. Scientists may expect to publish a number of articles on an annual basis.
- Difficulty in establishing trust.
- Limited financial resources.
- Limited time.

A potential strategy to optimize CBR on eliminating exposures contributing to health risks requires understanding of the factors leading to differential exposures. Recognizing that community interests in resolving inequities will differ, only a general discussion of overarching strategies is presented. These are organized along the lines of the primary pathways to health inequities related to urban air pollution: pollutant sources, education and training, community infrastructure and services, and the planning and financing process.

Pollutant sources. Some community-based strategies to address the disproportionate number of sources in highly affected communities include

Source reduction. Local point sources like bus and truck depots, dry cleaners, auto-body shops, hair salons, and other polluting facilities are examples of common sources in urban communities. These sources often affect receptors both indoors and outdoors. Community-based strategies to reduce sources often are framed as the struggle for environmental justice and take many different forms including political, legal, and educational campaigns. Mobile sources in particular are major contributors to air pollution in most urban communities. Strategies to reduce traffic (e.g., campaigns to improve public transit) are particularly important for reducing health inequities. Stationary polluting facilities can significantly reduce air emissions by using best management practices.

Cleaner fuels. Because mobile sources are a major source of pollution in urban neighborhoods, using vehicles that emit lower pollution levels through the use of cleaner fuels is one strategy for reducing air toxics in highly affected communities. A community-driven campaign for cleaner fuels in public bus and truck fleets is one strategy toward that end.

Cleaner products. Encouraging the use of cleaner products in both businesses and households is another strategy to reduce pollution sources and consequent health inequities in urban communities. Chemical and toxic products can add to urban air pollution. When hair salons reduce their use of chemicals and households use less-toxic cleaners, fewer toxics are released into the indoor and outdoor environment. Educational campaigns for both businesses and residents are one strategy to encourage the use of cleaner products. Making cleaner products more accessible and affordable in urban communities is another strategy.

Education and training. The lack of knowledge about the relationship between pollution sources, receptors, and resulting health effects is one of the root causes of health inequities from air pollution. Healthcare providers, residents in highly affected areas, and regulators all lack the information necessary to take real action. Some community-based intervention strategies to address this lack of knowledge include the following:

Healthcare provider education. Specifically, there is a need to include curricula in both medical schools and continuing medical education forums on the relationship between outdoor air quality and health effects. By including this kind of curriculum in medical schools, there is an opportunity to train future doctors and healthcare providers to recognize and explore the connections between urban air pollution and health inequities. Practitioners will be better reached by continuing medical education methods. Other means of educating healthcare providers about these issues include grand rounds, peer education, and partnerships with community health workers.

Recruitment of scientists of color and other ethnic minorities. Currently there are few scientists of color and other ethnic minorities working on public health issues. By exposing young people from highly affected communities to public health and medical fields at an early age, there is an opportunity to attract them to these careers. Ultimately, this may lead to more scientists concerned about and researching these issues. Public health curricula in public schools, medical and healthcare internship opportunities, and scholarships for scientific fields are all strategies to engage and train ethnically diverse scientists.

Patient/resident education. One strategy to reach residents in highly affected communities is through patient groups where patients suffering from similar diseases meet regularly to discuss causes and management. Another is through public schools. Curricula on public health, specific diseases (like asthma), and environmental issues can help students of many levels understand the relationship between urban air pollution and health effects. Curriculum materials and information can then reach parents and families through the students. Working with school nurses can be an excellent means of reaching students who are highly affected by urban air pollution. Because they see students suffering from asthma regularly, they are in a position to share information about the disease and possible environmental triggers with both students and families. Once armed with information about the relationship between pollution sources, receptors, and health effects, residents in highly affected areas can use a number of strategies (e.g., community organizing, political advocacy) to reduce pollution sources and health inequities.

Regulator education. Regulators also lack adequate information on the relationship between exposure and health effects, particularly in the context of exposure inequities. Without this information it is extremely difficult to create regulatory strategies that effectively address health inequities.

Community infrastructure and services. One of the major root causes of health inequities resulting from urban air pollution is the lack of access in low-income communities and communities of color to quality housing, transportation, healthcare, and other social resources. Community-based interventions to address these social problems are complicated. They include efforts to improve

- Quality of housing in highly affected communities. Poor indoor environments such as those with mold, dust mites, rodents, or cockroaches can add to or exacerbate health inequities resulting from urban air pollution.
- Access to and quality of public transportation. This will not only help to reduce traffic, but will facilitate access to quality jobs and healthcare.
- Access to and quality of healthcare. Many residents of low-income neighborhoods and communities of color are forced to

use emergency room care rather than basic maintenance care to address health problems. As a result, diseases like asthma are much more serious and many more people are hospitalized than in communities where disease management is more adequate.

- Other social concerns such as education and job training. Without adequate education and employment, access to basic services such as healthcare is impaired. In addition, scientific research is showing that psychosocial stressors play a role in health inequities from urban air pollution and other environmental problems (*134–136*).

Planning and financing process. The process and opportunities by which disenfranchised communities become involved in planning and financing activities affecting their lives should be improved. Often we find that communities with inequitable exposures to air pollutants also encounter poor housing, poor schools, higher unemployment, and fewer options for healthcare. Where these communities comprise more recent immigrants, they tend to be disconnected from public participation because of language barriers and mistrust of government. CBR strategies for addressing these problems include the following:

Developing a code of "Best Practices for Community-Based Research." Such a code should include guidance on how to more fully incorporate community participation. Negotiating among competing interests of scientists, community activists, public agencies, and community members is often not an explicit activity for CBR, but needs to become so.

Forming a multidisciplinary team of investigators. Effective treatment of complex societal problems requires not just epidemiologists, biostatisticians, and environmental scientists, but also others experienced in public finance and administration, community organization, and social sciences, among other specialties. CBR needs to be based in a capacity to assess underlying causes, not just overt symptoms of inequities.

Recognizing that communities have assets, not just problems. In order for our academic institutions to play a meaningful role in CBR, it is important to also acquire the experiential sensitivity that comes from those who have lived with inequities. Often we find well-intentioned investigators who have had all their formative education and experiences shaped by middle-class American values. Working with underserved urban populations requires a reframing not easily understood by many academic scientists. One method of ensuring that both research and interventions are adequate and address the problem at hand is to involve community residents at all levels of research, intervention, and policy development. Residents living in communities suffering from health inequities related to urban air pollution can provide information and expertise that is vital to successful research, but often ignored.

Developing competent staff and professionals from the ranks of racial/ethnic minority groups through training programs. Funding agencies should recognize this as a specific need and provide new categories of training. A focus on CBR must be incorporated into individual and institutional training programs in environmental health, especially those in epidemiology. Moreover, although separate funding is already available for minority candidates for advanced scientific training, funding is needed for internships for high school and college students to work directly on CBR. Targeted funding to develop interest and experience among promising young people will also help to bridge the sensitivity gap of investigators. An interest among minorities for careers in environmental and public health sectors should be cultivated by exposing high school and college students early on to the skills and knowledge required of these professions. The mentoring that will take place as these students work closely with staff and investigators on CBR projects will motivate educational choices. In addition, it is critical that students are well advised on the high school and college curricula that prepare them for advanced studies.

CBR offers the opportunity to confront disproportionate exposures and health effects from a participatory, action-oriented framework that unites disparate groups, leverages individual resources, and influences research, intervention, and policy at all levels. Public participation is fundamental to CBR and presents the best means of addressing health disparities in an effective and efficient manner. To facilitate this process, long-term support for community-based organizations and for researchers conducting such work is required. Coalitions among communities, scientists and providers need to be fostered. In addition, similar partnerships among industries, unions, and regulators can lead to enhanced management practices. Finally, the underlying social environment and its contribution to health inequities must be better understood at the time we address the physical environment, thus requiring collaborations among behavioral and biomedical scientists. With contributions from all of these parties, community-based strategies can be developed to assess and reduce hazardous exposures and to prevent adverse health effects.

Public Policy

Management of differential air pollution exposures though public policy needs to be viewed in the broader context of distributive inequities across society. Under Executive Order 12898 (*6*), federal environmental regulatory programs must address differential distributions of exposure. Early analysts of clean air regulation recognized equity as a trade-off between two competing forces. On one hand, air pollution control measures had a regressive economic impact on low-income and racial/ethnic minority communities, costing those at the bottom of the economic ladder much more than those at the top. On the other hand, those same measures promised greater net improvements as uniform standards took hold in these same disproportionately polluted communities (42 USC Sections 1857c-4, 1957c-5, reflecting the Clean Air Act as passed in 1970, promised uniform pollution control "regardless of where…persons reside" across the land) (*137,138*).

The principal regulatory structure for addressing ambient air pollution, the Clean Air Act of 1970 (*102*), provides different control strategies for ambient air (or criteria) pollutants and HAPs (*139–141*). Although the U.S. EPA is responsible for promulgating ambient air quality standards for the criteria pollutants, the states must develop SIPs to attain and maintain these standards according to time lines established in relation to the baseline air quality of each state. Factored into the planning process at the state level are the U.S. EPA mobile source emissions standards, though some states, notably California and several in the Northeast, have been granted a statutory dispensation to develop more stringent emissions standards. The HAPs program was amended in 1990 to require stationary sources of 189 specified toxicants to effectuate 90% emissions reductions by the end of the decade, using existing technologies (*142*). Any further reductions after that time are to be based on an assessment of residual risks. In some states, regulation of stationary sources falls within the jurisdiction of local air pollution control agencies; in others, regulatory efforts are concentrated at the state level.

Multiple opportunities exist to address environmental justice concerns in the regulatory programs for criteria pollutants: *a*) standard-setting; *b*) establishing requirements for monitoring, including siting criteria; *c*) developing and reviewing SIPs submitted by the states; *d*) reviewing permit decisions made by state and local air pollution control agencies; and *e*) imposing more stringent requirements for public participation at all stages of the regulatory process, which would also be facilitated by increasing data availability and transparency. The U.S. EPA could also apply the latter two strategies to the HAPs program and could, in addition, incorporate incentives for reducing health inequities into the agency's cross-pollutant initiatives, such as the Urban

Air Toxics Strategy (*143*) and the Economic Incentives Program (*144*). The directives of Executive Order 12898 could be invoked at each level. We present several examples.

Air quality standard-setting. Ambient air quality standards are to be set with a margin of safety adequate to protect public health (*140*). Such standards are not intended to provide absolute protection to all individuals, but the legislative history of the Clean Air Act makes it clear that identifiable susceptible subgroups such as persons with asthma should be protected. It could be argued that such recognition should be explicitly extended to large subsets of susceptible subgroups. Such a subset might include poor, urban, African Americans with asthma who may be rendered more susceptible to the health impacts of air pollution by suboptimal access to appropriate medical care, widespread exposures to indoor aeroallergens and tobacco smoke, and a variety of other psychosocial, nutritional, and environmental stressors. Although it is still uncertain whether such persons with asthma are differentially susceptible to the impacts of air pollution, such concerns could be factored into the margin of safety component of ambient air standards. Under Executive Order 12898 this scenario, or others representative of the urban poor, may warrant consideration by the U.S. EPA.

Monitoring requirements and siting criteria. In principle, the states and local air districts, which are responsible for siting and operation of air quality monitors, are required to locate some monitors in hot spots as well as in sites representing regional rather than local pollutant concentrations (*145*). In many jurisdictions, the vast majority of criteria air pollutant monitors are sited away from hot spots, as the latter are considered unrepresentative of regional population exposures. Thus, an air basin or a state may be considered to be in attainment with the federal ambient standard for a given pollutant even though substantial numbers of people residing, working, or going to school near busy roads or stationary sources may be exposed to concentrations exceeding the standard. Recent studies have indicated that low-income and racial/ethnic minority communities live in closer proximity to such hot spots than other population groups (*146,147*).

Intense, localized monitoring efforts are needed for hot spot identification and exposure reduction. The U.S. EPA should provide specific guidelines to state and local authorities to monitor traffic-related and point-source hot spots affecting substantial numbers of people. There are also opportunities for collaboration among community groups, university researchers, and regulatory agencies in the identification and pilot air monitoring of such hot spots. The results of hot spot monitoring should also be factored into criteria for determining attainment with ambient air quality standards.

Although the issue of hot spots highlights the difficulty of monitoring for inequities across different spatial scales, lack of long-term monitoring data pertinent to many low-income and racial/ethnic minority communities is an equally substantial obstacle to meaningful health interventions. The decommissioning of monitoring networks (due to urban economic crises over the last 20 years in cities like New York; Cincinnati, Ohio; Gary, Indiana; and Detroit, Michigan) has left inadequate data for many inner-city communities to evaluate relationships between contemporary disease patterns and historical air pollution trends. In the interest of study design, major personal exposure studies have omitted racial/ethnic minority populations (*27*). Only recently has there been renewed interest in understanding personal exposure in urban communities (*119,148*).

State Implementation Plan review and development. The NAAQS are set by the U.S. EPA for carbon monoxide, lead, nitrogen dioxide, ozone, PM, and sulfur dioxide, as mandated under the Clean Air Act. Low-income and racial/ethnic minority people live in disproportionately high numbers in areas out of attainment with the NAAQS and are thus dependent on air quality policy and regulation for eventual relief (*149,150*). SIPs are required for all such nonattainment areas and embody a jurisdiction's policy priorities for clean air. These plans offer the structure within which local and regional air quality is regulated. The major concerns of urban communities are the equity implications of core SIP provisions like emissions trading, transportation control measures, and other technologic or regulatory mandates.

Emissions trading. In the name of economic efficiency, many economists and regulators have embraced the concept of market-based emissions trading as a cost-effective way to improve regional air quality. For instance, large stationary sources may contribute to vehicle buy-back programs intended to remove older, more highly polluting vehicles from circulation. A local source that has contributed to such a program may then receive credits against its own emissions, allowing it to forego emissions reductions that might otherwise be required. This approach may lead to a net regional reduction in air pollution while allowing continued or increased exposures at the local level. A number of state and local air pollution control districts have encouraged the trading of VOCs or HAPs as a way of reducing regional ozone precursors, yet point sources of these same pollutants may pose even greater risks to local communities. Undesirable distributional outcomes are an inherent risk for any system developed primarily to ensure efficiency, and emissions trading is no exception (*151*).

In many cases, emissions trading is a compliance alternative available to local authorities in lieu of specific SIP measures, but verifiability and enforceability of trades may be problematic. In the case of mobile-to-stationary trades, vehicle scrapping programs may not achieve reductions above and beyond normal obsolescence. In Los Angeles, local officials have sought credit for reductions in military flights completely unrelated to air pollution control measures (*152*). Such trades may not meet the legal threshold of being surplus or above and beyond what would normally have been expected to happen.

A number of environmental justice groups, particularly in California, have suggested the elimination of emissions trading (*153*), or at the very least, great caution in its deployment. General criteria for limiting trades have been proposed:

- Ensure no net increase in air pollution in heavily affected communities, defined as those currently in violation of ambient standards for criteria pollutants or their precursors (*154*). Specifically, in-trading to such areas could be proscribed. Even for communities currently below the ambient standards, the potential cumulative health impacts in a community should be considered in HAPs trading when emissions are projected to increase above existing baseline emissions.
- Endow lower-income communities with property rights within the trading system. Regulatory authorities could allocate trading credits to heavily polluted communities, which would allow them to negotiate with more distant intraregional point sources to effectuate reductions locally (e.g., through the purchase of low-emitting mass transit vehicles).
- Assure verifiability and enforceability of trading practices. Given the discretion granted regulators and polluters in most trading programs, higher standards of accountability must be introduced to ensure that trades that do occur in fact lead to permanent reductions in the overall emissions inventory.

Objective, reliable judgments about the efficacy of these or other criteria would be promoted by standardizing methods for quantifying emissions reductions, which currently vary substantially among and, in some cases, within jurisdictions (*152*). Such standardized methods should be promulgated at the federal rather than at the state or local level in order to achieve widespread uniformity and predictability in application.

Transportation control measures, and other State Implementation Plan–based policy initiatives. A wide range of initiatives pertaining to infrastructure, mass transit, regional planning, and technology change (e.g., low-emission vehicles) is written into the Clean Air Act Amendments of 1990 (*142*) under the rubric of transportation control measures. The emphasis placed on different measures and the timing under which they are introduced have significant implications for the health and well-being of urban communities. New road usage fees and construction, new mass transit projects, and altered planning priorities all offer the hope of reducing emissions. They also have the potential, however, to disrupt the economic viability of inner-city communities. In Los Angeles, for example, analysis of plans had shown that the new subway would have an adverse impact on racial/ethnic minority bus riders while providing minimal service in their communities (*155*). New road usage fees and stricter tail pipe controls also have a disproportionate impact on poorer commuters (*156,157*). Stricter environmental controls on small industries often threaten the livelihoods of already marginal employees (*158*). These issues can and should be considered and addressed in the development and implementation of air pollution control strategies.

From an environmental justice perspective, the SIP is a two-edged sword. It is the blueprint for a region's compliance under the Clean Air Act, but without environmental justice stakeholder participation in its design and close attention to the equity impacts of its implementation, it can exacerbate environmental conditions in local communities.

Permitting and environmental justice. One key mechanism of disproportionate exposure may be the issuing of permits by state and local regulatory agencies to facilities in close proximity to vulnerable populations, or the failing of the agencies to trigger permit and/or new source review requirements. In the latter case, the cumulative effect of a number of small facilities may be enough to create air pollution hot spots. Consequently, the siting of permitted facilities has come under increased scrutiny in recent years. Title VI of the Civil Rights Act prohibits the use of federal funding in activities that lead to discriminatory outcomes (*159*). This provision has been invoked in recent times to challenge local and state permitting processes that may have led to a disproportionate local pollution burden. Many complaints filed under Title VI in the past few years have been against air permitting programs (*160*). Although at the time of this workshop the U.S. EPA was still drafting guidelines for permitting compliance with Title VI, state and local agencies should consider Title VI for preventing disproportionate siting of noxious facilities in urban communities.

Some facilities with significant local air quality impacts may not fall under SIP permitting requirements, depending on the jurisdiction. These include waste transfer stations, bus depots, small industry, and new commercial or residential development. Nonattainment areas are, however, often still required to account for such facilities under federal, state, or local environmental impact requirements. New York City, for example, requires no individual permits for over 270 transfer stations in its five boroughs. Many persistent local sources of air pollution can only be addressed through renewed attention to the environmental review process at the facility level. A number of tools exist to improve this process. For example, several U.S. EPA regions and some states and localities have added a cumulative dimension to environmental impact reviews to better contextualize the overall burden faced by local communities. The addition of performance standards for siting facilities that otherwise comply with zoning codes represents another innovative approach to incorporating air quality concerns in land use regulations. Finally, the environmental review process itself needs to be taken more seriously by local government and stakeholders. Clean air will not come as a result of a "rubber-stamp" process. Environmental review of siting and development decisions often represents the only regulatory handle available for bringing the bulk of a region's polluting activities into compliance.

Public participation and increased data availability. Substantive public participation is a fundamental concept of environmental justice. In principle, public participation is already an administrative requirement in the SIP process (*102,142*). Attempted notification of the public through newspaper announcements alone, however, will not reach large numbers of individuals who have a legitimate stake in the process. The U.S. EPA, as well as state and local regulatory agencies, could expand public participation requirements and practices consistent with Executive Order 12898. For instance, active local involvement in siting decisions could be enhanced through specific outreach to community-based organizations, churches and other religious agencies, and possibly directly to individual residents (*161*).

To facilitate public participation, air quality and emissions data should be made widely available, and all the steps leading to data publication should be as transparent as possible. For example, agencies at local, state, and federal levels could make relevant emissions, monitoring, and modeling data readily accessible (e.g., on the Internet) in user-friendly formats as soon as such data have received appropriate internal reviews. In publishing data electronically, agencies should also ensure that underlying raw data (i.e., those that have been subject to standard quality assurance/quality control) can be accessed so that users can derive reports useful to their specific needs. Agencies should attempt to give a context to such data to avoid unnecessary confusion while at the same time increasing their outreach to assist community organizations to understand, evaluate, and use the information.

To remedy the historical underrepresentation of poor urban populations in environmental decision making, agencies should also consider forming specialized stakeholder groups for environmental justice. Such groups can provide useful, direct policy feedback and can also help ensure broader participation in specific cases. New Jersey, California, and New York are a few of the states that presently have statewide committees advising decision makers on environmental justice policies. Local governments should follow suit.

Measures to address diesel emissions. Within urban areas, fleets of diesel-powered buses and trucks represent a significant source of exposure to fine particles, VOCs, nitrogen oxides, and other toxicants. Though some transit agencies and school districts have considered the air quality impacts of their activities, others have continued to augment their fleets with diesel technology. Several approaches to reduce the urban burden of diesel exposures include the following:

- Providing funding incentives to transit agencies to purchase compressed natural gas, electric, or other less-polluting vehicles.
- Providing federal guidance for stricter interpretation of transportation conformity rules that govern the methods that state and local agencies must follow in preparing an emissions budget for transportation.
- Requiring diesel inspection and maintenance, if not emissions control retrofits, for heavy- and medium-duty diesel vehicles as a part of the SIP process.
- Requiring the production and use of cleaner, reformulated diesel fuels similar to those used in Sweden, and the retrofit of state-of-the-art pollution controls on existing diesel fleet vehicles.

Odor control. Uncontrolled odors represent one of the major inequities of exposure to stationary source pollution. With the diminution of grossly visible pollution from stationary sources, olfactory cues have become more important in public perception of air pollution. Exposure to unpleasant odors can result in annoyance and interference with one's quality of life and may also produce headache and nausea and elicit respiratory symptoms in some individuals with asthma (*162*). In the 1970s the U.S. EPA opted against developing any national strategy to address odors (*163*). In the absence of national regulatory guidance, local districts rely on complaint-based systems for enforcement, which are variations of common-law remedies for public nuisances. Numerous institutional aspects of such complaint-based systems impede resolutions satisfactory to the surrounding communities (*164*).

Odor pollution can be addressed at both the federal and local levels. For a selected number of common, well-characterized odorants, the U.S. EPA could develop ambient exposure standards to be enforced at the local level. As this undoubtedly would take several years to accomplish, local agencies could require interim odor pollution prevention measures, rather than emissions control technology.

Urban air toxics. The Urban Air Toxics Strategy (*143*) is a program for cross-pollutant and cross-media toxics reduction. The Economic Incentives Program (*144*) provides local and state agencies with a wide array of economic tools for reducing air pollution. Both of these U.S. EPA programs hold promise and risks. Failure of community members to participate in the Urban Air Toxics Strategy process may lead to the development of toxics reduction plans that may not address the concerns of those communities most affected. The U.S. EPA could take additional steps to give affected communities a formal stake in the process, such as facilitating opportunities for early and meaningful participation in regional deliberative processes. In the Economic Incentives Program, low-income communities could be endowed with credits that would allow them to trade out high pollution levels. Furthermore, Economic Incentives Programs should prohibit trades that raise local hot spot pollutant concentrations, and provide incentives for trades that remove sources from those communities.

Currently, there is no mandate for federal, state, or local air permitting authorities to consider siting issues while evaluating permit applications. Failure to consider the aggregate of localized emissions in this process has, however, led to clustering of large stationary sources in some communities. To remedy this

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problem and avoid creating new hot spots, all permit applications could be evaluated in their site-specific context, including an assessment of potential cumulative health impacts (e.g., cancers, acute and chronic effects) within the affected community.

In many states, evaluation of potential cumulative and other health effects of exposure to air pollution is hampered to various degrees by the institutional separation of public health and risk assessment expertise from regulatory authority. Forging better organizational linkages between public health agencies and those responsible for implementing air pollution control strategies would likely be helpful not only in siting and permitting decisions, including the evaluation of environmental impact reports, but also in helping to address other community concerns about localized health impacts of air pollution.

Conclusions

The term environmental justice summarizes an extraordinarily complex set of social, political, economic, ethical, and scientific issues in only two words. For urban air pollution, there are disproportionately greater exposures to the poor and to minorities—a problem of environmental justice—as made clear by the available monitoring and modeling data, as well as by the experiences of communities. This workshop explored the environmental justice problem of urban air pollution in depth, considering the tools available to characterize the problem, the needed research evidence to find solutions, approaches through community-based mechanisms to carry out the research, and policy approaches to address inequities of urban air pollution. Although the workshop proceedings document a formidable research problem, useful tools and research approaches are already available, and the participants set out a template for their application. Pathways for gathering existing evidence for policy development are also proposed. However, success will not be achieved without multidisciplinary research groups that include communities as partners. A long-range, progressive research agenda is needed, and funding agencies will need to make the appropriate commitments. New pathways to policy formulation may be needed, particularly to assure that the disproportionately exposed communities are given sufficient voice in policy development.

This workshop offers many suggestions for starting to address urban air pollution and health inequities. The participants were optimistic that much can be accomplished to reduce the risks of urban air pollution; it is time to start a major, national program to achieve that goal.

REFERENCES AND NOTES

1. Council on Environmental Quality. Environmental Quality: The Second Annual Report of the Council on Environmental Quality. GPO Stock #041-011-00089-1. Washington, DC:U.S. Government Printing Office, 1971.
2. McCowan CS Jr, Young JR, Beall KB. Environmental Racism? Available: *http://www.kmlaw.com/eracism.htm* [cited 31 August 2000].
3. Thurgood Marshall School of Law, Environmental Justice Clinic. Environmental Justice History. Available: *http://www.tsulaw.edu/environ/envhist.htm* [cited 31 August 2000].
4. U.S. General Accounting Office. Siting of Hazardous Waste Landfills and Their Correlation With Racial and Economic Status of Surrounding Communities. GAO/RCED-83-168. Washington, DC:U.S. Government Printing Office, 1983.
5. U.S. EPA, Office of Environmental Justice. Environmental Justice. Available: *http://es.epa.gov/oeca/main/ej/index.html* [cited 31 August 2000].
6. White House Office of the President. Executive Order 12898. Federal Actions To Address Environmental Justice in Minority Populations and Low-Income Populations, 1994.
7. Institute of Medicine, Committee on Environmental Justice. Toward Environmental Justice. Research, Education, and Health Policy Needs. Washington, DC:National Academy Press, 1999.
8. American Thoracic Society, Committee of the Environmental and Occupational Health Assembly. Health effects of outdoor air pollution. Part 1. Am J Respir Crit Care Med 153:3–50 (1996).
9. American Thoracic Society, Committee of the Environmental and Occupational Health Assembly. Health effects of outdoor air pollution. Part 2. Am J Respir Crit Care Med 153:477–498 (1996).
10. Holgate ST, Samet JM, Koren HS, Maynard RL, eds. Air Pollution and Health. San Diego, CA:Academic Press, 1999.
11. U.S. EPA. Air Quality Criteria for Ozone and Related Photochemical Oxidants, Vol 3 of 3. EPA-600-P-93-004cF. Washington, DC:U.S. Environmental Protection Agency, 1996.
12. U.S. EPA. Air Quality Criteria for Particulate Matter, Volume 1 of 3. EPA-600-P-95-001aF. Washington, DC:U.S. Environmental Protection Agency, 1999.
13. American Thoracic Society. Guidelines as to what constitutes an adverse respiratory health effect, with special reference to epidemiologic studies of air pollution. Am Rev Respir Dis 131:666–668 (1985).
14. Samet JM. Defining an adverse respiratory health effect. Am Rev Respir Dis 131:487 (1985).
15. American Thoracic Society. What constitutes an adverse health effect of air pollution? Am J Respir Crit Care Med 161:665–673 (2000).
16. Leikauf GD, Kline S, Albert RE, Bater CS, Bernstein DI, Buncher CR. Evaluation of a possible association of urban air toxics and asthma. Environ Health Perspect 103(suppl 6):253–271 (1995).
17. National Research Council, Committee on Research Priorities for Airborne Particulate Matter. Research Priorities for Airborne Particulate Matter. I: Immediate Priorities and a Long-Range Research Portfolio. Washington, DC:National Academy Press, 1998.
18. National Research Council, Board on Environmental Studies and Toxicology. Research Priorities for Airborne Particulate Matter II: Evaluating Research Progress and Updating the Portfolio. Washington, DC:National Academy Press, 1999.
19. U.S. EPA. Environmental Equity: Reducing Risk for All Communities. EPA-230-R-92-008. Washington, DC:U.S. Environmental Protection Agency, 1992.
20. Sexton K, ed. Environmental justice. J Expos Anal Environ Epidmiol 9:1–78 (in entirety) (1999).
21. White MC, Etzel RA, Wilcox WD, Lloyd C. Exacerbations of childhood asthma and ozone pollution in Atlanta. Environ Res 65:56–68 (1994).
22. Schwartz J. Air pollution and daily mortality in Birmingham, Alabama. Am J Epidemiol 137:1136–1147 (1993).
23. Schwartz J, Dockery DW. Increased mortality in Philadelphia associated with daily air pollution concentrations. Am Rev Respir Dis 145:600–604 (1992).
24. Burnett RT, Dales RE, Raizenne ME, Krewski D, Summers PW, Roberts GR, Raad-Young M, Dann T, Brook J. Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals. Environ Res 65:172–194 (1994).
25. Dockery DW, Pope CA III. Acute respiratory effects of particulate air pollution. Annu Rev Public Health 15:107–132 (1994).
26. Vedal S. Ambient particles and health: lines that divide. J Air Waste Manage Assoc 47:551–581 (1997).
27. Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE. An association between air pollution and mortality in six U.S. cities. N Engl J Med 329:1753–1759 (1993).
28. Abbey DE, Nishino N, McDonnell WF, Burchette RJ, Knutsen SF, Lawrence Beeson W, Yang JX. Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. Am J Respir Crit Care Med 159:373–382 (1999).
29. Pope CA III, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, Heath CW Jr. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am J Respir Crit Care Med 151:669–674 (1995).
30. McDonnell WF, Abbey DE, Nishino N, Lebowitz MD. Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the AHSMOG Study. Environ Res 80:110–121 (1999).
31. Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B, Allen G, Verrier M, Cherry R, Verrier R. Ambient pollution and heart rate variability. Circulation 101:1267–1273 (2000).
32. Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, Mittleman M, Baliff J, Oh JA, Allen G, Monahan K, et al. Air pollution and incidence of cardiac arrhythmia. Epidemiology 11:11–17 (2000).
33. Pope CA III, Kanner RE. Acute effects of PM_{10} pollution on pulmonary function of smokers with mild to moderate chronic obstructive pulmonary disease. Am Rev Respir Dis 147:1336–1340 (1993).
34. Spengler JD, Koutrakis P, Dockery DW, Raizenne M, Speizer FE. Health effects of acid aerosols on North American children: air pollution exposures. Environ Health Perspect 104:492–499 (1996).
35. Suh HH, Nishioka Y, Allen GA, Koutrakis P, Burton RM. The metropolitan acid aerosol characterization study: results from the summer 1994 Washington, D.C. field study. Environ Health Perspect 105:826–834 (1997).
36. Wjst M, Reitmeir P, Dold S, Wulff A, Nicolai T, von Loeffelholz-Colberg EF, Von Mutius E. Road traffic and adverse effects on respiratory health in children. BMJ 307:596–600 (1993).
37. Nitta H, Sato T, Nakai S, Maeda K, Aoki S, Ono M. Respiratory health associated with exposure to automobile exhaust. I: results of cross-sectional studies in 1979, 1982, and 1983. Arch Environ Health 48:53–58 (1993).
38. Edwards J, Walters S, Griffiths RK. Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. Arch Environ Health 49:223–227 (1994).
39. Duhme H, Weiland SK, Keil U, Kraemer B, Schmid M, Stender M, Chambless L. The association between self-reported symptoms of asthma and allergic rhinitis and self-reported traffic density on street of residence in adolescents. Epidemiology 7:578–582 (1996).
40. Oosterlee A, Drijver M, Lebret E, Brunekreef B. Chronic respiratory symptoms in children and adults living along streets with high traffic density. Occup Environ Med 53:241–247 (1996).
41. Pekkanen J, Timonen KL, Ruuskanen J, Reponen A, Mirme A. Effects of ultrafine and fine particles in urban air on peak expiratory flow among children with asthmatic symptoms. Environ Res 74:24–33 (1997).
42. Van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B. Motor vehicle exhaust and chronic respiratory symptoms in children living near freeways. Environ Res 74:122–132 (1997).
43. Brunekreef B, Janssen N, de Hartog J, Harssema H, Knape M, Van Vliet P. Air pollution from truck traffic and lung function in children living near motorways. Epidemiology 8:298–303 (1997).
44. Ciccone G, Forastiere F, Agabiti N, Biggeri A, Bisanti L, Chellini E, Corbo G, Dell'Orco V, Dalmasso P, Volante TF, et al. Road traffic and adverse respiratory effects in children. SIDRIA Collaborative Group. Occup Environ Med 55:771–778 (1998).
45. English P, Neutra R, Scalf R, Sullivan M, Waller L, Zhu L. Examining associations between childhood asthma and traffic flow using a geographic information system. Environ Health Perspect 107:761–767 (1999).
46. Koren HS, Graham DE, Devlin RB. Exposure of humans to a volatile organic mixture. III: Inflammatory response. Arch Environ Health 47:39–44 (1992).
47. Ware JH, Spengler JD, Neas LM, Samet JM, Wagner GR, Coultas D, Ozkaynak H, Schwab M. Respiratory and irritant health effects of ambient volatile organic compounds. The Kanawha County Health Study. Am J Epidemiol 137:1287–1301 (1993).
48. Raaschou-Nielsen O, Lohse C, Thomsen BL, Skov H, Olsen JH. Ambient air levels and the exposure of children to benzene, toluene, and xylenes in Denmark. Environ Res 75:149–159 (1997).
49. Yang CY, Wang JD, Chan CC, Chen PC, Huang JS, Cheng MF.

Respiratory and irritant health effects of a population living in a petrochemical-polluted area in Taiwan. Environ Res 74:145–149 (1997).

50. Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Margolis H, Rappaport E, Gong H, et al. A study of twelve southern California communities with differing levels and types of air pollution. I: prevalence of respiratory morbidity. Am J Respir Crit Care Med 159:760–767 (1999).
51. Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Margolis H, Rappaport E, Vora H, Gong H Jr, et al. A study of twelve southern California communities with differing levels and types of air pollution. II: effects on pulmonary function. Am J Respir Crit Care Med 159:768–775 (1999).
52. Boezen HM, van der Zee SC, Postma DS, Vonk JM, Gerritsen J, Hoek G, Brunekreef B, Rijcken B, Schouten JP. Effects of ambient air pollution on upper and lower respiratory symptoms and peak expiratory flow in children. Lancet 353:874–878 (1999).
53. Thomas SD, Whitman S. Asthma hospitalizations and mortality in Chicago. An epidemiologic overview. Chest 116:135S–141S (1999).
54. Gottlieb DJ, Beiser AS, O'Connor GT. Poverty, race, and medication use are correlates of asthma hospitalization rates. A small area analysis in Boston. Chest 108:28–35 (1995).
55. Ray NF, Thamer M, Fadillioglu B, Gergen PJ. Race, income, urbanicity, and asthma hospitalization in California: a small area analysis. Chest 113:1277–1284 (1998).
56. Carr W, Zeitel L, Weiss K. Variations in asthma hospitalizations and deaths in New York City. Am J Public Health 82:59–65 (1992).
57. Lang DM, Polansky M. Patterns of asthma mortality in Philadelphia from 1969 to 1991. N Engl J Med 331:1542–1546 (1994).
58. Marder D, Targonski P, Orris P, Persky V, Addington W. Effect of racial and socioeconomic factors on asthma mortality in Chicago. Chest 101(suppl 6):426S–429S (1992).
59. Seal E Jr, McDonnell WF, House DE, Salaam SA, Dewitt PJ, Butler SO, Green J, Raggio L. The pulmonary response of white and black adults to six concentrations of ozone. Am Rev Respir Dis 147:804–810 (1993).
60. Heath SK, Koenig JQ, Morgan MS, Checkoway H, Hanley QS, Rebolledo V. Effects of sulfur dioxide exposure on African-American and Caucasian asthmatics. Environ Res 66:1–11 (1994).
61. Mortimer KM, Tager IB, Dockery DW, Neas LM, Redline S. The effect of ozone on inner-city children with asthma: identification of susceptible subgroups. Am J Respir Crit Care Med 162:1–8 (2000).
62. Ostro BD, Lipsett MJ, Mann JK, Braxton-Owens H, White MC. Air pollution and asthma exacerbations among African-American children in Los Angeles. Inhal Toxicol 7:711–722 (1995).
63. Rusznak C, Devalia JL, Davies RJ. The impact of pollution on allergic disease. Allergy 49:21–27 (1994).
64. Delfino RJ, Coate BD, Zeiger RS, Seltzer JM, Street DH, Koutrakis P. Daily asthma severity in relation to personal ozone exposure and outdoor fungal spores. Am J Respir Crit Care Med 154:633–641 (1996).
65. Delfino RJ, Zeiger RS, Seltzer JM, Street DH, Matteucci RM, Anderson PR, Koutrakis P. The effect of outdoor fungal spore concentrations on daily asthma severity. Environ Health Perspect 105:622–635 (1997).
66. Jorres R, Nowak D, Magnussen H. The effect of ozone exposure on allergen responsiveness in subjects with asthma or rhinitis. Am J Respir Crit Care Med 153:56–64 (1996).
67. Tunnicliffe WS, Burge PS, Ayres JG. Effect of domestic concentrations of nitrogen dioxide on airway responses to inhaled allergen in asthmatic patients. Lancet 344:1733–1736 (1994).
68. Peterson B, Saxon A. Global increases in allergic respiratory disease: the possible role of diesel exhaust particles. Ann Allergy Asthma Immunol 77:263–270 (1997).
69. Diaz-Sanchez D. The role of diesel exhaust particles and their associated polyaromatic hydrocarbons in the induction of allergic airway disease. Allergy 52:52–56 (1997).
70. Wiesch DG, Samet JM. Epidemiology and natural history of asthma. In: Allergy: Principles and Practice, Volume II (Middleton E Jr, Reed CE, Ellis EF, Adkinson NF Jr, Yunginger JW, Busse WW, eds). St. Louis, MO:Mosby—Year Book, Inc.,1998;799–815.
71. Kattan M, Mitchell H, Eggleston P, Gergen P, Crain E, Redline S, Weiss K, Evans R III, Kaslow R, Kercsmar C, et al. Characteristics of inner-city children with asthma: the National Cooperative Inner-City Asthma Study. Pediatr Pulmonol 24:253–262 (1997).
72. Zeiger RS, Dawson C, Weiss S. Relationships between duration of asthma and asthma severity among children in the Childhood Asthma Management Program. J Allergy Clin Immunol 103:376–387 (1999).
73. Wallace D, Wallace R. A Plague on Your Houses: How New York Was Burned Down and National Public Health Crumbled. New York:Verso Books, 2000.
74. Perera FP, Jedrychowski W, Rauh V, Whyatt RM. Molecular epidemiologic research on the effects of environmental pollutants on the fetus. Environ Health Perspect 107:451–460 (1999).
75. WHO. Principles for Evaluating Health Risks From Chemicals During Infancy and Early Childhood: The Need for a Special Approach. Environmental Health Criteria 59. Geneva:World Health Organization, 1986.
76. Calabrese EJ. Age and Susceptibility to Toxic Substances. New York:Wiley and Sons, 1986.
77. Dejmek J, Selevan SG, Benes I, Solansky I, Sram RJ. Fetal growth and maternal exposure to particulate matter during pregnancy. Environ Health Perspect 107:475–480 (1999).
78. Svanes C, Omenaas E, Heuch JM, Irgens LM, Gulsvik A. Birth characteristics and asthma symptoms in young adults: results from a population-based cohort study in Norway. Eur Respir J 12:1366–1370 (1998).
79. O'Campo P, Xue X, Wang MC, Caughy M. Neighborhood risk factors for low birthweight in Baltimore: a multilevel analysis. Am J Public Health 87:1113–1118 (1997).
80. Orr ST, James SA, Miller CA, Barakat B, Daikoku N, Pupkin M, Engstrom K, Huggins G. Psychosocial stressors and low birthweight in an urban population. Am J Prev Med 12:459–466 (1996).
81. Sunyer J, Anto JM, Sabria J, Rodrigo MJ, Roca J, Morell F, Rodriguez-Roisin R, Codina R. Risk factors of soybean epidemic asthma. The role of smoking and atopy. Am Rev Respir Dis 145:1098–1102 (1992).
82. Leikauf GD, Prows DR, Shertzer HG, Carrigan PE, Zhao Q, Borchers MT, Ormerod E, Simpson LG, Weaver TE, Clark JC, et al. Asthma and acute lung injury: transgenic models and genetic determinants. In: Relationships Between Respiratory Disease and Exposure to Air Pollution (Mohr U, Dungworth DL, Brain JD, Driscoll KE, Grafstrom RC, eds). Washington, DC:ILSI Press,1998;45–58.
83. Leikauf GD, McDowell SA, Gammon K, Wesselkamper SC, Bachurski CJ, Puga A, Wiest JS, Leikauf JE, Prows PR. Functional genomics of particle-induced lung injury. Inhal Toxicol 12:59–73 (2000).
84. Smith CA, Harrison DJ. Association between polymorphism in gene for microsomal epoxide hydrolase and susceptibility to emphysema. Lancet 350:630–633 (1997).
85. Ishii T, Matsuse T, Teramoto S, Matsui H, Hosoi T, Fukuchi Y, Ouchi Y. Association between alpha-1-antichymotrypsin polymorphism and susceptibility to chronic obstructive pulmonary disease. Eur J Clin Invest 30:543–548 (2000).
86. Wiesch DG, Meyers DA, Bleecker ER. Genetics of asthma. J Allergy Clin Immunol 104:895–901 (1999).
87. Holgate ST. Genetic and environmental interaction in allergy and asthma. J Allergy Clin Immunol 104:1139–1146 (1999).
88. Barnes PJ. Molecular genetics of chronic obstructive pulmonary disease. Thorax 54:245–252 (1999).
89. Karjalainen A, Kurppa K, Virtanen S, Keskinen H, Nordman H. Incidence of occupational asthma by occupation and industry in Finland. Am J Ind Med 37:451–458 (2000).
90. Toren K, Balder B, Brisman J, Lindholm N, Lowhagen O, Palmqvist M, Tunsater A. The risk of asthma in relation to occupational exposures: a case-control study from a Swedish city. Eur Respir J 13:496–501 (1999).
91. Kogevinas M, Anto JM, Sunyer J, Tobias A, Kromhout H, Burney P. Occupational asthma in Europe and other industrialised areas: a population-based study. European Community Respiratory Health Survey Study Group. Lancet 353:1750–1754 (1999).
92. Duran-Tauleria E, Rona RJ. Geographical and socioeconomic variation in the prevalence of asthma symptoms in English and Scottish children. Thorax 54:476–481 (1999).
93. Sandberg S, Paton JY, Ahola S, McCann DC, McGuinness D, Hillary CR, Oja H. The role of acute and chronic stress in asthma attacks in children. Lancet 356:982–987 (2000).
94. McCord C, Freeman HP. Excess mortality in Harlem. N Engl J Med 322:173–177 (1990).
95. Weil CM, Wade SL, Bauman LJ, Lynn H, Mitchell H, Lavigne J. The relationship between psychosocial factors and asthma morbidity in inner-city children with asthma. Pediatrics 104:1274–1280 (1999).
96. Cohen S, Doyle WJ, Skoner DP. Psychological stress, cytokine production, and severity of upper respiratory illness. Psychosom Med 61:175–180 (1999).
97. Coe CL, Erickson CM. Stress decreases lymphocyte cytolytic activity in the young monkey even after blockade of steroid and opiate hormone receptors. Dev Psychobiol 30:1–10 (1997).
98. Esterling BA, Kiecolt-Glaser JK, Glaser R. Psychosocial modulation of cytokine-induced natural killer cell activity in older adults. Psychosom Med 58:264–272 (1996).
99. Schmidt DD, Schmidt PM, Crabtree BF, Hyun J, Anderson P, Smith C. The temporal relationship of psychosocial stress to cellular immunity and herpes labialis recurrences. Fam Med 23:594–599 (1991).
100. Martin-Romero C, Santos-Alvarez J, Goberna R, Sanchez-Margalet V. Human leptin enhances activation and proliferation of human circulating T lymphocytes. Cell Immunol 199:15–24 (2000).
101. Lord GM, Matarese G, Howard JK, Baker RJ, Bloom SR, Lechler RI. Leptin modulates the T-cell immune response and reverses starvation-induced immunosuppression. Nature 394:897–901 (1998).
102. Clean Air Act of 1970. Public Law 91-104, 1970.
103. Rosenbaum AS, Axelrad DA, Woodruff TJ, Wei YH, Ligocki MP, Cohen JP. National estimates of outdoor air toxics concentrations. J Air Waste Manag Assoc 49:1138–1152 (1999).
104. Woodruff TJ, Axelrad DA, Caldwell J, Morello-Frosch R, Rosenbaum A. Public health implications of 1990 air toxics concentrations across the United States. Environ Health Perspect 106:245–251 (1998).
105. South Coast Air Quality Management District. Multiple air toxics exposure study in the South Coast Air Basin (MATES-II). Draft final report. Diamond Bar, CA:South Coast Air Quality Management District, 1999. Available: *http://www.aqmd.gov/mateii/exec_summary/executsummary.htm* [cited 21 July 2000].
106. Akland GG, Hartwell TD, Johnson TR, Whitmore RW. Measuring human exposure to carbon monoxide in Washington, D.C., and Denver, Colorado, during the winter of 1982–1983. Environ Sci Technol 19:911–918 (1985).
107. Buckley TJ, Payne-Sturges DC, Weaver VM. Unpublished data.
108. Litt J. An Evaluation of Public Health and Environmental Aspects of Brownfields in Baltimore City [PhD Thesis]. Baltimore, MD:Johns Hopkins University, 2000.
109. Samet JM, Schwab M, Geyh A, Buckley TJ, Strickland P. Unpublished data,
110. Chillrud SN, Ross JM, Morris A, Pederson D, Aggarwal M, Ramstrom S, Spengler JD, Kinney PL. Filter reflectance as a tracer for the outdoor contribution to indoor fine particle concentration: results from the New York City TEACH Study. In: Engineering Solutions to Indoor Air Quality Problems: Proceedings of a Symposium, 17-19 July 2000, Raleigh, North Carolina. Pittsburgh, PA:Air and Waste Management Association, 2000:130–139.
111. Weisel CP, Colome S, Morandi M, Stock T, Spektor D, Turpin B, Zhang J. The RIOPA (Relationship Among Indoor, Outdoor and Personal Air) Study [Abstract]. Epidemiology 10:S92 (1999).
112. Lioy PJ, Freeman NC, Wainman T, Stern AH, Boesch R, Howell T, Shupack SI. Microenvironmental analysis of residential exposure to chromium-laden wastes in and around New Jersey homes. Risk Anal 12:287–299 (1992).
113. Schwab M, Colome SD, Spengler JD, Ryan PB, Billick IH. Activity patterns applied to pollutant exposure assessment: data from a personal monitoring study in Los Angeles. Toxicol Ind Health 6:517–532 (1990).
114. Schwab M. The influence of daily activity patterns on differential exposure to carbon monoxide among social groups. In: Proceedings of the Research Planning Conference on Human Activity Patterns, 8–10 May 1988 (Starks TH, ed). EPA/600/4-89/004. Las Vegas, NV:U.S. Environmental Protection Agency, Office of Research and Development, 1989;18.1–18.21.
115. Wallace LA. Appendix A. Sources of exposure to volatile organic chemicals: An analysis of personal exposures in the TEAM study. EPA/600/6-87/002a. Washington, DC:U.S. Environmental Protection Agency, Office of Research and Development, 1987.
116. Schwab M. An examination of the intra-SMSA distribution of carbon monoxide exposure. J Air Waste Manag Assoc 40:331–336 (1990).
117. Spengler J, Schwab M, Ryan PB, Colome S, Wilson AL, Billick I, Becker E. Personal exposure to nitrogen dioxide in the Los Angeles Basin. J Air Waste Manag Assoc 44:39–47 (1994).
118. Brajer V, Hall JV. Recent evidence on the distribution of air pollution effects. Contemp Policy Issues 10:63–71 (1992).
119. Kinney PL, Aggarwal M, Northridge ME, Janssen NA, Shepard P. Airborne concentrations of $PM_{2.5}$ and diesel exhaust particles on Harlem sidewalks: a community-based pilot study. Environ Health Perspect 108:213–218 (2000).
120. Dubowsky SD, Wallace LA, Buckley TJ. The contribution of traffic to indoor concentrations of polycyclic aromatic hydrocarbons. J Expos Anal Environ Epidmiol 9:312–321 (1999).
121. Bollweg G. Personal communication.
122. Dockery DW, Cunningham J, Damokosh AI, Nea LM, Spengler JD, Koutrakis P, Ware JH, Raizenne M, Speizer FE. Health

effects of acid aerosols on North American children: respiratory symptoms. Environ Health Perspect 104:500–505 (1996).

123. Mitchell H, Senturia Y, Gergen P, Baker D, Joseph C, McNiff-Mortimer K, Wedner HJ, Crain E, Eggleston P, Evans R III, et al. Design and methods of the National Cooperative Inner-City Asthma Study. Pediatr Pulmonol 24:237–252 (1997).
124. Drossman DA. Presidential address: gastrointestinal illness and the biopsychosocial model. Psychosom Med 60:258–267 (1998).
125. Miller BD, Wood BL. Psychophysiologic reactivity in asthmatic children: a cholinergically mediated confluence of pathways. J Am Acad Child Adolesc Psychiatry 33:1236–1245 (1994).
126. Lobel M, Dunkel-Schetter C, Scrimshaw SC. Prenatal maternal stress and prematurity: a prospective study of socioeconomically disadvantaged women. Health Psychol 11:32–40 (1992).
127. Levenstein S. The very model of a modern etiology: a biopsychosocial view of peptic ulcer. Psychosom Med 62:176–185 (2000).
128. Clark R, Anderson NB, Clark VR, Williams DR. Racism as a stressor for African Americans. A biopsychosocial model. Am Psychol 54:805–816 (1999).
129. U.S. EPA, Office of Air Quality and Standards, CICA. Border Air Quality Information. Available: *http://www.epa.gov/ttn/catc/cica/airq_e.html* [cited 21 January 2000].
130. U.S. EPA, National Center for Environmental Research. Environmental Monitoring for Public Access and Community Tracking (EMPACT). EMPACT metro grants announcement. Appendix A: EMPACT metropolitan areas. Available: *http://es.epa.gov/ncerqa/rfa/2000empact.html* [cited 15 December 1999].
131. Bansal S, Bacon D, Davis S. Holding Our Breath: the Struggle for Environmental Justice in Southeast Los Angeles. Oakland, CA:Communities for a Better Environment, 1998.
132. Zanobetti A, Schwartz J, Gold D. Are there sensitive subgroups for the effects of airborne particles? Environ Health Perspect 108:841–845 (2000).
133. Pew Environmental Health Commission. Attack Asthma: Why America Needs a Public Health Defense System to Battle Environmental Threats. Baltimore, MD:Pew Environmental Health Commission, 2000. Available: *http://pewenvirohealth.jhsph.edu/html/reports/pehcasthmareport.pdf*.
134. Smyth JM, Soefer MH, Hurewitz A, Kliment A, Stone AA. Daily psychosocial factors predict levels and diurnal cycles of asthma symptomatology and peak flow. J Behav Med 22:179–193 (1999).
135. Malveaux FJ, Fletcher-Vincent SA. Environmental risk factors of childhood asthma in urban centers. Environ Health Perspect 103 (suppl 6):59–62 (1995).
136. Wright RJ, Rodriguez M, Cohen S. Review of psychosocial stress and asthma: an integrated biopsychosocial approach. Thorax 53:1066–1074 (1998).
137. Asch P, Seneca JJ. Some evidence on the distribution of air quality. Land Economics 54:278–297 (1978).
138. Gianessi LP, Peskin HM, Wolfe E. The distributional effects of uniform air pollution policy in the U.S. Q J Econ 93:281–301 (1979).
139. Clean Air Act Amendments of 1990, Section 108. Public Law 101–549, 1990.
140. Clean Air Act Amendments of 1990, Section 109. Public Law 101–549, 1990.
141. Clean Air Act Amendments of 1990, Section 112. Public Law 101–549, 1990.
142. Clean Air Act Amendments of 1990. Public Law 101–549, 1990.
143. U.S. EPA. National Air Toxics Program: the Integrated Urban Strategy. Fed Reg 64:38705–38740 (1999).
144. U.S. EPA, Office of Air and Radiation. Draft Economic Incentive Program Guidance. EPA-452/D-99-001. Washington, DC:U.S. Environmental Protection Agency, 1999.
145. Title 40. Protection of Environment. Chapter I: Environmental Protection Agency—Part 58: Ambient Air Quality Surveillance. Appendix D: Network design for state and local air monitoring stations (SLAMS), national air monitoring stations (NAMS), and photochemical assessment monitoring stations (PAMS). 40 CFR 58. Washintgon, DC:U.S. Government printing Office, 2000.
146. Coequyt J, Ross Z, Walker B, Wiles R. Above the Law: How California's Major Air Polluters Get Away With It. Washington, DC:Environmental Working Group, 1999. Available: *http://www.ewg.org/pub/home/reports/ca_abovethelaw/abovethelawca.html* [cited 21 July 2000].
147. Morello-Frosch RA. Environmental Justice and California's "Riskscape": The Distribution of Air Toxics and Associated Cancer and Non-cancer Health Risks Among Diverse Communities [PhD Thesis]. Berkeley, CA:University of California, 1997.
148. Northridge ME, Yankura J, Kinney PL, Santella RM, Shepard P, Riojas Y, Aggarwal M, Strickland P. Diesel exhaust exposure among adolescents in Harlem: a community-driven study. Am J Public Health 89:998–1002 (1999).
149. Wernette DR, Nieves LA. Minorities and Air Auality Non-attainment Areas: a Preliminary Geo-demographic Analysis. ANL-CP-75440. Argonne, IL:Argonne National Laboratory, 1992.
150. Wernette DR. Minorities, Age Groups, and Air Quality Non-attainment Areas: an Analysis of Regional and Urban Patterns. Argonne, IL:Environmental Assessment and Information Sciences Division, Argonne National Laboratory, 1992.
151. Baumol WJ, Oates WE. The Theory of Environmental Policy. New York:Cambridge University Press, 1988.
152. Drury R. Personal communication.
153. Drury RT, Belliveau ME, Kuhn JS, Bansal S. Pollution trading and environmental injustice: Los Angeles' failed experiment in air quality policy. Duke Environ Law Policy Forum 9:231–289 (1999).
154. U.S. EPA. Clean Air Act Federal Advisory Committee, Minutes, Spring 2000. Washington, DC:U.S. Environmental Protection Agency, 2000.
155. Mann E. A New Vision for Urban Transportation: The Bus Riders Union Makes History at the Intersection of Mass Transit, Civil Rights, and the Environment. Los Angeles, CA:Strategy Center Publications, 1996.
156. Freeman AM III. The Incidence of the Costs of Controlling Automotive Air Pollution. Cambridge, MA:Ballinger Publishing, 1977.
157. Cameron MW, Environmental Defense Fund. Efficiency and Fairness on the Road: Strategies for Unsnarling Traffic in Southern California. Oakland, CA:Environmental Defense Fund, 1994.
158. Joshi P. Producers of furniture eye the future; environment: AQMD, Edison show them less-polluting products that could help keep them in the area. Los Angeles Times 28 August 1993:D1.
159. Civil Rights Act of 1964, Title VI. Public Law 88–352, 1964.
160. U.S. EPA. Title VI Violations Filed with EPA. Available: *http://www.epa.gov/civilrights/docs/t6csoct2000.pdf* [cited 4 October 2000].
161. National Environmental Justice Advisory Council. The Model Plan for Public Participation. Washington, DC:National Environmental Justice Advisory Council, 1996. Available: *http://es.epa.gov/oeca/oej/nejac/pdf/modelbk.pdf*
162. Shusterman D. Critical review: the health significance of environmental odor pollution. Arch Environ Health 47:76–87 (1992).
163. U.S. EPA. Regulatory Options in the Control of Odors. EPA 450/5-80-003. Research Triangle Park, NC:U.S. Environmental Protection Agency, 1980.
164. Morag-Levine N. Chasing the Wind: Reactive Law, Environmental Equity and Localized Air Pollution Regulation [PhD Thesis]. Berkeley, CA:University of California, 1995.