

Health, Wealth, and Air Pollution: Advancing Theory and Methods

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The effects of both ambient air pollution and socioeconomic position (SEP) on health are well documented. A limited number of recent studies suggest that SEP may itself play a role in the epidemiology of disease and death associated with exposure to air pollution. Together with evidence that poor and working-class communities are often more exposed to air pollution, these studies have stimulated discussion among scientists, policy makers, and the public about the differential distribution of the health impacts from air pollution. Science and public policy would benefit from additional research that integrates the theory and practice from both air pollution and social epidemiologies to gain a better understanding of this issue. In this article we aim to promote such research by introducing readers to methodologic and conceptual approaches in the fields of air pollution and social epidemiology; by proposing theories and hypotheses about how air pollution and socioeconomic factors may interact to influence health, drawing on studies conducted worldwide; by discussing methodologic issues in the design and analysis of studies to determine whether health effects of exposure to ambient air pollution are modified by SEP; and by proposing specific steps that will advance knowledge in this field, fill information gaps, and apply research results to improve public health in collaboration with affected communities. *Key words:* air pollution, environmental justice, epidemiology, exposure assessment, socioeconomic factors. *Environ Health Perspect* 111:1861–1870 (2003). doi:10.1289/ehp.6334 available via <http://dx.doi.org/> [Online 2 September 2003]

Numerous epidemiologic studies have found associations between socioeconomic position (SEP) and health, with gradients observed for outcomes including mortality, infectious and chronic diseases, and psychiatric disorders (Haan et al. 1987; Krieger et al. 1997; Marmot 2001). Ambient air pollution has also been linked with a broad range of health effects, including mortality and morbidity from heart and lung disease, impaired lung function, and lung cancer (Brunekreef and Holgate 2002). Because of the importance of SEP as a determinant of health, and because air pollution exposure can vary according to socioeconomic circumstances, SEP has been included as a potential confounder (Bobak and Leon 1999; Dockery et al. 1993) and an effect modifier (Gouveia and Fletcher 2000; Gwynn and Thurston 2001; Zanobetti and Schwartz 2000) in epidemiologic studies. In this article, we describe current knowledge, hypotheses and theories, methodologic approaches, and research needs related to the effects and interactions of air pollution and socioeconomic conditions on human health and well-being. We structure the discussion with an interpretative framework based on three related propositions. First, groups with lower SEP may receive higher exposure to air pollution. Second, because lower-SEP groups already experience compromised health status due to material deprivation and psychosocial stress, they may be more susceptible to the health effects of air pollution. Third, because of the combination

of greater exposure and susceptibility, these groups are likely to suffer greater health effects from air pollution exposure.

International organizations have identified both air pollution and poverty as priority areas for public health intervention (Ezzati et al. 2002), and the intersection of these pressing problems requires strategic attention from researchers and policy makers. These concerns affect the lives of many people—approximately 1 billion people live in poverty (World Bank 2002). An estimated 1.5 billion people currently live in polluted urban areas, and 65% of the world's population is projected to live in cities by 2025 [World Health Organization (WHO) 2000]. More than 40% of the world's children are estimated to live in polluted cities of the developing world (Davis and Saldiva 1999). At the same time, evidence indicates that air pollution is an even more serious health concern than previously thought, with associations seen with reduced life expectancy, increased daily mortality and hospital admissions, birth outcomes, and asthma (Brunekreef and Holgate 2002; Clancy et al. 2002; McConnell et al. 2002; Ritz et al. 2002). These effects appear to be without thresholds (Schwartz and Zanobetti 2000), suggesting large attributable risks in both the developing and developed world (Working Group on Public Health and Fossil-Fuel Combustion 1997).

Experts attending a recent international workshop recommended further research and

collaboration on issues including vulnerability to air pollution exposure (Bell et al. 2002). The U.S. National Research Council has called the identification of subpopulations at elevated risk a priority research concern (National Research Council 1998), and persons with low SEP are one potential subpopulation. To support and better focus public health action in alleviating economic disparities and reducing air pollution, reliable estimates of the health effects of both, as well as potential confounding or effect modification one may exert on the other, are required.

Several steps to improving understanding of the interaction between socioeconomic disparities and air pollution exposures have already been taken. Research and initiatives to reduce inequities in pollution exposure and consequent health effects have increased in the United States since the early 1990s, partly as a result of the environmental justice movement (Bullard and Wright 1993). Community partnerships in research and decision making have been recommended to improve the relevance of scientific results and enhance understanding of the varied concerns of those affected (Israel et al. 1998). Accumulated environmental exposure from multiple sources (noise, water quality, crowding, housing quality, and neighborhood conditions) has been

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offered as one potential explanation for observed health gradients by SEP (Evans and Kantrowitz 2002). Although we focus on ambient air pollution, we believe that a thorough examination of one class of pollution may illuminate the studies of how other contaminants may affect and be affected by SEP.

Despite international interest in the effects of socioeconomic disparities and ambient air pollution on health, and growing awareness of the importance of considering both in epidemiologic research, few studies have looked carefully at how these factors interact with one another. In this review, we introduce readers to underlying approaches in both fields, articulate hypotheses of how air pollution and socioeconomic factors could interact to influence health, and recommend research methods for investigating these hypotheses.

A Primer on the Health Effects of SEP and Air Pollution

An analysis of the health effects of SEP and air pollution can and should consider all levels of the potential causal chain, from molecular mechanisms, to individual risk factors (including personal behaviors), to contextual factors (including economic and social policy). This holistic view has been articulated in recent publications (Kaplan 1998; McMichael 1998; Susser 1998). We will familiarize researchers with either social determinants of health or air pollution health effects, review available evidence on effect modification of air pollution health effects by SEP, and draw the literatures together under the hypotheses that explain why SEP may modify the health effects of air pollution.

SEP and health. There are four widely agreed upon facts and principles about the relationship between SEP and health. First, the relationship between SEP and poor health is not confined to poor people alone. Although it is clear that the highest risks of premature mortality and morbidity are concentrated among the poor, studies have also repeatedly demonstrated the existence of a graded relationship between low SEP (whether measured by income or educational attainment) and worse health outcomes. At each step of the socioeconomic hierarchy, individuals tend to have better health compared with those immediately below them. This gradient extends well into the range of incomes that can be termed “middle class.”

Second, SEP can be conceptualized and measured at both the individual level and the area level (e.g., neighborhoods). Evidence suggests that each level exerts an independent influence on an individual's chances of health. In other words, an individual with the same level of income or educational attainment could experience different chances of health depending upon the SEP of his or her neighbors.

Area-level SEP may pattern an individual's access to opportunities for good health. Examples of such patterning include differential access to the service environment (e.g., health clinics, supermarkets, sanitation or waste disposal), the physical environment (e.g., traffic burden, crowding, clean water for drinking or bathing), and the social environment (crime rate, social cohesion, and vandalized public areas) (Kawachi and Berkman 2003).

Third, in addition to the dimension of context or place, the dimension of time is important for conceptualizing and measuring SEP effects on health. SEP rarely remains static across the life course, and the measurement of income, for example, at a single point in time is unlikely to capture the dynamic as well as the cumulative effects of SEP on health. Childhood socioeconomic circumstances are now believed to exert an effect on adult health, independently of SEP attained in adulthood (Davey Smith et al. 2001). Income dynamics (e.g., downward social mobility, or accumulated spells of poverty) have been shown to predict mortality and other health outcomes (McDonough et al. 1997). We now understand that wealth (or some other measure of permanent income) is much more strongly related to health than is a single measure of income (Daly et al. 2002). A further important aspect of time relates to the history of a geographical location; migration in and out of areas will affect the health status profiles, behaviors, and possibly accumulated exposures of local population groups and should be accounted for in associative analyses.

Finally, social epidemiologists now stress the importance of distinguishing the concept of SEP from race. This point is particularly pertinent to the United States, where official statistics often conflate racial disparities in health with socioeconomic disparities. The two are not the same, despite the fact that racial minorities in the United States are overrepresented among lower-SEP groups. Race and SEP have been shown to have independent effects on health, and interpretation of studies using these variables should acknowledge that one variable may be a poor proxy of the other.

Air pollution and health. This section covers salient aspects of the associations between air pollution and health, focusing on three central topics: sources and emissions, concentrations and exposures, and health effects assessment.

Commonly studied pollutants include primary and secondary airborne particles and gases, including ozone, carbon monoxide, sulfur dioxide, and nitrogen oxides. The term “secondary” here refers to compounds (gases or particles) that are not directly emitted into the atmosphere but rather form because of reactions in the atmosphere, often driven by ultraviolet light. These pollutants frequently

form at some distance from the site of emission of their precursor compounds. Because lead has been the subject of a number of reviews and is no longer added to the gasoline in many countries, we do not consider it here, but it remains an important air pollutant in countries where leaded gasoline is still used.

Air pollution health studies assign exposure based on measurements at or near the individual's breathing zone or at ambient monitors intended to represent community exposure. The validity of ambient monitors as a reflection of actual personal exposure for epidemiologic studies varies by the nature of the air pollutant of interest, the epidemiologic study design, and the measurement technology (Janssen et al. 1998; Sarnat et al. 2000). In general, ambient monitoring data are fairly good surrogates for daily fine particle personal exposures but less so for gases such as nitrogen dioxide that display small-area spatial variation. For long-term exposure, it is unknown how well ambient monitors predict personal exposure, but available evidence suggests a potential for exposure measurement error (Liu et al. 1997).

Air pollution epidemiology has addressed several health outcomes, including mortality and morbidity, acute infections, lung cancer, impairments of lung function, hospitalization, chronic respiratory diseases, and reproductive anomalies (Brunekreef and Holgate 2002). Study designs include time-series analyses, in which daily measures of pollution are evaluated with respect to daily counts of morbidity and mortality outcomes; panel studies, wherein a defined group of subjects is followed longitudinally to assess responses to pollution exposure; ecologic studies using group contrasts in mortality and exposure (but no information on individual risk factors); and cohort studies in which associations between long-term exposure to pollution and health outcomes are evaluated within cities or across geographic regions with differing pollutant profiles. Although some contradictory results have emerged, overall significant positive associations between air pollution and various health outcomes have been established (Brunekreef and Holgate 2002). Because some laws—for example, the U.S. Clean Air Act—require that air quality standards protect “sensitive” populations, special attention has been paid to studying susceptibility related to age, disease status, and factors such as smoking status (Clean Air Act 1995).

Evidence on SEP modification of air pollution health effects. Several recent studies have addressed whether SEP modifies the health effects of particulate air pollution. Table 1 summarizes design features of some of these studies, most of which are time-series studies, although one large cohort study

tested effect modification by education. These studies were chosen to illustrate recent research, not comprehensively review it. Several factors evaluated are not direct measures of SEP but rather medical conditions that are differentially distributed by SEP, or race, which can have independent effects on health outcomes. The majority of studies evaluating individual-level characteristics did show effect modification with higher effects

(in general) among those of lower SEP, by race, or in those having medical characteristics associated with lower SEP. Low educational attainment seems to be a particularly consistent indicator of vulnerability in these studies. Those studies using group/county level indicators did not show important effect modification, as a whole; these results may relate to the relatively coarse resolution of these variables.

Although many of the studies reported effect modification via SEP variables, few have explicitly examined why SEP may modify this environmental health effect. In the next section we propose three possible explanations for potential effect modification.

Hypotheses and Theories

We hypothesize that the effects of air pollution exposure on health are differentially distributed

Table 1. Studies of particulate air pollution and socioeconomic conditions.

Reference	Population	Study design	Place/years	Main effect pollutant(s)	Health outcome	Socioeconomic variable	Level ^a /resolution	Key findings
HEI 2000	American Cancer Society (ACS) cohort ($n = \sim 500,000$); Harvard Six Cities Study ($n = 8,111$)	Prospective cohort	USA, 1982–1989 (ACS) 1975–1991 (6 cities)	PM _{2.5}	Mortality	Educational attainment (< high school, high school, > high school)	Individual/person	Greatest effects among least educated; monotonic dose response for all-cause mortality
Pope et al. 2002	ACS ($n = 500,000$)	Prospective cohort	USA, 1982–1998	PM _{2.5}	Mortality	Educational attainment (< high school, high school, > high school)	Individual/person	Greatest effects among least educated; monotonic dose response for all-cause mortality
Ito and Thurston 1996	Residents of Cook County, Illinois	Time series	Chicago, IL, USA 1985–1990	PM ₁₀	Daily mortality	Race (black/white), sex	Individual/person	Greatest effects among black women, then white women, black men, and white men
Gouveia and Fletcher 2000	Residents of São Paulo, Brazil	Time series	Brazil, 1991–1993	PM ₁₀	Daily mortality	Composite index of socioeconomic conditions (from 1991 Census)	Group/district (58 in a city of 9.5 million)	Air pollution effects larger in districts of higher socioeconomic level
Samet et al. 2000	Residents of 20 U.S. cities	Time series pooled data	USA, 1987–1994	PM ₁₀ (O ₃ , CO, SO ₂ , NO ₂)	Daily mortality	Educational attainment, income, (percent poverty, percent nonwhite, and transportation habits; the last three only in HEI NMMAPS)	Group/county	No effect modification by any of the variables considered
Zanobetti and Schwartz 2000	Residents of Chicago, Detroit, Minneapolis–St. Paul, Pittsburgh	Time series pooled data	USA, 1986–1993	PM ₁₀	Daily mortality	Race, sex, educational attainment	Individual/person	Higher effect in women; race and educational attainment were weak modifiers
Cifuentes et al. 1999	Residents of Santiago, Chile	Time series	Chile, 1988–1996	PM _{2.5}	Daily mortality	Educational attainment (elementary, high school, university)	Individual/person	Greatest effects among least educated; monotonic dose response for all-cause mortality
Wojtyniak et al. 2001	Residents of four Polish cities: Cracow, Lodz, Poznan, Wroclaw	Time series	Poland, 1990–1996	Black smoke, NO ₂ , SO ₂	Daily mortality	Educational attainment (elementary or less, secondary, above secondary or university)	Individual/person	Greatest effects among least educated; monotonic dose response for all-cause mortality for black smoke, SO ₂
Gwynn and Thurston 2001	Hospital admissions in New York, New York	Time series	New York, 1988–1990	PM ₁₀ , acidity (H ⁺), O ₃ , sulfate	Respiratory-cause hospital admissions	Non-Hispanic whites, all other races/ethnicities, insured and uninsured	Individual/person	Higher effects among races other than white and uninsured for O ₃ ; less marked differences for other pollutants
Zanobetti et al. 2000a	Hospital admissions in 10 U.S. cities	Time series meta-regression	U.S. cities, 1985–1994; time interval varies by city	PM ₁₀	Hospital admissions, respiratory and cardiovascular	Percent population living in poverty; percent nonwhite population	Group/person	No multiplicative-scale effect modification observed by socioeconomic factors
Zanobetti et al. 2000b	Medicare recipients in Cook County, Illinois	Time series	Cook County, IL, 1985–1994	PM ₁₀	Hospital admissions, respiratory and cardiovascular	Primary: race and sex; secondary: concurrent diagnosis/previous admission	Individual/person	No significant effect modification by sex or race; higher effects among those with respiratory infections/asthma, previous admissions
Norris et al. 2000	Residents of Seattle, Washington < 18 years of age	Time series	Seattle, WA, 1995–1996	PM ₁₀ (CO, NO ₂ , SO ₂)	Asthma ED visits	High vs. low ED-use regions	Group/zip code	No effect modification by ED use rates; significant difference in absolute visits
Linn et al. 2000	Residents of South Coast Air Basin (California)	Time series	Los Angeles, Riverside, San Bernardino, Orange Counties, 1992–1995	PM ₁₀ , CO, NO ₂ , O ₃	Hospital admissions, respiratory and cardiovascular	Sex, ethnicity (white, black, Hispanic, other)	Individual/person	Increased cardiovascular effects in blacks and whites relative to Hispanics and other
Tolbert et al. 2000	Children in Atlanta, Georgia (< 16 years)	Time series	Atlanta, GA, 1993–1995	PM ₁₀ , NO ₂ , O ₃	Asthma ED visits	Race, Medicaid status, sex	Individual/person	No effect modification due to race or Medicaid status
Zanobetti and Schwartz 2001	Medicare recipients in Cook County, Illinois	Time series	Cook County, IL, 1988–1994	PM ₁₀	Hospital admissions, respiratory and cardiovascular	Secondary: diabetic status	Individual/person	Higher cardiovascular hospital admission rates among diabetics vs. nondiabetics

Abbreviations: ED, emergency department; HEI, Health Effects Institute; NMMAPS, National Morbidity, Mortality, and Air Pollution Study; PM_{2.5}, particulate matter < 2.5 µm in aerodynamic diameter; PM₁₀, particulate matter < 10 µm in aerodynamic diameter.

^aGroup vs. individual.

by SEP and that, under most conditions, people in lower SEPs are at greater risk. Additionally, some of the observed disparities in health outcomes by SEP may be explained by air pollution exposure. We base this general hypothesis on three possible routes through which air pollution exposure may result in greater health effects among those in disadvantaged circumstances. These routes are as follows: *a*) air pollution exposure is differentially distributed by SEP; *b*) low SEP may directly increase susceptibility to air pollution-related health consequences; and *c*) some health conditions and traits that cause vulnerability to air pollution are linked to SEP. We recognize that there can be some blurring between categories *b* and *c*, but we feel these warrant separate consideration to underscore different aspects of the pathway from air pollution exposure to health effects.

Exposure differentials related to socioeconomic conditions. The intersection of late-twentieth-century social movements against racism and the degradation of the environment focused attention on issues of environmental justice and environmental equity. Researchers have investigated processes (e.g., land use; political, cultural, and economic structures) leading to unequal exposures (Pijawka et al. 1998). Outcome-focused research evaluates whether unequal exposure exists, without considering the processes that might have led to disparities. The scientific community, often working directly with disadvantaged communities, has documented disproportionate exposures to some pollutants in communities characterized by low SEP and/or “minority” racial composition. Explanations range from housing market dynamics (Been 1993) to systemic racism and class bias in land use decisions that push noxious facilities away from wealthy neighborhoods toward poor ones with more racial minorities (Pulido 2000). Regardless of racial or social composition, high land costs that discourage purchase for industrial uses in more affluent areas are likely to affect distribution of pollutant sources, over and above the effects of political and social influence and discrimination. Internationally, politically weaker and usually poorer nations may become repositories for pollution or waste generated in wealthier countries, or polluting facilities may be sited there because of lower labor costs or less stringent environmental regulations. In keeping with the health effects focus of this article, we emphasize studies that evaluate outcomes of unequal pollution exposure distribution, not necessarily those that seek to describe the processes leading to those results. A common hypothesis of such studies is that socially disadvantaged groups suffer greater exposures that explain, in part, persistent inequalities in health observed along social and racial

gradients (Evans and Kantrowitz 2002; Sexton and Adgate 1999).

Heterogeneity of exposure over space and thus the potential for inequity in exposure varies markedly by pollutant type. Fine particles are distributed fairly homogeneously over large urban areas (Burton et al. 1996; Suh et al. 1995), due mostly to the contribution of small, long-range transport particles. Thus, central air pollution monitors can be good surrogates for personal exposure, despite some variation in people's activities. Ultrafine particles can, however, have elevated concentrations (from traffic sources) adjacent to roadways (Buckeridge et al. 2002; Zhu et al. 2002). Significant spatial variation has also been seen for diesel-related pollutants, as indicated by elemental carbon and polycyclic aromatic hydrocarbon levels (Hitchins et al. 2000; Kinney et al. 2000; Nielsen 1996). Gaseous traffic-related pollutants such as NO₂ and CO display variations in concentration across small areas (Hewitt 1991; Ritz et al. 2002) and probably cluster close to emission sources on roadways (Briggs et al. 1997; Hoek et al. 2001; Rijnders et al. 2001). O₃ can be lower closer to areas with dense traffic because of scavenging by NO₂ (Godish 1991) but generally has fairly homogeneous distributions over large areas.

Reviews of the empirical findings on ambient air pollution exposure and socioeconomic level support the idea that disadvantaged groups are more highly exposed to some pollutants (Institute of Medicine 1999; Sexton et al. 1993). In some cases, the evidence is indirect. A number of studies have not been pollutant specific but have shown differences in health impacts as a function of proximity to a roadway (Brunekreef et al. 1997; English et al. 1999; Weiland et al. 1994). If proximity to traffic depresses property values, as suggested by the hedonic pricing literature (Graves 1988), then it is likely that the lower dwelling values will attract residents of lower SEP (Been 1993). Thus, higher ambient exposure is experienced by relatively disadvantaged groups living near roadways. Over larger areas, major roadways may also be routed through lower-income areas because of reduced political and economic power of these residents to oppose such siting. Indeed, lower values of dwellings were strongly associated with higher particle levels in one Canadian study (Jerrett et al. 2001). However, urban development may cause secondary roads to become busier, putting relatively expensive housing stock in the midst of high traffic volume.

European and Scandinavian studies show differential personal exposures to particles and traffic pollutants by occupation and education (Rotko et al. 2000, 2002). Studies in Helsinki, Finland; Mexico City, Mexico; Los Angeles, California; and the U.S. Great Lakes region

have shown differences in exposure to gaseous pollutants by occupation, education, minority status, and income (Korc 1996; Pellizzari et al. 1999; Romieu et al. 1999; Rotko et al. 2001). Although these studies suggested that people of lower SEP have higher exposure, O₃ exposure can be substantially higher among wealthier people who live farther from traffic sources. For example, in southern Mexico City, where more wealthy people reside, O₃ levels are substantially higher than in the poorer northern part of the city (Castillejos et al. 1995). Interpretation of studies that use personal exposure measures should take into account possible selection bias for participants. In Basel, Switzerland, and Helsinki, participants were more likely to be female, older, and better educated and to live in areas with lower traffic volume (Oglesby et al. 2000).

Many studies on exposure differentials rely on proximity to sources or ambient monitoring networks that are limited in number and location. Other studies have collected specific monitoring at additional locations, particularly for NO₂, and modeled exposure over finer geographic levels using data on population density, traffic density, location of residence, and so forth (Gehring et al. 2002; Hoek et al. 2001; Pikhart et al. 2001). One effort in Stockholm, Sweden, used 11,000 residential addresses to estimate individual cumulative exposure to air pollutants from traffic and home heating over several decades, using reconstructed emissions data and dispersion modeling (Bellander et al. 2001). This process yielded wide ranges of exposure for the 11,000 individuals. Although few of these studies have focused on differential exposure by SEP, methods for deriving individual-specific exposure data could provide a useful basis for examining modification of chronic pollution exposure effects by socioeconomic factors, both individual and contextual.

The exposure assessment literature describes several techniques, from more advanced exposure modeling to use of geographic information systems (GIS) and residential addresses, that improve our ability to evaluate whether gradients in exposure exist and are associated with socioeconomic gradients. Studies that involve more complex exposure modeling should consider type of pollutant and the pollutants' physicochemical behavior in the environment, in addition to residential settlement patterns, time-activity patterns, and the behavior of the subjects whose exposure is being estimated. Because human settlement patterns and behaviors as well as pollutant sources and composition differ across local, national, and international scales, studies examining exposure in a variety of geographic regions are desirable. Many other methodologic issues can influence the assessment of inequity in exposure, but these

have been the subject of recent reviews (Bowen 2002; Maantay 2001) and therefore are not included here.

Susceptibility directly related to social position. Susceptibility has been classified as encompassing “intrinsic” factors that include age, sex, genetics, and ethnicity or race, and “acquired,” which include factors such as chronic medical conditions, health care access, nutrition, fitness, other pollutant exposures, and drug and alcohol use (Sexton 1997). The boundaries between susceptibility acquired because of some aspect of social position and intrinsic susceptibility coincident with it are not clearly defined, and timing of a person’s socioeconomic experience can play an important role. Susceptibility may result from a whole cascade of events. For example, diagnosis with a chronic lung or heart condition could result in reduced income due to job change or loss, which in turn could initiate a cascade of damaging coping behaviors (increased smoking, drinking) that might lead to further deterioration of health.

People in lower socioeconomic circumstances may be more susceptible to air pollution for reasons directly related to their relative disadvantage and psychosocial stress. For example, they may lack access to grocery stores that sell fresh fruits and vegetables (Morland et al. 2002) or the income to buy them, resulting in reduced intake of antioxidant vitamins that can protect against adverse consequences of air pollution exposure (Romieu et al. 1998). Another possibility is reduced access to medical care, so poor people may not have the appropriate prescription for a respiratory condition such as asthma. Medication can alleviate symptoms aggravated by pollution exposure, and more consistent use of corticosteroids lowers baseline inflammation, potentially lowering responsiveness to proinflammatory pollutants. An additional hypothesis is that psychosocial stress and violence, which can be higher among those of lower SEP, can increase susceptibility (Wright et al. 1998; Wright and Steinbach 2001).

Characteristics of neighborhoods can affect susceptibility. In four U.S. communities, residence in a disadvantaged neighborhood was associated with coronary heart disease (CHD) incidence, even after controlling for established CHD risk factors and personal income, education, and occupation (Diez Roux et al. 2001). With current emphasis on cardiac effects of air pollution exposure, this finding is particularly relevant to the study of the air pollution and socioeconomic interaction. Because lower-income people are more likely to live closer to roadways, there is also evidence that increased traffic density has been associated with lack of neighborhood communication and collaboration

(thereby reducing available social networks) (Appleyard 1981).

Another potential mechanism of susceptibility directly related to social position is coexposure to other pollutants, including indoor pollutants. A person with a relatively high dose of other pollutants may be “weakened” and less able to withstand the additional insult of ambient air pollution. People with less wealth are more likely to be employed in dirtier occupations (Sexton et al. 1993) and in developing countries, they may also be more likely to be exposed to pollutants indoors from heating and cooking (Smith et al. 2000). Workers in the coke oven and farm industries and children who are in the workforce may suffer increased susceptibility due to cumulative lifetime dose (Rios et al. 1993). Workers in blue-collar occupations may also be more exposed to environmental tobacco smoke than are white-collar workers in cases where regulations limiting indoor smoking in the workplace are not applied consistently. Housing stock in poorer communities with high rates of crowding can have higher levels of certain allergens as well as other risk factors for asthma sensitization and exacerbation (Krieger et al. 2000; Leaderer et al. 2002). These differing allergen profiles may affect whether individuals sensitized to certain allergen burdens will be more responsive to air pollution exposure, and lower-income households may have difficulty obtaining follow-up asthma care that could reduce the severity of responses to air pollution (Kattan et al. 1997).

Although much research on susceptible subpopulations in air pollution epidemiology focuses on traits such as preexisting disease, age, and sex, evidence is growing that exposure to particular socioeconomic conditions can affect susceptibility through mechanisms (psychological, nutritional, etc.) that have not been widely studied in air pollution epidemiology.

Susceptibility from predisposing health conditions, behaviors, or traits. Several studies have shown that people with diabetes are more sensitive to the health effects of air pollution than is the general population (Bateson and Schwartz. In press; Goldberg et al. 2001; Zanobetti and Schwartz 2002). In the United States, diabetes is more common among the elderly, non-Hispanic blacks, and Mexican Americans and among people living in or near a central city. U.S. residents with non-insulin-dependent diabetes mellitus, or type 2 diabetes, usually have less education, lower income, and higher unemployment rates, adjusting for age, than do nondiabetics (Cowie and Eberhardt 1995). In Mexico, type 2 diabetes was more prevalent among low-income individuals, and in the United Kingdom, early childhood deprivation is a risk factor (Ekoe et al. 2001). Thus, diabetes can be associated with several of the indicators of lower SEP, as well as with more advanced

age, which also contributes to vulnerability. Even precursors to diabetes are differentially distributed by social level. Among civil service workers in the United Kingdom, risk factors for diabetes and coronary disease were higher among those employed in lower-paid occupations, independent of health-related behaviors (Brunner et al. 1997).

Mechanisms hypothesized to contribute to the extra sensitivity of diabetics to air pollution include their lower heart rate variability and higher levels of inflammatory markers in their blood; these factors have also been linked to vulnerability to air pollution in other studies (Gold et al. 2000; Peters et al. 2001). More prevalent among diabetics, obesity is a condition that increases with age and is associated with increased systemic inflammation, including markers of cardiovascular risk (Visscher and Seidell 2001; Visser et al. 1999).

Both diabetes and asthma are differentially distributed by socioeconomic level in international comparative studies. In 12 European countries, the prevalence of asthma and diabetes was higher in countries with lower gross national product (Bach 2002). Other international studies show, in general, much higher asthma prevalence in more industrialized countries, although prevalence is increasing overall (Pearce et al. 1998). Lower asthma prevalence has been seen in countries closer to the equator, compared with higher-latitude countries (Bach 2002; Hassan et al. 2002).

However, factors determining patterns of prevalence require additional investigation. One recent analysis found a gradient of increasing prevalence of coexistent asthma and hay fever among people with increasing levels of education, but decreasing prevalence of asthma without hay fever as educational level increased (Chen 2002). In some areas, asthma prevalence is higher in inner-city areas more likely to have large indoor and outdoor pollution burdens (Lin et al. 2001). Asthma is widely accepted to be aggravated by air pollution exposure (Norris et al. 2000), although new evidence suggests that O₃ may also contribute to asthma onset in children (McConnell et al. 2002).

In addition to diabetes and asthma, some genetic traits that may affect response to air pollution exposure are differentially distributed by race and/or ethnicity (Rios et al. 1993). These traits include fast versus slow acetylation, which affects the ability of the body to remove toxins; deficiency in glucose-6-phosphate dehydrogenase, an enzyme that affects the red blood cell membrane; and sickle cell trait (more common in those of West African descent), which can cause health problems even in heterozygous individuals when exposure to pollutants such as CO occurs (Rios et al. 1993).

Smoking behavior is unequally distributed across socioeconomic levels. In the United

States, smoking has become concentrated among individuals in lower socioeconomic strata, as measured by income and educational attainment (Centers for Disease Control and Prevention 2001). This contrasts with Mexico, where a national survey showed that higher income households consumed more tobacco in the form of cigarettes (Vazquez-Segovia et al. 2002).

Smoking-related lung conditions can affect uptake and response to exposure. Deposition of particles is relatively higher among persons who have chronic obstructive pulmonary disease, especially in the part of the lung that is functional (MacNee and Donaldson 2000). Lung function can decrease among smokers, resulting in increased ventilation-perfusion inhomogeneity, which can in turn affect delivered dose of particles. Smoking behavior may not necessarily result in heightened sensitivity; several studies have observed that smokers may experience less of an impact from air pollution because of a “healthy smoker” effect (Nyberg et al. 2000), wherein individuals who might be sensitive to air pollution effects will choose not to begin smoking because the inhaled smoke causes discomfort or irritation, or smokers experience physiologic changes such as thickening of bronchial mucosa, which make them less responsive to additional pollutant exposure.

The examples of diabetes, asthma, genetic characteristics, and smoking demonstrate that certain traits, health conditions, and behaviors that affect susceptibility to air pollution distribute differentially by SEP. Evaluation of whether air pollution causes adverse health effects to a greater extent among those of relative disadvantage can benefit from insights into biomedical and behavioral characteristics of those in different socioeconomic strata.

Conceptual model. If both exposures and susceptibilities vary across socioeconomic gradients, these factors are likely to act together to influence the health response of groups classified by socioeconomic level. An air pollution epidemiology study that considers air pollution exposure, SEP measures, and potentially other factors related to SEP (disease status, sex, behaviors) must be based, implicitly or explicitly, on a conceptual model that accounts for complex relationships among these factors. Such a model can guide hypothesis development as well as choice of statistical methods. Figure 1 is a simple prototype model showing potential pathways and considerations to guide study design. The model shows that race, ethnicity, and sex may be associated with SEP and are therefore useful to consider. In turn, both differences in vulnerability and exposure may influence or be influenced by SEP and each other. Both exposure and vulnerabilities may lead to disparate health outcomes, which then can cycle back to affect SEP, as in the example cited

above where a disabling illness affects one's ability to work.

The conceptual model developed for a given study will depend on the pollutant being considered and the health outcome. For example, plausible mechanisms for asthma exacerbation may differ from mechanisms for cardiac arrest, and will also depend on what pollutant or combination of pollutants are under consideration. Availability of data and indicators will also affect the pathways that can be explored for a research study.

Methodologic Issues in Research Design and Analysis

Data sources. Current approaches and challenges. An important step in research design is choosing variables that capture characteristics fundamental to the hypothesis being addressed. Published research on SEP and air pollution interactions has employed common individual-level indicators of SEP, including occupational status, income, educational attainment, and wealth; or area-level variables, including neighborhood compositional measures (e.g., percentage of households below the poverty threshold) as well as indices of deprivation (e.g., presence of overcrowding) (Krieger et al. 1997; Liberatos et al. 1988; Lynch and Kaplan 2000). Many of these commonly used variables are measured cross-sectionally, but using socioeconomic measures that incorporate the time dimension (e.g., childhood socioeconomic circumstances, migration history, income dynamics, and/or accumulated monetary resources over time, i.e., wealth) provides a more complete picture of the patterning of health by SEP. In addition to the temporal dimension, space is a critical component of many SEP variables. Recent articles examined which individual- and group-level variables provide the most robust indicators of an association with mortality, a defined and well-measured health outcome. Using longitudinal data from the U.S. Panel Study of Income Dynamics, Daly et al. (2002) examined education, occupation, income, and wealth as predictors of mortality. Wealth and recent family income were the most strongly associated with mortality (Daly et al. 2002).

In an examination of U.S. area-based SEP measurements with respect to cancer incidence and mortality detected socioeconomic gradients by block group and census tract, but using zip codes, Krieger et al. (2002) detected either no gradients or they were in the opposite direction. Economic poverty measures were more robust and sensitive indicators than were education and indices of deprivation (Krieger et al. 2002).

A novel measure of SEP attempts to determine people's subjective perceptions of their socioeconomic standing. The question asks people to place themselves on an imaginary

“ladder” with 10 rungs (John D. and Catherine T. MacArthur Research Network on Socioeconomic Status and Health 2003). Respondents are told that this ladder is a visual representation of people's “standing” within their community to society (based on prestige, wealth, education, etc.), after which they are asked to put themselves on one of the rungs. First developed by the MacArthur Network on Socioeconomic Status and Health, this measure of subjective SEP has now been asked on several cross-sectional and cohort studies. This subjective placement appears to predict health status independently of income, education, and occupational status. Interestingly, people's position on the ladder correlates only moderately (about 0.3–0.4) with objective indicators of SEP, such as income and education. However, further work is required to understand the exact meaning of responses to this question, as well as mechanisms underlying its empirical association with health outcomes (Goodman et al. 2001). Other important aspects of perception relate to the physical and service environment. For example, perceived excessive noise, heavy traffic, inadequate lighting, and limited access to public transportation were associated with increased risk of physical impairment among older adults (Balfour and Kaplan 2002).

As mentioned above, many epidemiology studies use air pollution data obtained from outdoor monitors that typically measure a few key pollutants, such as particles, O₃, and SO₂. Personal exposure is measured using portable samplers (Chang et al. 2000), although this kind of study is much more expensive than using routinely collected community-level data. Many of the limitations and advances in air pollution exposure estimation have been discussed in the preceding section on exposure differentials. Health outcome data can be taken from administrative records of vital statistics (births, deaths, hospital admissions) (Borja-Aburto et al. 1997; Ritz et al. 2000; Zanobetti et al. 2000a) or measured directly using methods including spirometry (for lung function) (Romieu et al. 1998), electrocardiograms (for heart function) (Gold et al. 2000), symptom diaries (Castillejos et al. 1992), and

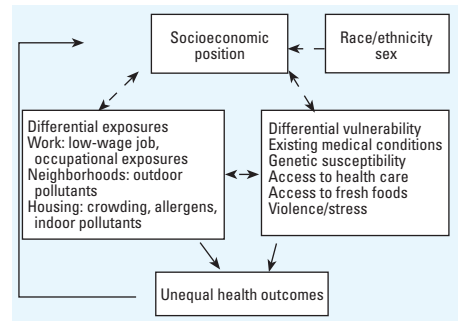


Figure 1. Potential pathways for SEP to increase susceptibility and exposure.

clinical assays of biologic samples such as blood (Schwartz 2001).

Recommendations. Choice of data sources and specific variables should be guided by development of hypotheses for the potential joint effects of individual and neighborhood effects, specifying the relevant geographic areas and their characteristics, and considering longitudinal/life-course features of socioeconomic exposures (Diez Roux 2001). For administrative reasons, data are often collected at the resolution of counties or postal codes. Researchers should evaluate whether these divisions encompass community units or neighborhoods that are of interest or relevance for health outcome studies (Krieger et al. 2002). Although research using census data or commonly measured individual SEP indicators aid comparisons and generalizability across study settings, air pollution researchers should also consider using some of the more innovative SEP indicators described previously and tap existing databases with advanced or high-resolution SEP indicators, such as those that allow linkage of detailed survey data on social cohesion and

other contextual exposures, for example (Sampson et al. 1997). Linking health outcome data from various sources, such as surveys and vital statistics records, can also enable more complete control of such characteristics as medical history in studies of SEP/air pollution interactions (Finkelstein et al. 2003).

Another approach is to use innovative metrics for evaluating both socioeconomic and environmental inequalities. The Gini coefficient is a commonly used measure of inequality, taking on a value of zero for perfect equality and one for total inequality. Several researchers have proposed the application of Gini coefficients to measure environmental inequality, and some suggest that environmental inequalities be measured within groups of comparable income, under the assumption that people should be compensated as a way of “making up” for increased environmental exposure (Millimet and Slotje 2002). This approach could be applied to air pollution exposure in cases where the pollutants of interest vary enough within or between communities to give an indication of how uneven the

distribution of pollution is among different social groups.

Although we have identified and discussed a number of articles, still little is known about differences in personal exposure patterns across socioeconomic groups. Further exposure assessment work that explicitly includes SEP as a selection criterion for participation would be useful. Other factors to consider include diurnal patterns in exposure, traffic density, and proximity to schools.

Statistical analyses. Current approaches and challenges. A number of challenges exist relating to the conduct and interpretation of health inequities studies using area-based socioeconomic variables. Greenland (2001) reviewed some of the potential sources of bias and problems in interpretation in studying contextual exposures and suggested multilevel study design as an appropriate method to address some of the identified concerns. Multilevel analysis evaluates the effects of individual-level and group-level exposure variables on individual-level outcomes at the same time (Diez Roux 2000). Researchers have applied such models to assess whether the effects of income inequality in a society are associated with poor health outcomes beyond what individual income attainment does, and whether this contextual effect is independent of a given individual's income (Kawachi 2000). The approach has also been described for air pollution studies (Navidi et al. 1994). Diez Roux (2000) provided a comprehensive introduction to these models and various hypotheses they can address.

Contextual SEP indicators may be affected by a common concern pertinent to area-based statistics: the “modifiable areal unit problem” (Ratcliffe and McCullagh 1999). Modifying the scale of aggregation zones (e.g., postal codes, counties, grids) and which geographic units are aggregated together can change study results by masking heterogeneity within them, among other problems (Anderton et al. 1994).

Air pollution exposure also has a strong spatial component. Most studies of air pollution and health outcomes have neither analyzed nor reported whether the residuals from regression models had spatial autocorrelation (Bowen 2002). In one study that has applied spatial regression techniques (Jerrett et al. 2001), control for autocorrelation through the application of simultaneous autoregression models affected not only which variables were significant but also the overall prediction accuracy of the model.

Examining geographic patterns of inequity using GIS systems can be informative and visually illustrate the problem. Figure 2 is an example from southeast England. Traffic density and most air pollutants are highest in east London, where many poorer population groups live, often with substantial ethnic

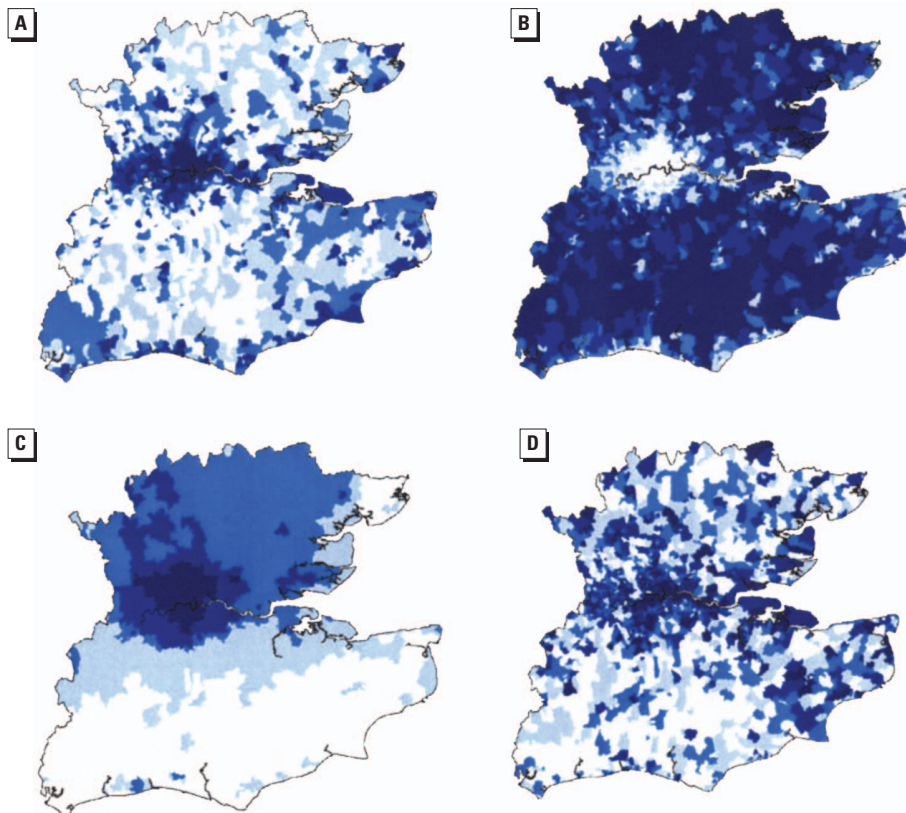


Figure 2. Ward-level maps for southeast England showing quintiles (with categories for variables going from lowest to highest levels, indicated by the lightest color to the darkest). (A) Carstairs index of socioeconomic deprivation (from most affluent to most deprived; based on data from the 1991 census; Census Disemination Unit 1999). (B) Percentage of households with access to a car ($\geq 91\%$, 88–90%, 81–87%, 69–80%, 27–68%; based on data from the 1991 census). (C) Annual mean NO_2 concentrations (in ppb) for 1991 data (0–11.8, 11.9–15.7, 15.8–18.7, 18.8–27.9, 28.0–35.8). (D) Standardized mortality ratio for respiratory disease, 0–74 years of age, 1986–1995 (0–0.64, 0.65–0.83, 0.84–1.02, 1.03–1.29, 1.30–6.56). This figure is based on work by C. Stephens, S. Stephenson, M. Landon, T. Fletcher, and P. Wilkinson for the U.K. Economic and Social Research Council (ESRC 2001), provided with the support of the ESRC and copyright by ED-LINE and Crown.

minority communities and higher prevalences of cardiorespiratory illness; wealthier populations, with lower underlying disease prevalence but greater car ownership, tend to reside in the lower-pollution areas of the suburbs and surrounding areas (Economic and Social Research Council 2001).

Innovative study designs such as “case crossover” (Maclure 1991) have recently been applied to air pollution epidemiology. The case-crossover design allows assessment of the effect of transient exposures on the risk of an acute event. Using a bidirectional approach where control data are obtained both before and after the health event of interest, factors such as temporal trends, season, day of week, and changes in population size and composition are controlled by design. But its greatest advantage is that individual-level characteristics such as SEP are controlled by design but can be analyzed as effect modifiers (Pope 1999).

A final statistical challenge relates to the study of effect modification. In the air pollution literature, multiplicative scale interactions (i.e., difference in relative risks across population subgroups) have been commonly evaluated. These are conceptually justified because, for example, a doubling of the population would be expected to result in a doubling of the incremental number of cases. However, additive scale effect modification may be useful for risk assessment. This is because results can be expressed in terms of numbers of deaths or hospitalizations attributable to air pollution exposure, compared with the percentage of changes that may be the same in two different population groups who have different baseline rates of the health event. For example, blacks in some areas have higher rates of asthma hospital admissions than do whites, so for the same percentage of excess admissions associated with pollution, more blacks than whites are actually affected, despite no evidence of differential susceptibility (Gwynn and Thurston 2001; Rothman and Greenland 1998). Indeed, two studies that evaluated multiplicative-scale interaction offered the baseline differences as one possible explanation for why little difference was seen [Health Effects Institute (HEI) 2000; Zanobetti et al. 2000b].

Recommendations. In light of the additive versus multiplicative effect modification issues, studies should account for baseline differences in health response across socioeconomic strata and consider reporting risk/rate differences in addition to ratios. In addition, statistical methods should account for spatial correlation in both pollution and SEP variables. Characterizing spatial relationships more systematically would also be useful, ideally using methods of exposure estimation that take account of the movement of individuals through the urban environment rather

than relying on static geography alone. And, innovative statistical approaches including case crossover should be employed for studying air pollution and SEP interaction. Our final suggestion is that multilevel modeling be more widely applied to studies of this nature. Below we provide a theoretical example of a multilevel model approach applied to a study of O₃ and daily mortality.

Consider a cohort study of n subjects with information about individual-level measures of SEP and medical history (including smoking) as well as contextual (e.g., density of liquor stores, parks, fast food) and compositional (e.g., literacy rates) variables pertaining to the neighborhood where they live. Air quality data can be modeled using regression or dispersion modeling to the level of individual address, including workplace exposures and indoor exposures based on questionnaires. Because people move around but spend more time out of their houses in their community than in distant communities, a contextual air pollution variable can then also be constructed. In such a scenario, consider a survival model where the risk ratio for mortality is modeled as

$$\begin{aligned} \log RR_{ij} = & \beta_0 + \beta_1 \\ & \times \text{personal risk factors}_i + \beta_2 \\ & \times O_{3j} + \beta_3 \times (O_{3i} - O_{3j}) + \beta_4 \\ & \times O_{3i} \times SEP_i + V_j + U_j \times O_{3j}, \quad [1] \end{aligned}$$

where i represents an individual in the j th area; V_j represents a random area effect capturing variations in risk, not explained by personal risk factors, that cluster within geographic areas; and U_j represents variation in the slope of O₃, not explained by the individual level interaction terms, which cluster geographically by areas. β_2 measures the effect of areawide O₃ exposure and β_3 the effect of the difference from that areawide exposure for the i th individual. β_4 represents the effect modification of individual SEP on the response to O₃. Because of the large number of groups, it is customary to treat the U_j and V_j as random. The second level of the multilevel model assumes that

$$\begin{aligned} U_j = & \alpha_j + \gamma_1 \\ & \times \text{contextual/compositional variable 1} \\ & + \dots \gamma_p \\ & \times \text{contextual/compositional variable } p \quad [2] \end{aligned}$$

$$\begin{aligned} V_j = & \sigma_j + \eta_1 \\ & \times \text{contextual/compositional variable 1} \\ & + \dots \eta_p \\ & \times \text{contextual/compositional variable } p. \quad [3] \end{aligned}$$

Here α_j and σ_j represent the remaining heterogeneity in baseline risk or pollution slope unexplained by the contextual/compositional variables. Fitting such models requires extensions to standard Cox regression packages [some of which do incorporate U_j (frailty models) but not V_j]. However, a proportional hazard model can be fit as a two-stage generalized linear model, allowing the use of generalized linear mixed model procedures. This approach has the additional advantage of allowing the use of flexible functions to examine the potential for nonlinear dependence of risk on some of the covariates. Of course, in reality, not all of the above data may be available or affordable, and in any case, power considerations will certainly limit the number of interactions than can be considered. For continuous, binomial, or count outcomes these models can be fit using generalized linear mixed models.

Conclusions

Research may show that groups most likely to be made ill from air pollution also receive the highest exposure, and this exposure then exerts larger effects on their health than it does on the average or reference population. The public health and regulatory implications of such a finding could be significant because most air pollution standards aim to reduce average exposure over large regions, rather than targeting exposure reduction and mitigation programs to those areas receiving the highest exposure (Jerrett et al. 2001; Levy et al. 2002). Thus, targeting exposure reduction would be justified on the grounds of maximizing public health benefits. Differential distribution of adverse health effects (as addressed in this article) also need to be considered alongside differential distribution of the benefits (e.g., employment or car ownership) related to the emission sources. In one of the few studies that has assessed the impact of air quality regulations, the overall conclusion was that poor people and communities tend to benefit most from air quality improvements (Bae 1997).

Including both air pollution and socioeconomic variables in epidemiologic studies can help inform public policy that aims to protect those most vulnerable to air pollution exposure; identify cost-effective, targeted mitigation efforts; ensure equitable protection from health risks; and develop physiologic explanations for the observed associations with SEP. As researchers evaluate how socioeconomic disparities and pollution exposure can affect health and quality of life, their work can benefit through careful consideration of the themes addressed in this article. First, researchers can clearly define their working hypotheses, considering exposures and susceptibilities and both temporal and spatial dimensions. Second, new collaborations can be formed

among environmental and social epidemiologists, exposure assessment experts, and other researchers to aid selection of appropriate tools and data sets. Third, research ideas can be developed in collaboration with affected communities and policy makers tasked with environmental and health protection, as well as social and economic policies. Finally, international perspectives and collaborative studies can enhance understanding and improve public health action by showing how the complex relationships among SEP, pollution, and health vary across communities and nations.

REFERENCES

- Anderton D, Anderson J, Oakes J, Fraser M. 1994. Environmental equity: the demographics of dumping. *Demography* 31:229–248.
- Appleyard D. 1981. *Livable Streets*. Berkeley, CA:University of California Press.
- Bach JF. 2002. Mechanisms of disease: the effect of infections on susceptibility to autoimmune and allergic diseases. *N Engl J Med* 347:911–920.
- Bae CHC. 1997. The equity impacts of Los Angeles' air quality policies. *Environ Plan A* 29:1563–1584.
- Balfour JL, Kaplan GA. 2002. Neighborhood environment and loss of physical function in older adults: evidence from the Alameda County study. *Am J Epidemiol* 155:507–515.
- Bateson TF, Schwartz J. In press. Who is sensitive to the effects of particles on mortality? A case-crossover analysis. *Epidemiology*.
- Been V. 1993. What's fairness got to do with it? Environmental justice and the siting of locally undesirable land uses. *Cornell Law Rev* 78:1001–1036.
- Bell ML, Davis D, Cifuentes L, Cohen A, Gouveia N, Grant L, et al. 2002. International Expert Workshop on the Analysis of the Economic and Public Health Impacts of Air Pollution: workshop summary. *Environ Health Perspect* 110:1163–1168.
- Bellander T, Berglund N, Gustavsson P, Jonson T, Nyberg F, Pershagen G, et al. 2001. Using geographic information systems to assess individual historical exposure to air pollution from traffic and house heating in Stockholm. *Environ Health Perspect* 109:633–639.
- Bobak M, Leon DA. 1999. The effect of air pollution on infant mortality appears specific for respiratory causes in the postneonatal period. *Epidemiology* 10:666–670.
- Borja-Aburto VH, Loomis DP, Bangdiwala SI, Shy CM, Rascon-Pacheco RA. 1997. Ozone, suspended particulates, and daily mortality in Mexico City. *Am J Epidemiol* 145:258–268.
- Bowen W. 2002. An analytical review of environmental justice research: what do we really know? *Environ Manage* 29:3–15.
- Briggs D, Collins S, Elliott P, Fischer P, Kingham S, Lebert E, et al. 1997. Mapping urban air pollution using GIS: a regression-based approach. *Int J Geogr Inf Sci* 11:699–718.
- Brunekreef B, Holgate ST. 2002. Air pollution and health. *Lancet* 360:1233–1242.
- Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knappe M, van Vliet P. 1997. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology* 8:298–303.
- Brunner EJ, Marmot MG, Nanchahal K, Shipley MJ, Stansfeld SA, Juneja M, et al. 1997. Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II study. *Diabetologia* 40:1341–1349.
- Buckeridge D, Glazier R, Harvey B, Escobar M, Amrhein C, Frank J. 2002. Effect of motor vehicle emissions on respiratory health in an urban area. *Environ Health Perspect* 110:293–300.
- Bullard RD, Wright BH. 1993. Environmental justice for all: community perspectives on health and research needs. *Toxicol Ind Health* 9:821–841.
- Burton RM, Suh HH, Koutrakis P. 1996. Spatial variation in particulate concentrations within metropolitan Philadelphia. *Environ Sci Technol* 30:400–407.
- Castillejos M, Gold DR, Damokosh AI, Serrano P, Allen G, McDonnell WF, et al. 1995. Acute effects of ozone on the pulmonary function of exercising schoolchildren in Mexico City. *Am J Respir Crit Care Med* 152:1501–1507.
- Castillejos M, Gold DR, Dockery D, Tosteson T, Baum T, Speizer FE. 1992. Effects of ambient ozone on respiratory function and symptoms in Mexico City schoolchildren. *Am Rev Respir Dis* 145:276–282.
- Census Dissemination Unit. 1999. 1991 Area Statistics. Available: http://census.ac.uk/cdu/Datasets/1991_Census_datasets/Area_Stats/ [accessed 30 October 2003].
- Centers for Disease Control and Prevention. 2001. Cigarette smoking among adults—United States, 1999. *Morb Mortal Wkly Rep* 50:869–873.
- Chang LT, Koutrakis P, Catalano PJ, Suh HH. 2000. Hourly personal exposures to fine particles and gaseous pollutants—results from Baltimore, Maryland. *J Air Waste Manage* 50:1223–1235.
- Chen JT, Krieger N, Van Den Eeden SK, Quesenberry CP. 2002. Different slopes for different folks: socioeconomic and racial/ethnic disparities in asthma and hay fever among 173,859 U.S. men and women. *Environ Health Perspect* 110(suppl 2):211–216.
- Cifuentes L, Vega J, Lave L. 1999. Daily mortality by cause and socio-economic status in Santiago, Chile 1988–1996. *Epidemiology* 10:S45.
- Clancy L, Goodman P, Sinclair H, Dockery DW. 2002. Effect of air-pollution control on death rates in Dublin, Ireland: an intervention study. *Lancet* 360:1210–1214.
- Clean Air Act. 2001. In: *Compilation of Selected Acts within the Jurisdiction of the Committee on Commerce: Environmental Law, Vol 1*. Available: <http://epw.senate.gov/cleanair.pdf> [accessed 7 October 2003].
- Cowie CC, Eberhardt MS. 1995. Sociodemographic characteristics of persons with diabetes. In: *Diabetes in America*. 2nd ed. NIH Publication No. 95-1468. Bethesda, MD:National Diabetes Data Group, National Institute of Diabetes and Digestive and Kidney Diseases.
- Daly MC, Duncan GJ, McDonough P, Williams DR. 2002. Optimal indicators of socioeconomic status for health research. *Am J Public Health* 92:1151–1157.
- Davey Smith G, Gunnell D, Ben-Shlomo Y. 2001. Life-course approaches to socio-economic differentials in cause-specific adult mortality. In: *Poverty, Inequality and Health: An International Perspective* (Leon D, Walt G, eds). Oxford, UK:Oxford University Press, 88–124.
- Davis DL, Saldava PHN. 1999. Urban Air Pollution Risks to Children: A Global Environmental Health Indicator. Internal Report. Washington, DC:World Resources Institute. Available: http://pdf.wri.org/urbanair_health.pdf [accessed 7 October 2003].
- Diez Roux AV. 2001. Investigating neighborhood and area effects on health. *Am J Public Health* 91:1783–1789.
- . 2000. Multilevel analysis in public health research. *Annu Rev Public Health* 21:171–192.
- Diez Roux AV, Merkin SS, Arnett D, Chambless L, Massing M, Nieto FJ, et al. 2001. Neighborhood of residence and incidence of coronary heart disease. *N Engl J Med* 345:99–106.
- Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME, et al. 1993. An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 329:1753–1759.
- Ekoe JM, Zimmet P, Williams R, eds. 2001. *The Epidemiology of Diabetes Mellitus: An International Perspective*. Chichester, UK:John Wiley & Sons, Ltd.
- English P, Neutra R, Scalf R, Sullivan M, Waller L, Zhu L. 1999. Examining associations between childhood asthma and traffic flow using a geographic information system. *Environ Health Perspect* 107:761–767.
- ESRC. 2001. Environmental Justice: Rights and Means to a Healthy Environment for All. Special Briefing No. 7. Swindon, UK:Economic and Social Research Council, Global Environmental Change Programme. Available: http://www.foe.co.uk/resource/reports/environmental_justice.pdf [accessed 7 October 2003].
- Evans GW, Kantrowitz E. 2002. Socioeconomic status and health: the potential role of environmental risk exposure. *Annu Rev Public Health* 23:303–331.
- Ezzati M, Lopez AD, Rodgers A, Vander Hoorn S, Murray CJ, Comparative Risk Assessment Collaborating Group. 2002. Selected major risk factors and global and regional burden of disease. *Lancet* 360:1347–1360.
- Finkelstein M, Jerrett M, De Luca P, Finkelstein N, Verma DK, Chapman K, et al. 2003. Environmental justice: a cohort study of income, air pollution and mortality. *Can Med Assoc J* 169:397–402.
- Gehring U, Cyrys J, Sedlmeir G, Brunekreef B, Bellander T, Fischer P, et al. 2002. Traffic-related air pollution and respiratory health during the first 2 years of life. *Eur Respir J* 19:690–698.
- Godish T. 1991. *Air Quality*. 2nd ed. Chelsea, MI:Lewis Publishers.
- Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B, et al. 2000. Ambient pollution and heart rate variability. *Circulation* 101:1267–1273.
- Goldberg MS, Burnett RT, Brook J, Bailer JC III, Valois MF, Vincent R. 2001. Associations between daily cause-specific mortality and concentrations of ground-level ozone in Montreal, Quebec. *Am J Epidemiol* 154:817–826.
- Goodman E, Adler NE, Kawachi I, Frazier AL, Huang B, Colditz GA. 2001. Adolescents' perceptions of social status: development and evaluation of a new indicator. *Pediatrics* 108:E31. Available: <http://pediatrics.aappublications.org/cgi/content/full/108/2/e31> [accessed 7 October 2003].
- Gouveia N, Fletcher T. 2000. Time series analysis of air pollution and mortality: effects by cause, age and socio-economic status. *J Epidemiol Community Health* 54:750–755.
- Graves P, Murdock JC, Thayer MA, Waldman D. 1988. The robustness of hedonic price estimation: urban air quality. *Land Econ* 64:220–233.
- Greenland S. 2001. Ecologic versus individual-level sources of bias in logistic estimates of contextual health effects. *Int J Epidemiol* 30:1343–1350.
- Gwynn RC, Thurston GD. 2001. The burden of air pollution: impacts among racial minorities. *Environ Health Perspect* 109(suppl 4):501–506.
- Haan M, Kaplan GA, Camacho T. 1987. Poverty and health. Prospective evidence from the Alameda County Study. *Am J Epidemiol* 125:989–998.
- Hassan MR, Kabir AR, Mahmud AM, Rahman F, Hossain MA, Bennoor KS, et al. 2002. Self-reported asthma symptoms in children and adults of Bangladesh: findings of the national asthma prevalence study. *Int J Epidemiol* 31:483–488.
- HEI. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Cambridge, MA:Health Effects Institute. Available: <http://www.healtheffects.org/Pubs/Rean-ExecSumm.pdf> [accessed 7 October 2003].
- Hewitt CN. 1991. Spatial variations in nitrogen dioxide concentrations in an urban area. *Atmos Environ* 25B:429–434.
- Hitchins J, Morawska L, Wolff R, Gilbert D. 2000. Concentrations of submicrometre particles from vehicle emissions near a major road. *Atmos Environ* 34:51–59.
- Hoek G, Fischer P, VanDenBrandt P, Goldbohm S, Brunekreef B. 2001. Estimation of long-term average exposure to outdoor air pollution for a cohort study on mortality. *J Expo Anal Environ Epidemiol* 11:459–469.
- Institute of Medicine. 1999. *Toward Environmental Justice: Research, Education, and Health Policy Needs*. Washington, DC:National Academy Press.
- Israel BA, Schulz AJ, Parker EA, Becker AB. 1998. Review of community-based research: assessing partnership approaches to improve public health. *Annu Rev Public Health* 19:173–202.
- Ito K, Thurston GD. 1996. Daily PM₁₀/mortality associations: an investigation of at-risk subpopulations. *J Expo Anal Environ Epidemiol* 6:79–95.
- Janssen NA, Hoek G, Brunekreef B, Harssema H, Mensink I, Zuidhof A. 1998. Personal sampling of particles in adults: relation among personal, indoor, and outdoor air concentrations. *Am J Epidemiol* 147:537–547.
- Jerrett M, Burnett RT, Kanaroglou P, Eyles J, Finkelstein N, Giovis C, et al. 2001. A GIS-environmental justice analysis of particulate air pollution in Hamilton, Canada. *Environ Plan A* 33:955–973.
- Kaplan GA. 1998. The role of epidemiologists in eradicability of poverty. *Lancet* 352:1627–1628.
- Kattan M, Mitchell H, Eggleston P, Gergen P, Crain E, Redline S, et al. 1997. Characteristics of inner-city children with asthma: the national cooperative inner-city asthma study. *Pediatr Pulm* 24:253–262.
- Kawachi I. 2000. Income inequality and health. In: *Social Epidemiology* (Berkman LF, Kawachi I, eds). New York:Oxford University Press, 76–94.
- Kawachi I, Berkman L. 2003. *Neighborhoods and Health*. New York:Oxford University Press.
- Kinney PL, Aggarwal M, Northridge ME, Janssen NA, Shepard P. 2000. Airborne concentrations of PM_{2.5} and diesel exhaust particles on Harlem sidewalks: a community-based pilot study. *Environ Health Perspect* 108:213–218.
- Korc ME. 1996. A socioeconomic assessment of human exposure to ozone in the south coast air basin of California. *J Air Waste Manage* 46:547–557.

- Krieger JW, Song L, Takaro TK, Stout J. 2000. Asthma and the home environment of low-income urban children: preliminary findings from the Seattle-King County healthy homes project. *J Urban Health* 77:50–67.
- Krieger N, Waterman P, Chen JT, Soobader MJ, Subramanian SV, Carson R. 2002. ZIP code caveat: bias due to spatiotemporal mismatches between zip codes and US census-defined geographic areas—the Public Health Disparities Geocoding Project. *Am J Public Health* 92:1100–1102.
- Krieger N, Williams DR, Moss NE. 1997. Measuring social class in U.S. public health research: concepts, methodologies, and guidelines. *Annu Rev Public Health* 18:341–378.
- Leaderer BP, Belanger K, Triche E, Holford T, Gold DR, Kim Y, et al. 2002. Dust mite, cockroach, cat, and dog allergen concentrations in homes of asthmatic children in the northeastern United States: impact of socioeconomic factors and population density. *Environ Health Perspect* 110:419–425.
- Levy JI, Greco SL, Spengler JD. 2002. The importance of population susceptibility for air pollution risk assessment: a case study of power plants near Washington, DC. *Environ Health Perspect* 110:1253–1260.
- Liberatos P, Link BG, Kelsey JL. 1988. The measurement of social class in epidemiology. *Epidemiol Rev* 10:87–121.
- Lin RS, Sung FC, Huang SL, Gou YL, Ko YC, Gou HW, et al. 2001. Role of urbanization and air pollution in adolescent asthma: a mass screening in Taiwan. *J Formos Med Assoc* 100:649–655.
- Linn WS, Słazhczic Y, Gong H Jr, Kinney PL, Berhane KT. 2000. Air pollution and daily hospital admissions in metropolitan Los Angeles. *Environ Health Perspect* 108:427–434.
- Liu LJ, Delfino R, Koutrakis P. 1997. Ozone exposure assessment in a Southern California community. *Environ Health Perspect* 105:58–65.
- Lynch J, Kaplan J. 2000. Socioeconomic position. In: *Social Epidemiology* (Berkman L, Kawachi I, eds). New York:Oxford University Press, 13–35.
- Maantay J. 2001. Zoning, equity, and public health. *Am J Public Health* 91:1033–1041.
- John D. and Catherine T. MacArthur Research Network on Socioeconomic Status and Health. 2003. Network Research. Available: <http://www.macses.ucsf.edu/Research/overview.htm> [accessed 13 August 2003].
- Maclure M. 1991. The case-crossover design: a method for studying transient effects on the risk of acute events. *Am J Epidemiol* 133:144–153.
- MacNee W, Donaldson K. 2000. Exacerbations of COPD: environmental mechanisms. *Chest* 117:390S–397S.
- Marmot M. 2001. Inequalities in health. *N Engl J Med* 345:134–136.
- McConnell R, Berhane K, Gilliland F, London SJ, Islam T, Gauderman WJ, et al. 2002. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359:386–391.
- McDonough P, Duncan GJ, Williams D, House J. 1997. Income dynamics and adult mortality in the United States, 1972 through 1989. *Am J Public Health* 87:1476–1483.
- McMichael AJ. 1998. The role of epidemiologists in eradicability of poverty [Letter]. *Lancet* 352:1627.
- Millimet DL, Słottje D. 2002. An environmental Paglin-Gini. *Appl Econ Lett* 9:271–274.
- Morland K, Wing S, Diez Roux A, Poole C. 2002. Neighborhood characteristics associated with the location of food stores and food service places. *Am J Prev Med* 22:23–29.
- National Research Council. 1998. *Research Priorities for Airborne Particulate Matter I. Immediate Priorities and a Long-range Research Portfolio*. Washington, DC:National Academy Press.
- Navidi W, Thomas D, Stram D, Peters J. 1994. Design and analysis of multilevel analytic studies with applications to a study of air pollution. *Environ Health Perspect* 102(suppl 8):25–32.
- Nielsen T. 1996. Traffic contribution of polycyclic aromatic hydrocarbons in the center of a large city. *Atmos Environ* 30:3481–3490.
- Norris G, Larson T, Koenig J, Claiborn C, Sheppard L, Finn D. 2000. Asthma aggravation, combustion, and stagnant air. *Thorax* 55:466–470.
- Nyberg F, Gustavsson P, Jarup L, Bellander T, Berglund N, Jakobsson R, et al. 2000. Urban air pollution and lung cancer in Stockholm. *Epidemiology* 11:487–495.
- Oglesby L, Rotko T, Krutli P, Boudet C, Kruize H, Nen MJ, et al. 2000. Personal exposure assessment studies may suffer from exposure-relevant selection bias. *J Expo Anal Environ Epidemiol* 10:251–266.
- Pearce N, Beasley R, Burgess C, Crane J, eds. 1998. *Asthma Epidemiology: Principles and Methods*. New York:Oxford University Press.
- Pellizzari E, Perritt R, Clayton C. 1999. National human exposure assessment survey (NHEXAS): exploratory survey of exposure among population subgroups in EPA Region V. *J Expo Anal Environ Epidemiol* 9:49–55.
- Peters A, Frohlich M, Doring A, Immervoll T, Wichmann HE, Hutchinson WL, et al. 2001. Particulate air pollution is associated with an acute phase response in men; results from the Monica-Augsburg study. *Eur Heart J* 22:1198–1204.
- Pijawka DJ, Blair J, Guhathakurta SLS, Ashur S. 1998. Environmental equity in central cities: socioeconomic dimensions and planning strategies. *J Plan Educ Res* 18:113–123.
- Pikhart H, Bobak M, Gorynski P, Wojtyniak B, Danova J, Celko MA, et al. 2001. Outdoor sulphur dioxide and respiratory symptoms in Czech and Polish school children: a small-area study (SAVIAH). Small-area variation in air pollution and health. *Int Arch Occup Environ Health* 74:574–578.
- Pope CA III. 1999. Mortality and air pollution: associations persist with continued advances in research methodology. *Environ Health Perspect* 107:613–614.
- Pope CA III, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, et al. 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA* 287:1132–1141.
- Pulido L. 2000. Rethinking environmental racism: white privilege and urban development in Southern California. *Ann Assoc Am Geogr* 90:12–40.
- Ratcliffe JH, McCullagh MJ. 1999. Hotbeds of crime and the search for spatial accuracy. *Geogr Syst* 1:385–398.
- Rijnders E, Janssen NA, van Vliet PH, Brunekreef B. 2001. Personal and outdoor nitrogen dioxide concentrations in relation to degree of urbanization and traffic density. *Environ Health Perspect* 109(suppl 3):411–417.
- Rios R, Poje GV, Detels R. 1993. Susceptibility to environmental pollutants among minorities. *Toxicol Ind Health* 9:797–820.
- Ritz B, Yu F, Chapa G, Fruin S. 2000. Effect of air pollution on preterm birth among children born in Southern California between 1989 and 1993. *Epidemiology* 11:502–511.
- Ritz B, Yu F, Fruin S, Chapa G, Shaw GM, Harris JA. 2002. Ambient air pollution and risk of birth defects in Southern California. *Am J Epidemiol* 155:17–25.
- Romieu I, Meneses F, Ramirez M, Ruiz S, Perez Padilla R, Sienra JJ, et al. 1998. Antioxidant supplementation and respiratory functions among workers exposed to high levels of ozone. *Am J Respir Crit Care Med* 158:226–232.
- Romieu I, Ramirez M, Meneses F, Ashley D, Lemire S, Colome S, et al. 1999. Environmental exposure to volatile organic compounds among workers in Mexico City as assessed by personal monitors and blood concentrations. *Environ Health Perspect* 107:511–515.
- Rothman KJ, Greenland S, eds. 1998. *Modern Epidemiology*. 2nd ed. Philadelphia:Lippincott-Raven.
- Rotko T, Hanninen O, Jantunen MJ. 2002. Time used in traffic by sociodemographic groups in Expolis-Helsinki. *Epidemiology* 13:S256–257.
- Rotko T, Koistinen K, Hanninen O, Jantunen M. 2000. Sociodemographic descriptors of personal exposure to fine particles (PM_{2.5}) in Expolis Helsinki. *J Expo Anal Environ Epidemiol* 10:385–393.
- Rotko T, Kousa A, Alm S, Jantunen M. 2001. Exposures to nitrogen dioxide in Expolis-Helsinki: microenvironment, behavioral and sociodemographic factors. *J Expo Anal Environ Epidemiol* 11:216–223.
- Samet JM, Dominici F, Currier FO, Coursac I, Zeger SL. 2000. Fine particulate air pollution and mortality in 20 U.S. cities, 1987–1994. *N Engl J Med* 343:1742–1749.
- Sampson RJ, Raudenbush SW, Earls F. 1997. Neighborhoods and violent crime: a multilevel study of collective efficacy. *Science* 277:918–924.
- Sarnat JA, Koutrakis P, Suh HH. 2000. Assessing the relationship between personal particulate and gaseous exposures of senior citizens living in Baltimore, MD. *J Air Waste Manage* 50:1184–1198.
- Schwartz J. 2001. Air pollution and blood markers of cardiovascular risk. *Environ Health Perspect* 109:405–409.
- Schwartz J, Zanobetti A. 2000. Using meta-smoothing to estimate dose-response trends across multiple studies, with application to air pollution and daily death. *Epidemiology* 11:666–672.
- Sexton K. 1997. Sociodemographic aspects of human susceptibility to toxic chemicals: do class and race matter for realistic risk assessment? *Environ Toxicol Pharmacol* 4:261–269.
- Sexton K, Adgate JL. 1999. Looking at environmental justice from an environmental health perspective. *J Expo Anal Environ Epidemiol* 9:3–8.
- Sexton K, Gong H Jr, Bailar JC, Ford JG, Gold DR, Lambert WE, et al. 1993. Air pollution health risks: do class and race matter? *Toxicol Ind Health* 9:843–878.
- Smith KR, Samet JM, Romieu I, Bruce N. 2000. Indoor air pollution in developing countries and acute lower respiratory infections in children. *Thorax* 55:518–532.
- Suh HH, Allen GA, Koutrakis P, Burton RM. 1995. Spatial variation in acidic sulfate and ammonia concentrations within metropolitan Philadelphia. *J Air Waste Manage* 45:442–451.
- Susser M. 1998. Does risk factor epidemiology put epidemiology at risk—peering into the future. *J Epidemiol Community Health* 52:608–611.
- Tolbert PE, Mulholland JA, MacIntosh DL, Xu F, Daniels D, Devine DJ, et al. 2000. Air quality and pediatric emergency room visits for asthma in Atlanta, Georgia, USA. *Am J Epidemiol* 151:798–810.
- Vazquez-Segovia LA, Sesma-Vazquez S, Hernandez-Avila M. 2002. Tobacco consumption in Mexican households: results from the national household income and expenditure survey, 1984–2000. *Salud Publica Mex* 44:S76–S81.
- Visscher TL, Seidell JC. 2001. The public health impact of obesity. *Annu Rev Public Health* 22:355–375.
- Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. 1999. Elevated C-reactive protein levels in overweight and obese adults. *JAMA* 282:2131–2135.
- Weiland SK, Mundt KA, Ruckmann A, Keil U. 1994. Self-reported wheezing and allergic rhinitis in children and traffic density on street of residence. *Ann Epidemiol* 4:243–247.
- WHO. 2000. *Air Quality Guidelines for Europe*. Copenhagen:World Health Organization, Regional Office for Europe.
- Wojtyniak B, Rabaczko D, Stokwiszewski J. 2001. Does air pollution have respect for the socio-economic status of people? [Abstract.] *Epidemiology* 12:S64.
- Working Group on Public Health and Fossil-Fuel Combustion. 1997. Short-term improvements in public health from global-climate policies on fossil-fuel combustion: an interim report. *Lancet* 350:1341–1349.
- World Bank. 2002. *Millennium Development Goals: Eradicate Extreme Poverty and Hunger*. Available: <http://www.developmentgoals.org/Poverty.htm> [accessed 13 August 2003].
- Wright RJ, Rodriguez M, Cohen S. 1998. Review of psychosocial stress and asthma: an integrated biopsychosocial approach. *Thorax* 53:1066–1074.
- Wright RJ, Steinbach SF. 2001. Violence: an unrecognized environmental exposure that may contribute to greater asthma morbidity in high risk inner-city populations. *Environ Health Perspect* 109:1085–1089.
- Zanobetti A, Schwartz J. 2000. Race, gender, and social status as modifiers of the effects of PM₁₀ on mortality. *J Occup Environ Med* 42:469–474.
- . 2001. Are diabetics more susceptible to the health effects of airborne particles? *Am J Respir Crit Care Med* 164:831–833.
- . 2002. Cardiovascular damage by airborne particles: are diabetics more susceptible? *Epidemiology* 13:588–592.
- Zanobetti A, Schwartz J, Dockery DW. 2000a. Airborne particles are a risk factor for hospital admissions for heart and lung disease. *Environ Health Perspect* 108:1071–1077.
- Zanobetti A, Schwartz J, Gold D. 2000b. Are there sensitive subgroups for the effects of airborne particles? *Environ Health Perspect* 108:841–845.
- Zhu YF, Hinds WC, Kim S, Shen S, Sioutas C. 2002. Study of ultra-fine particles near a major highway with heavy-duty diesel traffic. *Atmos Environ* 36:4323–4335.