

National environmental health measures for minority and low-income populations: Tracking social disparities in environmental health

Devon Payne-Sturges^{a,*}, Gilbert C. Gee^b

^aOffice of Children's Health Protection, US Environmental Protection Agency, Ariel Rios Bldg. MC 1107A, 1200 Pennsylvania Ave., NW, Washington, DC 20460, USA

^bDepartment of Health Behavior and Health Education, University of Michigan School of Public Health, USA

Received 27 October 2005; received in revised form 18 May 2006; accepted 23 May 2006

Available online 27 July 2006

Abstract

Healthy People 2010 [US Department of Health and Human Services, 2004. Healthy People 2010. Available: <http://www.healthypeople.gov/Publications/> [accessed May 22, 2004]] has established as a top priority the elimination of health disparities. Current research suggests that characteristics of the social, physical and built environment contribute to these disparities. In order to track progress and to assess the potential contributions of the various components of the “environment,” tools specific to environmental health disparities are required.

In this paper, we discuss one potential tool, a set of candidate measures that may be used to track disparities in outcomes, as well as measures that may be used analytically to assess potential causal pathways. Several other reports on health and environmental measures have been produced, including the Environmental Protection Agency's (EPA) *America's Children and the Environment*. However, there has not been a comprehensive discussion about environmental measures that focus on racial, ethnic and socioeconomic disparities in health. Therefore, we focus on measures specific to historically disadvantaged populations.

Based on a conceptual framework that views health disparities as partially driven by differential access to resources and exposures to hazards, we group the measures into four categories: social processes, environmental contaminants/exposures, bodyburdens of environmental contaminants, and health outcomes. We provide a few examples to illustrate each category, including residential segregation, PM_{2.5} exposures, blood mercury concentrations, and asthma morbidity and mortality. These measures and categories are derived from a review of environmental health disparities from several disciplines.

As a next step in a long-term effort to better understand the relationship between social disadvantage, environment, and health disparities, we hope that the proposed measures and literature review serve as a foundation for future monitoring of environmental health disparities. These efforts may aid community organizations, local agencies, scientists and policy makers in allocating resources and developing interventions.

© 2006 Elsevier Inc. All rights reserved.

Keywords: Environmental justice; Measures; Social disparities; Race

1. Introduction

There is continuing concern that minority and economically disadvantaged populations bear a disproportionate share of environmental exposures and related illnesses. These issues first gained national attention through publications such as the report by the Commission on Racial Justice of the United Church of Christ, *Toxic Waste*

and *Race in the United States (1987)*, and *Dumping in Dixie: Race, Class and Environmental Quality (1990)* by Dr. Robert Bullard. A 1990 University of Michigan conference on “Race and the Incidence of Environmental Hazards” pressured the US Environmental Protection Agency (EPA) to establish an Office of Environmental Equity (Brown, 1995). In 1994, in response to the growing environmental justice movement, President Clinton issued Executive Order 12898 requiring all federal agencies to work towards ending the disproportionate exposures of minority and poor people to many environmental hazards.

*Corresponding author. Fax: +1 202 564 2733.

E-mail address: payne-sturges.devon@epa.gov (D. Payne-Sturges).

A wide range of activities have been undertaken by various sectors of US society to address inequality in exposures to environmental hazards, including federally funded research programs on environmental hazards, initiatives to increase citizen involvement in environmental decisions, and community-based efforts to address local concerns about environmental hazards. However, it is difficult to evaluate the success of these efforts, especially with regard to eliminating the disparities between minority and majority communities. This is because the tools needed to understand and assess disparities have not been fully developed. The goal of this paper is to examine potential measures that might aid efforts to monitor health disparities.

Before proceeding further, we define the terms used in this paper. “*Health disparities*” and “*racial disparities*” will be used interchangeably to refer to gaps in morbidity and mortality between racial and ethnic groups. “*Ethnicity*” refers to the linguistically defined group of Hispanics, while “*race*” refers to the socially constructed groups specified by Directive 15 of the Office of Management and Budget, namely Black or African American, Asian American, American Indian or Alaska Native, Native Hawaiian or Other Pacific Islander, and White (US Office of Management and Budget, 1997). As noted by Directive 15 and numerous observers, racial and ethnic groups are social categories and not biological taxons. While we adopt the Directive 15 approach because federal and other data sets use these conventions, this approach’s limitations have been widely discussed (Bhopal et al., 2000; Kaplan and Bennett, 2003; LaVeist, 1994). The term “*environment*” encompasses the natural, built and social worlds. Thus, environmental influences are not limited to physical (e.g., radiation), chemical (e.g., lead), and biological (e.g., pathogens) agents, but also includes social stressors (e.g., poverty), institutional processes (e.g., housing policy), and resiliency factors (e.g., social capital). For the purpose of this paper, we define “*environmental health disparities*” as racial/ethnic and socioeconomic inequities in illness and exposures that are at least partially mediated by factors associated with the physical, social, and built environments.

Finally, we use the word “*measures*” in this document to encapsulate both the terms “*measures*” and “*indicators*.” Some have suggested that “*indicators*” denote an etiological process, whereas “*measures*” are more descriptive. Although we use the term “*measures*” we do not imply that the variables reviewed are merely of value for descriptive purposes. The use of a variable as a descriptive or analytic factor depends upon the research question. These designations challenge us to more precisely designate our conceptual assumptions and may have important policy implications. Since there is no consensus as to which variables are measures or indicators (especially with regards to issues surrounding race/ethnicity), we use the broader and more conservative label of “*measures*” in this discussion (Gordis, 2000; Maldonado and Greenland, 2002).

2. The need: tracking disparities in environmental health

The paucity of tools for measuring important elements of environmental health has been a concern since the early 1990s (Institute of Medicine, 1999; Northridge et al., 2003; Sexton, 1997; Sexton et al., 1993). Several observers (Bullard and Wright, 1993; Lee, 2002; Shepard, 2002) have speculated that the totality of environmental conditions—whether from exposure to chemical toxins or the availability of healthy food products or the opportunities for gainful employment—contributes to health. However, little empirical research has evaluated the relative weights of these factors. Because the field of research is still nascent, standardized ways of measuring environmental conditions, especially as relevant to ethnic minorities, are lacking. As a result, fundamental questions about the relationships between race, social class and the environment remain unanswered. The challenge is to find valid and reliable measures of environmental risk factors (exposures, susceptibilities, distribution of hazards) and health outcomes associated with environmental hazards that can be applied nationally. To more effectively address disparities, longitudinal data are needed to track health conditions and risk factors. One approach to begin to address these issues is the development of measures to track environmental health disparities.

3. Previous measures

The concept of health measures is not new to public health. Health measures are basic tools that public health practitioners use to characterize community well-being and assess trends in risk factors, mortality, and morbidity (Thacker and Berkelman, 1988; Thacker et al., 1988). These measures have been incorporated into national health planning activities such as Healthy People 2010, the guidebook for monitoring the public’s health. Goal 8 of Healthy People 2010 is to “promote health for all through a healthy environment.” Related to this goal are 30 objectives that include the assessment of ambient air quality, water quality, toxics and waste, healthy homes, infrastructure, and surveillance of environmental health conditions. Of these measures, five are constructed to describe conditions for racial and low-income populations: ambient air quality, lead and radon testing, blood lead levels of children, water quality, and sanitation in US–Mexico border communities. Healthy People 2010 also provides a cross-listing of goals/objectives related to the environment, including reductions in heart disease, respiratory diseases, low birth weight rates, kidney disease, and tobacco smoke, for which racial/ethnic stratifications of the data are presented. Health measures have been discussed in a variety of other reports as well (Centers for Disease Control, 2003; Pew Environmental Health Commission, 2000; Rothwell et al., 1991).

Recent applications of environmental health measures at a national level include EPA’s report *America’s Children*

and the Environment: A First View of Available Measures (Woodruff et al., 2000) and the second edition *America's Children and the Environment: Measures of Contaminants, Body Burdens, and Illnesses* (Woodruff et al., 2003), and EPA's *Draft Report on the Environment* (US Environmental Protection Agency, 2004) (see website www.epa.gov/indicators/roe/index.htm). *America's Children and the Environment* (ACE) presents data on trends in levels of environmental contaminants in air, water food, and soil; concentrations of contaminants measured in bodies of children and women; and childhood illnesses that may be influenced by exposure to environmental contaminants. The ACE report discusses racial/ethnic and socioeconomic disparities for five of 19 measures, with most found in Appendix A. Although these reports touch upon disparities, applications relevant to racial minorities and low socioeconomic groups are underdeveloped.

Because the development of environmental health measures has been undertaken by a variety of entities and published in separate reports, a broad understanding or public debate about measuring the environmental impact on the health of ethnic and racial minorities has not taken place to date. We seek to build upon this previous work by compiling the extant information relevant to the study and monitoring of environmental health disparities.

4. Our approach

Developing a parsimonious set of measures for environmental health disparities is a daunting task since the list of potential measures is nearly endless. For the prevention of illness and the promotion of public health, we need to track not only diseases, but also the risk factors for disease (Centers for Disease Control, 2003). The surveillance of health outcomes will allow for assessments of public health progress, and the surveillance of risk factors may allow for the study of etiological mechanisms and for the prediction of future trends. For example, identification of an increase in pesticide exposures (a risk factor) may suggest a future increase in the outcomes of unintentional poisonings.

In reviewing previously published measures, it quickly became clear that a first step would be to find a way to conceptualize and group measures. This would help provide some coherence to the abundance of measures available. Therefore, our first task was to review the literature and develop a framework from which to understand how environmental conditions may contribute to health disparities. We review the framework briefly in the next section, then describe criteria for selecting and identifying candidate measures, data sources and discuss applied quantitative approaches to construct relevant and informative measures to track environmental health disparities.

5. The framework

Previously, we had reviewed the recent scientific literature on health disparities, psychosocial stressors and resources, environmental justice, vulnerability/susceptibility to environmental exposures (e.g., pre-existing health status, occupational exposures), and past work by US EPA, HHS, and CDC (Gee and Payne-Sturges, 2004). The literature suggests that racial groups differ in health outcomes because of greater exposure to illness risk factors (Geronimus et al., 1999; Geronimus et al., 2001; Lee, 1993; Sexton et al., 1993; Williams, 1999; Williams and Collins, 2001; Williams and Yu, 1997), due at least in part to the fact that Whites and minorities often do not “work, live and play” in the same places (Lee, 2002). People of color are more likely to encounter high risk settings, including residence in high-poverty neighborhoods and employment in more hazardous occupations (Jargowsky, 1997; Williams, 1999; Wilson, 1996). Thus, differences in *settings* contribute to increased risk for illness among minority populations. The literature review suggested a framework (Fig. 1) that views health disparities as partially driven by differential access to resources and exposures to hazards. In particular, one important observation is that monitoring efforts should not be limited to physical and chemical toxicants, but also include the social stressors that may confer additional vulnerability and potential resources which might counterbalance health risks. In the present article, we show the next sequence to this work by presenting a summary of candidate measures informed by the framework.

6. Criteria for selection of candidate measures

Another major challenge lies in defining the types of measures that would be most useful for environmental health disparities. We adopted three criteria for choosing measures: (1) theoretical/empirical relevancy to the health of minority and economically disadvantaged populations; (2) currency and national scope; (3) potential for future research and intervention.

First, we focused on measures that may be associated with the health of racial/ethnic and low-income populations. This is a minimal criteria granted that our interest is in health disparities. As a corollary, this requires that measures have the potential to be disaggregated by race/ethnicity and socioeconomic position. We emphasize that capturing race/ethnicity and socioeconomic position is critical since disparities cannot be monitored if the data are aggregated by these characteristics. Also, to the extent possible, we examined measures that can be further disaggregated within racial/ethnic groups (e.g., between Mexicans and Cubans, within Hispanics).

Second, we focused on measures that exist currently and are available at the national level. Ensuring that the data are currently allowed for the establishment of benchmarks to examine future trends. Having data at the national level

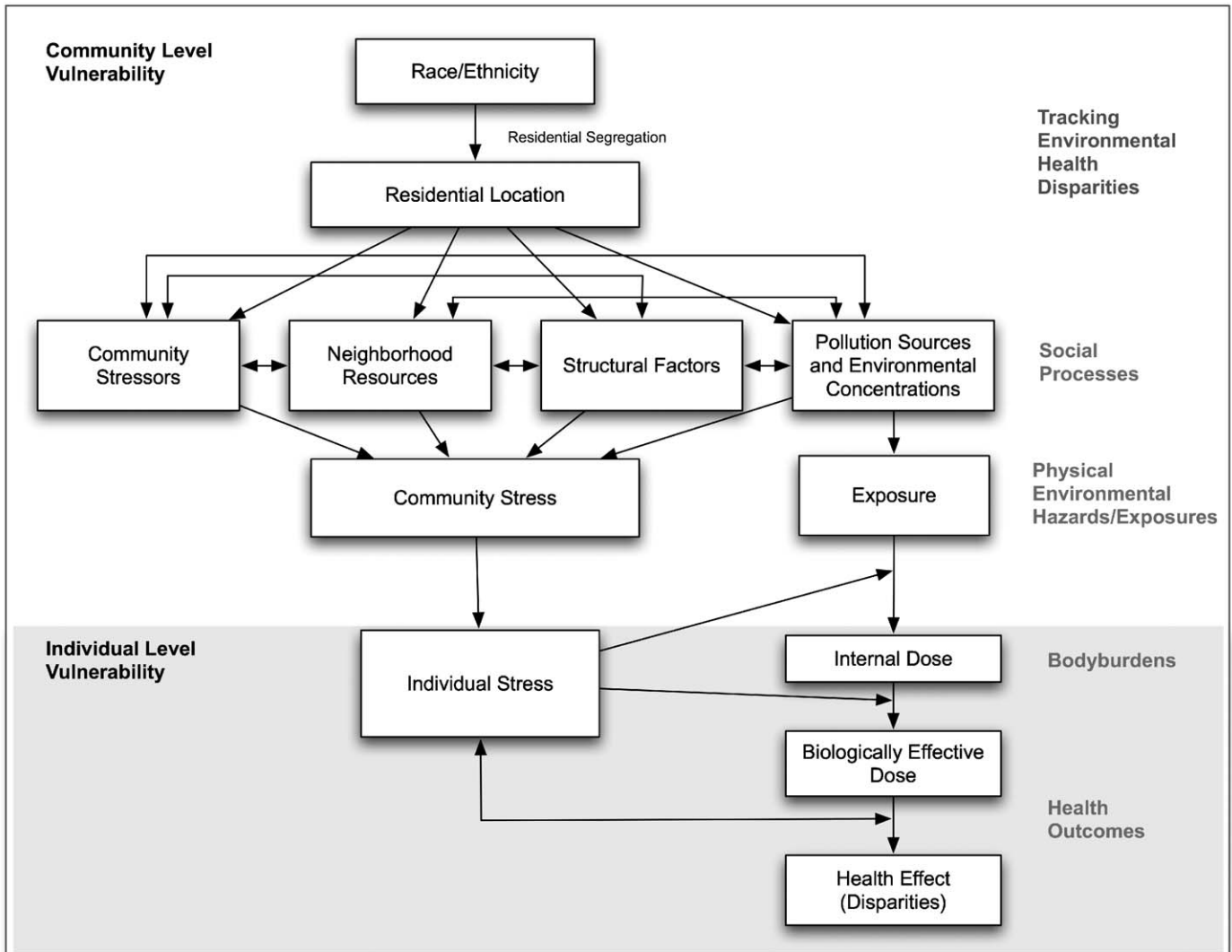


Fig. 1. Framework for Understanding racial/ethnic disparities in environmental health.

obviously allows for national estimates of health and health risks, but also, allows local communities a point of comparison.

Third, we also examined measures that have potential for future research and intervention. One of the purposes of environmental health measures proposed by the Pew Environmental Health Commission, CDC, and the Council of State and Territorial Epidemiologists (CSTE) is to facilitate prevention of known or suspected adverse public health events associated with environmental exposures and to detect new adverse health events associated with environmental exposures (Pew Environmental Health Commission, 2000; Rothwell et al., 1991; Thacker et al., 1996). In identifying the health outcomes for our candidate measures we took this preventive view since there are few diseases for which clear environmental etiologies have been established. Identifying specific environmental causes of disease is often hampered by long latency; lack of unique markers; and multiple causes. Although disease outcomes have been linked to many biological and physical agents,

very few of the millions of known chemical agents have been studied adequately (Thacker et al., 1996). Several of the health endpoints identified are conditions for which environmental exposures have been implicated (Centers for Disease Control, 2003; Kjellstrom and Corvalan, 1995; Pew Environmental Health Commission, 2000; Silbergeld and Tonat, 1994). We also identified pre-existing health conditions that may be exacerbated by exposures to environmental pollutants or render people susceptible/vulnerable to environmental pollutants (Pew Environmental Health Commission, 2000; Rios et al., 1993).

We also examined measures that might be useful for analytic purposes. Although the monitoring of health outcomes is of intrinsic interest, there will be great value added in tracking measures that might be useful to test hypotheses. For example, emerging research suggests that residential segregation may be part of the causal pathway for health disparities in cancer (see Morello-Frosch and Lopez, in press). By tracking these measures simultaneously, we may be able to test competing hypotheses and

get closer to understanding cumulative risks. Additionally, we examined measures from a multi-level perspective, considering factors at a very proximal level (e.g., individual-level body burdens of lead) to local level (e.g., housing quality) to more macro levels (e.g., income inequality). A fuller discussion of multi-level approaches can be found in the accompanying article by Soobader and colleagues (Soobader et al., in press).

7. Candidate measures

Based on our review of the scientific literature and identification of national health and environmental data sources, we identified 112 candidate environmental public health measures to assess progress and status of environmental health of minority and low-income populations at the national level. Embedded in the concept of the environment are risks associated with both the physical (e.g., soil lead) and social environment (e.g., the distribution of wealth). Recent research has also emphasized measures of bodyburdens of chemical toxicants. Following this reasoning and building on the categories proposed by Thacker et al. (1996), we organize our measures into four broad areas: *social processes, physical environmental hazards/exposures, bodyburdens, and health outcomes*. Table 1 presents definitions for each category. The four categories of measures are complementary and it may not be necessary (or even possible) to monitor or track all four for a particular issue. This heuristic may be helpful in organizing research efforts for a given issue, but obviously, potential causal connections will differ by research area. Further, it is important to remember that exposure to one environmental contaminant may lead to multiple health impacts, and a particular health impact may derive from cumulative exposure to many different environmental contaminants interacting with host vulnerabilities and other underlying causes.

Presented in Table 2 is a general overview of the candidate measures. The data to construct these measures may be obtained from national-scale databases/data sets, such as the National Health Interview Survey, National Health Examination and Nutrition Survey, National Science Foundation's General Social Survey, US Census, American Community Survey, American Housing Survey,

EPA's Aerometric Information Retrieval System, US Department of Agriculture's Continuing Survey of Food Intake by Individuals (CSFII), National Agricultural Statistics Service, and US EPA's Resource Conservation and Recovery Information Systems. For more details on the types of information that can be obtained, please refer to CDC's report *Environmental Public Health Indicators* (Centers for Disease Control, 2003).

We discuss a few example candidate measures below which highlight concepts in our framework. Asthma is chosen as a sample measure for health outcomes because there are racial/ethnic and socioeconomic differences in both prevalence and mortality rates; national and contemporary data are available; extant research suggests that asthma is associated with the social (e.g., psychosocial stress), physical (e.g., ambient air pollution and environmental tobacco smoke) and built (e.g., housing quality) environments. Measure for residential racial segregation is chosen as a sample measure for social processes because segregation has been associated with disparities in mortality and exposures to environmental hazards and are currently available at a national level. Measures on criteria air pollutants are chosen as an indicator for physical hazards because current research shows disparities in population exposures to air pollutants and links between exposures to particulate matter pollution with medical conditions such as asthma and heart disease. Finally the example bodyburden indicator is blood mercury levels in women of childbearing age, an emerging area for racial/ethnic disparities in environmental exposures. Details on the data sources and methodology for constructing the example measures for asthma, air quality and blood mercury are presented in Appendix A. A draft report with a more detailed description of the other indicators shown in Table 2 is available from the authors upon request.

8. Health outcomes—respiratory illnesses—asthma prevalence and mortality

Epidemiologic and laboratory studies demonstrate that ambient air pollutants (e.g., particulate matter, ozone, sulfur dioxide, and nitrogen dioxide) contribute to various respiratory problems including bronchitis, emphysema,

Table 1
Categories of indicators

Social process indicators or measures: Psychosocial factors that may directly or indirectly lead to illness. These include factors operating at the interpersonal (e.g. socioeconomic position) as well as societal level (e.g. residential racial segregation).

Physical environmental hazards/exposure indicators: condition or activities that identify the potential for or occurrence of exposure to an environmental contaminant or hazardous condition (e.g., toxic chemical agents, physical agents, biomechanical stressors, as well as biological agents).

Bodyburden indicators: biological markers in tissue or fluid that identify the presence of a substance or combination of substances that impact human health.

Health outcome indicators: Diseases or conditions that may be related to exposure to an environmental hazard (or environmental pollutant).

Table 2
Overview of candidate indicators/measures

Social processes	Physical environmental hazards/exposures
<i>Residential segregation</i>	<i>Outdoor air pollution</i>
Dissimilarity	Exposure to criteria air pollutants
Isolation	Estimated noncancer risks from air pollutant exposures
Minority composition	Estimated cumulative cancer risk from air pollutant exposure
Ethnic churning	<i>Indoor air pollution</i>
<i>Community stressors</i>	Smoking
Crowding & density	ETS exposure
Crime	Radon
Noise	Lead hazards
Lack of control	Substandard quality housing
Household poverty	Jurisdictions with anti-smoking ordinances for public spaces
Stigma	<i>Drinking water and ambient water quality</i>
Family income	Population served by public water systems not meeting standards
Employment opportunities	Migrant worker camps water systems not meeting standards
Housing quality	US–Mexico border community water systems
Living standards	Access to recreational waters meeting standards
Income inequality	Populations in areas with high-quality watersheds
<i>Neighborhood resources</i>	
Social capital	Populations with in states with fish advisories
Voter participation	Fish consumption patterns
Neighborhood quality	<i>Pesticides</i>
Faith-based institutions	Foods with detectable pesticide residues
Recreational facilities: parks, etc.	Pesticide related illnesses among agricultural workers
Greenways	
Neighborhood associations	Reported pesticide use by farmers
Schools, libraries	Estimated pesticide exposure through fish consumption/subsistence fishing
Cultural institutions	Reports of indoor pesticide use
<i>Structural factors</i>	
Zoning policies	<i>Land contaminants and waste sites</i>
Governance structure	Population living within 1 and 3 mile radii of hazardous waste sites and landfills
Taxation system	Population living within 1 and 3 mile radii of
Regulatory environment	Superfund sites designated as public health
Physical constraints: temperature, elevation, humidity	Hazard
Bodyburden	Health outcomes
Lead (in children and adult workers)	
Cadmium	<i>Life expectancy</i>
Mercury (in women of childbearing age)	<i>Mortality</i>
Arsenic	All cause mortality
Cotinine	Cancer mortality
OP pesticides	Asthma mortality
Pyrethroid pesticides	Infant mortality
PCBs	<i>Cancer</i>
DDT/DDE	Lung cancer
Estimated pesticide doses based on body burden measures	Bladder cancer
	Leukemia
	Breast cancer
	<i>Respiratory illnesses</i>
	Hospitalization rates for respiratory illnesses
	Sarcoidosis
	Asthma
	<i>Other chronic diseases</i>
	Heart disease
	Kidney disease
	Liver disease
	Hypertension
	Diabetes
	Neurological diseases
	Lupus
	<i>Children's health</i>
	Cancer in children
	Low birth weight
	Birth defects
	Childhood asthma
	<i>Infectious diseases</i>
	Foodborne and waterborne illnesses

and asthma (American Lung Association, 2001; McConnell et al., 2002; McConnell et al., 1999). For example, numerous reports have documented significant increases in asthma morbidity and mortality in US beginning in the 1970s, with African Americans disproportionately affected (Akinbami and Schoendorf, 2002; Mannino et al., 2002; Ostro et al., 2001). African American and Puerto Ricans have the highest rates of active and lifetime asthma compared with other racial/ethnic groups (Carter-Pokras and Gergen, 1993). These racial disparities in asthma prevalence do not appear to be explained by socioeconomic status (Gergen et al., 1988).

Pre-existing health conditions may lead to greater vulnerability. For example, epidemiological studies suggest that individuals already suffering from cardiopulmonary conditions, including asthma, chronic obstructive pulmonary disease (COPD) and cardiovascular diseases are at increased risk for developing adverse health outcomes from exposure to air pollution (American Lung Association, 2001). Ostro et al. (2001) found that air pollution (PM, ozone) was associated with exacerbation of asthma symptoms in a group of African-American children in Los Angeles. McConnell et al. (1999) suggest that children with a prior diagnosis of asthma are more likely to develop persistent lower respiratory tract symptoms when exposed to air pollution in Southern California. In a study evaluating the effects of low air pollution levels, McConnell et al. (2002) found that children with asthma were very vulnerable to ozone at levels that are lower than current EPA standards.

Asthma prevalence and mortality are candidate measures for environmental tracking. Asthma prevalence is an important environmental health measure, as exacerbation of asthma, asthma prevalence, and hospitalization rates have been linked to exposure to air pollution, while asthma mortality reflects the influence of socioeconomic factors that also contribute to health disparities such as access to quality health care and effective preventative medical therapy (Akinbami and Schoendorf, 2002; Delfino et al., 2003; O'Neill et al., 2003; Schwartz et al., 1993). Presented in Figs. 2 and 3 are asthma attack prevalence (individuals who had previously received diagnosis of asthma and who had 1 or more asthma attacks in the past 12 months) and current asthma prevalence rates by race/ethnicity. For the period 1997–2004 there appears to be a decreasing trend in asthma attacks, however, Black Non-Hispanics and American Indian/Alaska Natives generally have highest rates compare to the other racial/ethnic groups (Fig. 2). Similarly measure for current asthma (Fig. 3) show declines if not leveling of rates for 2001–2004 across the racial/ethnic groups with Blacks and Native Americans with highest rates around 10%. Presented in Fig. 4 is the annual rate of asthma deaths (age-adjusted) from 1999 to 2002 by race. While death rates for all groups are declining, Blacks continue to have highest asthma mortality rate, twice that of Whites.

9. Social processes—residential racial segregation

Residential segregation refers to the process whereby members of racial and ethnic groups live apart from one another. The most common measure of segregation is the Index of Dissimilarity (D), which can be calculated from public release data from the US Census. D is scored from zero (complete integration) to 100 (complete segregation) and can be interpreted as the proportion of minorities (or Whites) who would have to move in order to integrate a given metropolitan area (Massey and Denton, 1993) (formulas for D can be found in Massey and Denton, 1993). In the year 2000, about two-thirds of all African Americans, and roughly half of all Hispanics and Asian Americans and Pacific Islanders would have to interchange residences with White counterparts in order to fully integrate metropolitan areas in the United States (Massey, 2001).

Segregation is associated with a variety of health outcomes. Studies have documented a positive association between segregation and infant mortality (Laveist, 1989; Laveist, 1993; Polednak, 1991; Polednak, 1993), adult mortality (Fang et al., 1998; Hart et al., 1998; Polednak, 1993), life expectancy (Potter, 1991), homicide (Shihadeh and Flynn, 1996), all cause and cancer mortality (Collins and Williams, 1999), and tuberculosis (Acevedo-Garcia, 2001). Lopez (2002) found that residential segregation was associated with model estimates of air toxic exposures, even after controlling for poverty, population density, neighborhood industry, and vehicular use. Morello-Frosch and Jesdale (2006) reported associations between segregation and estimated cancer risks associated with ambient air toxics. In multi-level studies, segregation is associated with self-rated health (Subramanian et al., 2005).

It has been hypothesized that segregation concentrates social disadvantage (e.g., poverty), which in turn leads to health outcomes (Gee, 2002; Massey and Denton, 1993; Williams and Collins, 2001). Compared with Whites, minorities are overrepresented in neighborhoods with diminishing and constrained economic opportunities (Jargowsky, 1997; Wilson, 1987). For example, in Los Angeles in 1990, only 4.9% of Blacks lived in high job growth areas, compared with 52.3% of Whites (Pastor, 2001). The concentration of minorities in poor areas contributes in part to socioeconomic differences between Blacks and Whites (Massey and Denton, 1993). Cutler and Glaeser (1997) estimated that a one standard deviation decrease in segregation (13%) would eliminate one-third of the Black-White differences in education and employment. Thus, segregation may be partly responsible for the production of class differences between African Americans and Whites (Williams and Collins, 2001). A fuller discussion of segregation and environmental health disparities can be found in Gee and Payne-Sturges (2004) and in the accompanying article by Morello-Frosch and Lopez. The degree of residential segregation can serve as a measure of general community vulnerability and can be combined with

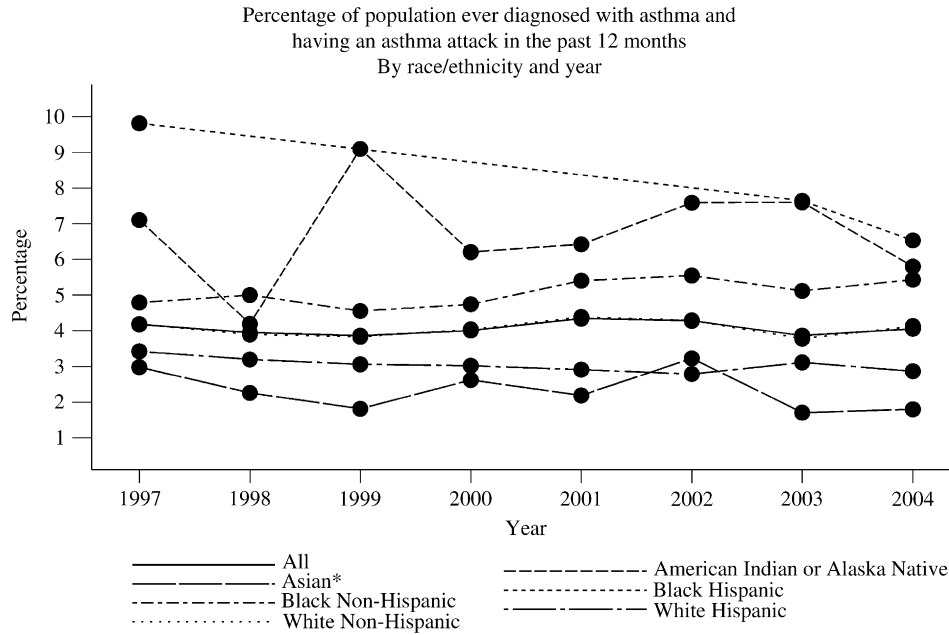


Fig. 2. Percentage of population ever diagnosed with asthma and having an asthma attack in the past 12 months by race/ethnicity and year.

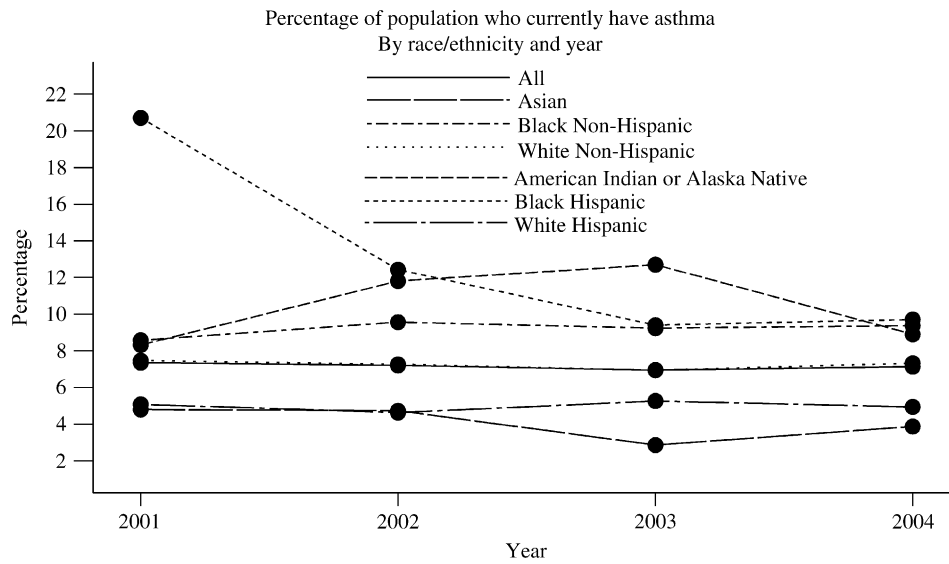


Fig. 3. Percentage of population who currently have asthma by race/ethnicity and year.

data on environmental pollutants (e.g., ambient air pollutants) (Lopez, 2002; Morello-Frosch and Jesdale, 2006).

Fig. 5 shows dissimilarity between Whites and other minority groups (African Americans, Asian Americans, Hispanics, Native Americans, and Pacific Islanders) for metropolitan areas. Segregation for African Americans has declined slightly between 1980 and 2000. However, at a national dissimilarity value of 64, African American segregation remains extremely high (Massey, 2001). Segregation for Hispanics, Asians remains moderately high and stable between 1980 and 2000. Segregation for American

Indians is relatively low and has declined during this period.

10. Physical environmental hazards/exposures—ambient air pollution—criteria air pollutants

Air pollution is an important public health problem, associated with premature death, cancer and long-term damage to the respiratory and cardiovascular systems, psychological distress, and negative behavior (American Lung Association, 2001; Evans, 1994; Evans et al., 1988; Lundberg, 1996; Sexton et al., 1993; US Department of

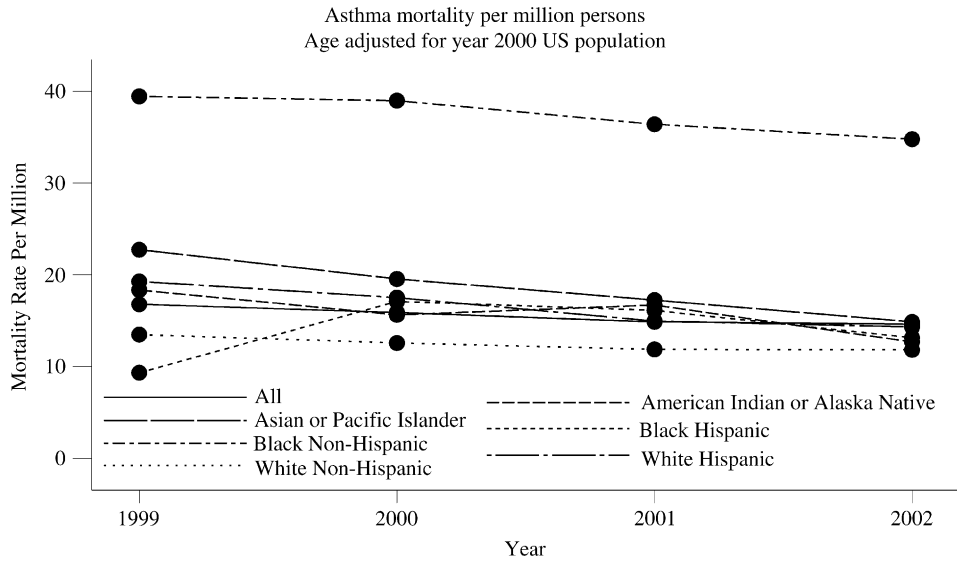


Fig. 4. Asthma mortality per million persons age adjusted for year 2000 US population.

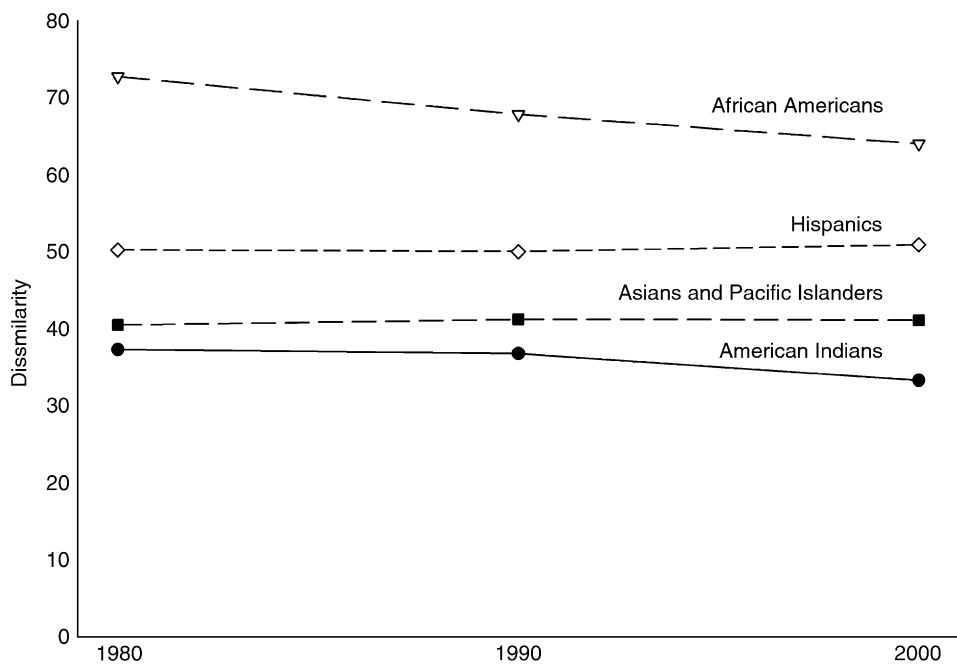


Fig. 5. Dissimilarity of ethnic minorities to whites, United States 1980–2000.

Health and Human Services, 2004; Woodruff et al., 2003). These are some of the same illnesses for which health disparities have been observed (Gwynn and Thurston, 2001; Pope et al., 2004; Schwartz, 1999; Schwartz and Morris, 1995; US Department of Health and Human Services, 2004; Zanobetti et al., 2000).

Spatial patterns of air pollution have been linked to land-use decisions (Maantay, 2001). Environmental justice activists and communities have often raised concerns about the potential for disproportionate exposure to air pollution among disadvantaged or racial/ethnic minority populations in urban areas due to the proximity of

pollution emission sources, such as bus depots, truck distribution facilities, high-volume roadways, waste treatment and transfer stations, and major industrial sources to residential areas (Maantay, 2001).

Previous analyses of disparities in potential exposure to outdoor air pollution have focused on criteria air pollutants (lead, ozone, particulate matter, nitrogen dioxide, sulfur dioxides, and carbon monoxide) (Wernette and Nieves, 1992). EPA has established National Ambient Air Quality Standards (NAAQS), permissible ambient concentration levels, for these 6 pollutants. The standards are designed to protect the public from adverse health

effects that can occur after either short-term exposure (e.g., 1- and 8-h standards for carbon monoxide) or long-term exposure (e.g., 1-year standard for nitrogen dioxide).

For these criteria air pollutants, candidate measures include the percentages of US population by race/ethnicity/poverty living in counties in which air quality standards were exceeded for ozone, CO, PM, sulfur dioxide, nitrogen dioxide and lead standards. Figs. 6–8 show data for one of the candidate measures, trends in the proportion of population affected by race/ethnicity and poverty group for PM_{2.5} 24-h standard of 65 µg/m³. PM_{2.5}, particulate matter with particle size diameter of 2.5 µm or smaller, considered fine particles, are able to travel deeply into the respiratory tract, reaching the lungs. Scientific studies have linked increases in daily PM_{2.5} exposure with increased respiratory and cardiovascular hospital admissions, emergency department visits and deaths (US Environmental Protection Agency, 2005). Recent studies suggest that long-term exposure to particulate matter may be associated with increased rates of bronchitis and reduced lung function. People with breathing and heart problems, children, and the elderly may be particularly sensitive to PM_{2.5} (US Environmental Protection Agency, 2005).

The trend graph Fig. 6 shows distinct patterns of populations potentially impacted by PM_{2.5} concentrations, with Asian and Pacific Islanders showing the greatest proportions affected and White with the lowest. The proportions of Asian or Pacific Islanders and of White Hispanics living in counties exceeding the PM_{2.5} standard are about double those for other race/ethnicity groups (approximately 30% and 15% in 1999–2001 and 2003–2004). The affected population was significantly greater in 2002 for all race/ethnicity groups, which may

be attributable to meteorological conditions particularly conducive to high particulate matter concentrations. In 2002, the percentage of Black Hispanics affected jumped to nearly 50%, indicating that the counties newly affected in 2002 had large Black Hispanic populations. The number of counties affected went from 47 in 2001 to 74 in 2002, including 24 additional counties in New Jersey and New York. Among these were the New York counties of Bronx, Kings, Queens, and New York, which alone had a combined Black Hispanic population of 415,000 in 2002—approximately 26% of the 2002 US Black Hispanic total of 1.6 million. For comparison the 2002 US total population of Black non-Hispanics was 36 million.

The term Hispanic is often used for comparison between racial and ethnic groups. However, this term usually lumps together many different groups of people, which may mask intra-Hispanic differences. To explore this further, we present the same data on PM_{2.5}, but for all the groups that identify Hispanic. As shown in Fig. 7, among the Hispanic groups, Mexican Americans have the larger proportion of population, 40%, living in counties where ambient concentrations of PM_{2.5} exceed the 24-h standard in comparison to other Hispanic groups. We are able to look for intra-Hispanic differences because this measure is geographically based and the Census data, which provide population counts for these ethnic groups, can be linked to the counties.

Additionally, Fig. 8 presents data for each racial/ethnic group by poverty status. Poverty threshold levels in 1999 were used and varied from \$ 8607 (one person under 65) to \$36,897 (9 or more persons with no related children). We provide data for below poverty, between 100% and 200% poverty (near poor) and above 200% poverty. As discussed above, Asians, Pacific Islanders, and Hispanics all have

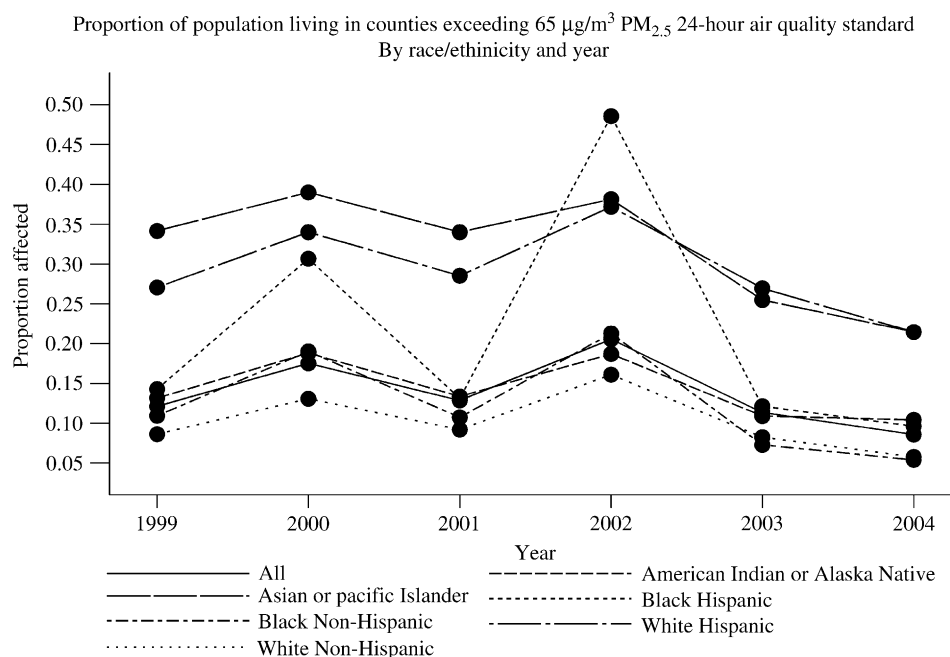


Fig. 6. Proportion of population living in counties exceeding 65 µg/m³ PM_{2.5} 24-h air quality standard by race/ethnicity and year.

Proportion of population living in counties exceeding 65 µg/m³ PM_{2.5} 24-hour air quality standard
For Hispanic populations
Based on Census 2000 county population estimates and EPA AQS ambient air quality data

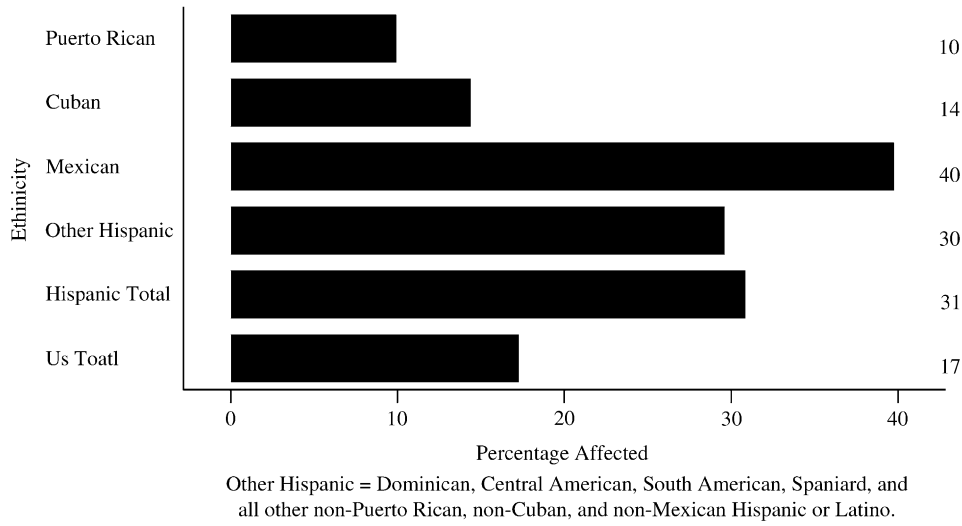
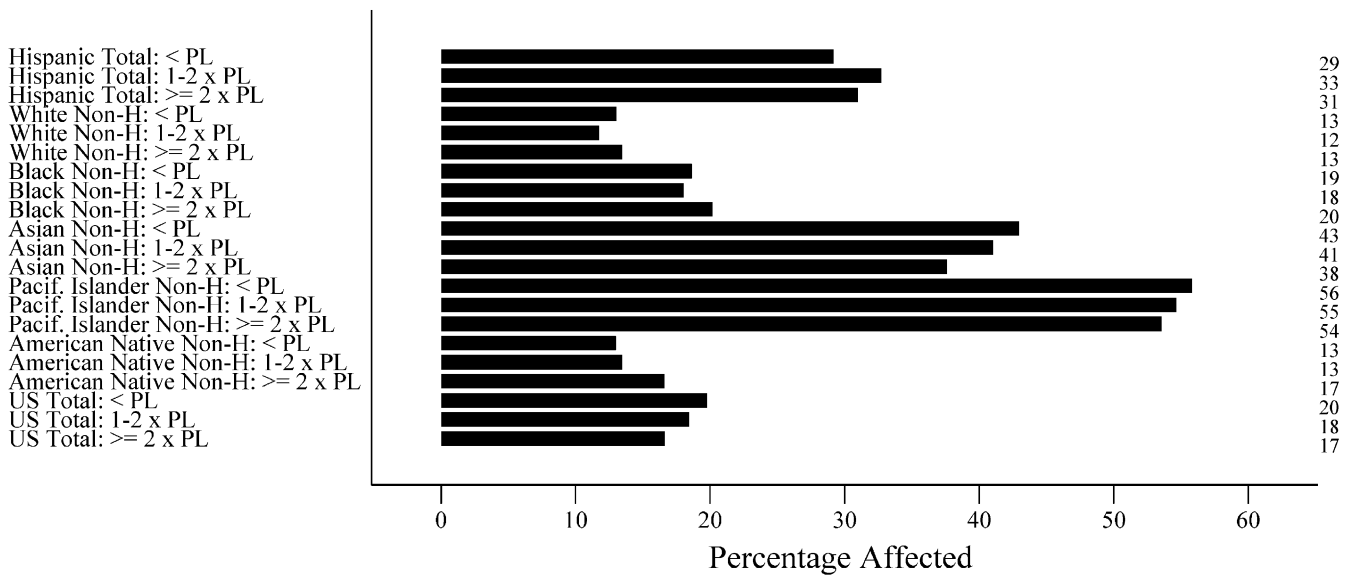


Fig. 7. Proportion of population living in counties exceeding 65 µg/m³ PM_{2.5} 24-h air quality standard for Hispanic populations.

Proportion of population living in counties exceeding 65 µg/m³ PM_{2.5} 24-hour air quality standard
By race/ethnicity and income (multiple of poverty level, PL)
Based on Census 2000 county population estimates and EPA AQS ambient air quality data



Based on family income and poverty levels for 1999.

The Poverty Level (PL) depends upon total family size, number of children under 18, and, for families of 1 or 2, whether or not the Reference Person is under 65.

Poverty Levels range from an average of \$8,501 for a family of one to \$34,417 for a family of nine or more.

'< PL' = 'Income Below Poverty Level'

'1-2 x PL' = 'Income Between 100 % and 200 % of Poverty Level'

'≥ 2 x PL' = 'Income At Least 200 % of Poverty Level'

Fig. 8. Proportion of population living in counties exceeding 65 µg/m³ PM_{2.5} 24-h air quality standard by race/ethnicity and income.

relatively large affected population proportions. For the US as a whole, those with higher incomes have a slightly lower tendency to live in counties exceeding

the PM_{2.5} standard: <PL 14.7%; 1–2 × PL 14.2%; ≥ 2 × PL 12.6%. However, this pattern does not apply consistently for all race/ethnicity groups, and the

proportion affected depends much more on race/ethnicity than on poverty.

The measures for criteria air pollutants illustrate the importance of examining the assumption of within-group homogeneity. That is, subgroups within a given racial/ethnic group (e.g., Mexicans within Hispanics) may differ in terms of risk exposure. These subgroups can include ethnic origin (e.g., Mexicans, Cubans) and socioeconomic position, as well as other important dimensions (e.g., age, gender). Although these principles are widely recognized, they are not always implemented in national tracking efforts. Subgroups are often excluded because their samples are often too small for reliable analyses. However, granted continuing recognition that these group differences are meaningful, it will be important to consider oversampling subgroups.

11. Bodyburden—mercury

Biological monitoring or biomonitoring is the measurement of environmental contaminants or their metabolites either in tissues (e.g., blood, serum or plasma, placenta hair, nails), secretions (e.g., breast milk, urine, feces), expired air, or any combination of these, in order to evaluate exposure and illness risk compared to an appropriate reference (Maroni et al., 2000). Measurements of the levels of pollutants in humans provide direct information about exposures to environmental contaminants.

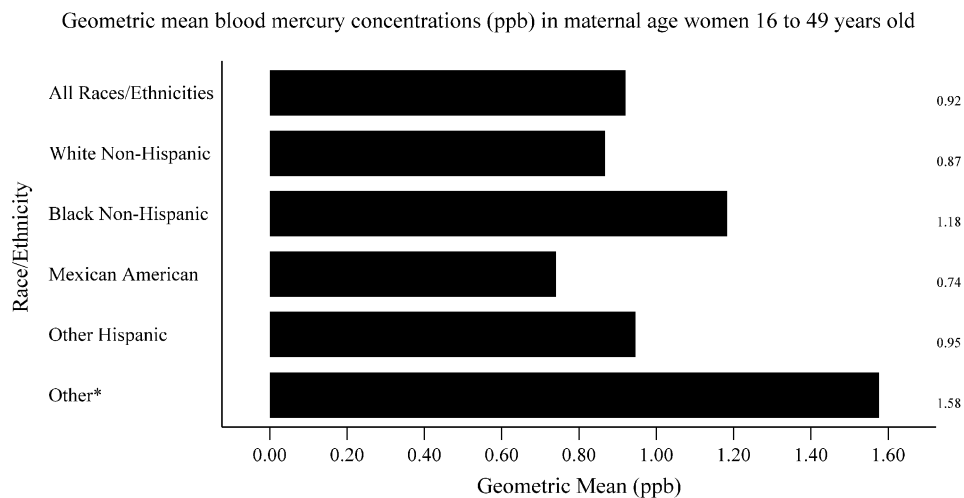
While one of the most well-known example of a chemical that has been monitored by measuring absorption into human tissue and shows persistent racial/ethnic and income disparities is blood lead levels (we include blood lead in our list of candidate measures), blood mercury levels in women of childbearing age are emerging as an important environmental health disparities issue. Mercury

is a highly persistent, highly bioaccumulative and toxic pollutant. Human exposure to mercury occurs mainly through consuming contaminated fish and shellfish. Studies show that subsistence fishing is more common among racial/ethnic minorities and minorities are potentially more exposed to contaminants found in fish such as methylmercury (Burger, 2002; Burger et al., 2002; Burger et al., 2001; Burger et al., 1999a; Burger et al., 1999b; Corburn, 2002; Hightower et al., 2006).

Mercury can cause health problems at even low levels of exposure, especially neurological damage to fetuses and children, who are thought to be more sensitive to methylmercury’s effects because of the enhanced vulnerability of the developing nervous system. Health effects of concern in children include learning deficits. Recent studies show that mercury exposure can also have adverse health effects on the nervous, immune and cardiovascular systems of adults as well as children (Landrigan et al., 1994; Woodruff et al., 2003).

Presented in Figs. 9 and 10 are examples of proposed measures for bodyburden of mercury. Biomonitoring data from the National Health and Nutrition Examination Survey (NHANES) carried out by the Centers for Disease Control and Prevention (CDC) and National Center for Health Statistics (NCEH) is the primary data source.

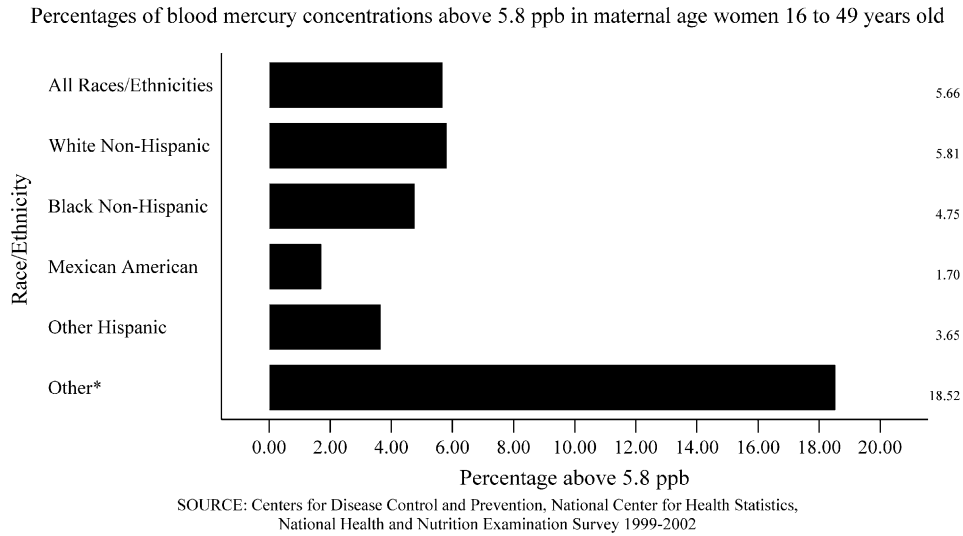
As shown in Fig. 9, from the 1999–2002 NHANES survey, women who self-identified as Asian/Pacific Islander, Native American or non-Hispanics reporting multi-race without specifying a main race other than Black or White had the highest geometric mean (1.58 ppb) of blood mercury concentrations of maternal age women 16–49 years. Further, as shown in Fig. 10, this same group of women had a higher prevalence (18.52%) of elevated blood mercury (levels above 5.8 ppb) in comparison to all the other racial/ethnic groups in the survey. About 8% of all



SOURCE: Centers for Disease Control and Prevention, National Center for Health Statistics, National Health and Nutrition Examination Survey 1999-2002

* "Other" includes Asian; Pacific Islander; Native American; those non-Hispanics reporting multi-racial without specifying a main race other than White or Black; and those with a missing value for race/ethnicity.

Fig. 9. Geometric mean blood mercury concentrations (ppb) in maternal age women 16–49 years old.



* "Other" includes Asian; Pacific Islander; Native American; those non-Hispanics reporting multi-racial without specifying a main race other than White or Black; and those with a missing value for race/ethnicity.

Fig. 10. Percentages of blood mercury concentrations above 5.8 ppb in maternal age women 16–49 years old.

US maternal age women have blood mercury levels >5.8 ppb, a level considered by EPA to cause increase risk of adverse health effects to babies (Woodruff et al., 2003). Asians, Pacific Islanders, and Native Americans are a potentially high-risk group for dietary exposure to methylmercury through fish consumption (Hightower et al., 2006). These measures call attention to the need for lowering risk of methylmercury exposure in this population.

In addition to monitoring blood lead and mercury levels through NHANES, The National Center for Environmental Health (NCEH) at the CDC measures the bodyburdens of the American public for many other environmental contaminants. Results from these surveys are now being published in a biannual report, The National Report on Human Exposure to Environmental Chemicals. Racial and ethnic disparities in bodyburden have also been noted for other chemicals (e.g., pesticides, dioxins and PCBs and phthalates and cumulative chemical exposures (Environmental Justice and Health Union, 2004). The differences in bodyburdens are important to understand because they may reflect differences in activities or conditions contributing to exposure that could be altered to reduce/eliminate exposure. While the CDC cautions not to interpret the presence of a chemical in the blood or urine as a marker of disease, bodyburden data do indicate that some exposure has taken place. When there is sufficient toxicological information, some interpretation of risk based on the bodyburden data may be possible (e.g., lead, mercury) (National Center for Environmental Health, 2003; Woodruff et al., 2003). An important limitation of these data is that they are cross-sectional, and do not provide information to determine the time or the duration of exposure.

12. Discussion of challenges and next steps

Eliminating health disparities is an overarching goal for improving our nation’s health. Increasingly, scientists, community members, policymakers and advocates have called for a broader and multi-faceted understanding of the risks that may contribute to illness. Current research suggests that health disparities are produced by both environmental hazards and psychosocial stressors (Institute of Medicine, 1999).

We propose the development of a comprehensive set of environmental health measures to assess/monitor environmental contributors to racial, ethnic and class disparities in health. We expanded on EPA’s report *America’s Children and the Environment* by including measures of social processes (e.g., segregation) that may be useful in understanding environmental health disparities, and by highlighting emerging issues that may be potential avenues for future research. Based on review of literature and available national data sets, we identified 112 measures that fall into the following topic areas: residential segregation, community stressors (income inequality), neighborhood resources, structural factors, outdoor air, indoor air, ambient water and drinking water quality, pesticides land contaminants, bodyburden and health outcomes. Some of the proposed measures have been previously reported by other agencies and organizations. For example, several Healthy People 2010 objectives on environmental health, cancer, respiratory disease, and tobacco overlap with our candidate measures. Because Healthy People 2010 has made the elimination of health disparities—the gap in morbidity and mortality between social groups (e.g., racial/ethnic minorities and low-income populations)—a top national priority, this overlap increases the relevance of

the proposed measures and provides focal points for federal interagency activity. In addition, based on our framework we present a number of novel measures that integrate social processes with environmental health conditions or highlight new and emerging environmental health issues. These candidate measures are presented to *stimulate dialogue* on the choice of appropriate measures, feasible and defensible methodologies, and elucidation of etiological mechanisms. Undoubtedly, these issues will be best resolved through public debate with community members, scientists, and policymakers.

In developing the candidate measures, we highlighted some, but not all of the scientific questions that should be addressed. Therefore, in moving forward, we suggest engaging stakeholders (federal agencies, environmental health and health disparities researchers, policy makers, community health advocates, etc.) to review the full suite of proposed measures to assist in: (1) addressing scientific details (e.g., quality of databases, interpretation of measures); (2) prioritizing/ranking of measures in terms of usability, importance to environmental health and health disparities, scientific validity and reliability, time scale of data, and geographic and population coverage; (3) identifying additional measures and alternative approaches to construct measures (measures that track the gap between groups using rate ratios or absolute differences); (4) identifying data sources to improve population coverage for Native Americans and emerging subgroups (e.g., Arab Americans, African immigrants, multi-racial Americans); (5) incorporating occupational exposures and health; (6) addressing the role of risk perception, and (7) identifying alternatives or additional approaches to express socio-economic position (e.g., income, income inequality and neighborhood poverty) disparities and relevant social processes for the environmental health measures.

While our candidate measures focus on national level data, the importance of data aggregated on smaller scales (e.g., regional or local levels) should not be overlooked. National level data may mask important “hot spots” in exposures and health outcomes that may be important at local levels. More local data is needed to provide comparable data across localities that could be aggregated to inform national efforts to eliminate disparities. Local data would enable comparisons between comparable (in size and resources) local areas and would bolster efforts to both incorporate community-level factors and intervene at multiple levels. Several of our candidate measures are amenable to local scaling such as those measures based on census data. However, other available data sets we identified are based on health surveys that may not scale to other levels. One potential mechanism to address this limitation is the current effort by the CDC to establish an environmental public health tracking (EPHT) network (<http://www.cdc.gov/nceh/tracking/>). One of the goals for this network is to provide more locally relevant data. Stakeholders could be engaged to develop core environmental health disparities measures that are comparable

with national data and yet be flexible enough to incorporate factors unique to individual communities. An important avenue that should be explored is the potential for multi-level analysis of existing environmental data which may be accomplished by merging environmental data sets with existing individual-level data sets (Diez Roux, 2002). By considering multiple levels of analysis, we can more fully evaluate which exposures may potentially lead to which outcomes, which exposures might *not* be associated with illness, and how these exposure pathways might generate not only illness, but produce health disparities.

The proposed measures can facilitate the tracking of environmental health status of disadvantaged populations; aid in assessing the contribution of the environment to health disparities; and inform discussion among policy makers and the public. Specifically for EPA, these measures will provide the Office of Environmental Justice with tools for identifying and communicating environmental justice issues within a public health context. In addition, the measures will provide critical baseline information for the federal interagency task force on environmental justice and health disparities, co-chaired by Department of Health and Human Services (HHS) Office of Minority Health and EPA’s Office of Environmental Justice. Finally, these measures may provide important information for EPA to consider in conducting risk assessments (population vulnerability has recently been identified by National Environmental Justice Advisory Council and EPA’s Risk Assessment Forum Cumulative Risk Tech Panel as an important factor to consider in risk assessment) and in economic cost/benefit and distributional analyses per Executive Order 12898 on Environmental Justice and US EPA’s Guidelines for Economic Analysis (Executive Order 12898, 1994; US Environmental Protection Agency, 2000).

As articulated in the accompanying editorial “We Can’t Do it Alone: Building a Multi-Systems Approach for Assessing and Eliminating Environmental Health Disparities,” US EPA cannot be viewed as the only stakeholder. Other federal and local agencies, as well as community and professional organizations, have a role to play in supplying data for the candidate measures and using the “story” told by these data in developing cross disciplinary, multi-agency, community-based actions to eliminate disparities.

Undoubtedly, the process of identifying, collecting, and tracking a set of measures will not occur overnight. Further, it will take even more time to move from tracking to a full understanding of disparities. Although all of these actions will take time, they should not be seen as an excuse for inaction or to dismantle extant policy. Rather, scientists, community members, funding agencies, and policy makers should continue to work to monitor and improve the public’s health through extant mechanisms, all the while building towards our goal of a systematic and scientific national tracking effort.

Recognizing that effective public participation in policy decisions and development of interventions to reduce and eliminate health disparities requires public access to information, a report jointly published by EPA and HHS on the proposed measures populated by national data would fill the need for a single document presenting scientific information and data on environmental health in minority and low-income populations, similar to EPA's *America's Children and the Environment* report. In publishing such a report, we should be mindful to provide measures that will be useful for communities, policymakers, and scientists. This report could serve as the national reference point from which state and local agencies could compare their own trends. A systematic examination will help evaluate which measures are associated with social inequities and which are not, thereby facilitating the development of interventions and policy recommendations. The ability to reflect trends at a national and local level, over time, and across a diverse set of social and physical factors may provide a key element in the effort to eliminate health disparities.

Acknowledgements

We thank Woody Neighbors and the University Michigan Center for Research on Ethnicity, Culture and Health for encouragement and administrative resources. Thank you to Jonathan Cohen of ICF Consulting for data analysis for air quality, blood mercury and asthma measures. Finally we would like to thank all of our reviewers: Dan Axelrad, Onyemaechi Nweke, Lanelle Wiggins, and Tracey Woodruff of the US EPA Office of Policy Economics and Innovation, and Arlene Rosenbaum and Jim Laurenson of ICF Consulting.

Disclaimer: The views expressed in this document are those of the authors and do not represent official US EPA policy.

Funding Sources: This paper was supported with funding from the US Environmental Protection Agency's Office of Children's Health Protection, National Health and Environmental Effects Research Laboratory (NHEERL) and The National Institute of Environmental Health Sciences (NIEHS). This paper was written for the "Environmental Health Disparities Workshop: Connecting Social and Environmental Factors to Measure and Track Environmental Health Disparities."

Note: No human subjects or experimental animals were used in this study.

Appendix A. —Methods

A1. Asthma measures

The asthma prevalence data for children and adults for Figs. 2 and 3 were obtained from the National Health

Interview Survey (NHIS) carried out by the Centers for Disease Control and Prevention, National Center for Health Statistics, <http://www.cdc.gov/nchs/nhis.htm>. The interview survey is designed to assess the health status of the non-institutionalized civilian population, using a complex multi-stage, stratified, clustered sampling design. The survey was redesigned in 1997. One new asthma measure was lifetime prevalence ("ever diagnosed with asthma"), the second measured the occurrence of an asthma episode or attack in the past 12 months (a period prevalence: "having an asthma attack in the last 12 months"). In 2001, a point prevalence measure was added to assess current asthma prevalence. If the respondent answered "yes" to the lifetime question, a second question asked, "Do you still have asthma?" For adults 18 and older, the subject would usually be the respondent. For children, the responses were provided by an adult in the same family. Estimated percentages are not shown for years and race/ethnicity groups where the relative standard error (standard deviation/estimated percentage) is 30% or greater, which indicates a possibly unstable or unreliable value.

The race/ethnicity categories used for these analyses were based on the main race category, so that those responding multiple races were excluded from the specific categories. Primarily due to OMB requirements, the race question was revised for 1999 and later, so that Pacific Islanders were included in the "Other" category (not shown). Therefore, the "Asian" category also includes Pacific Islanders for the years 1997 and 1998 only.

Asthma mortality counts by race/ethnicity and year for Fig. 4 were obtained from the Centers for Disease Control and Prevention, National Center for Health Statistics published reports Orig291F and 292F: Worktable 292F. Deaths from 358 selected causes by 5-year age groups, race, and sex: United States, 1999–2002, http://www.cdc.gov/nchs/data/dvs/mortfinal2002_work292f.pdf. Worktable Orig291F. Deaths from 113 selected causes, alcohol-induced causes, drug-induced causes, and injury by firearms, by 5-year age groups, Hispanic origin, race for non-Hispanic population, and sex: United States, 1999–2002, http://www.cdc.gov/nchs/data/dvs/mortfinal2002_workOrig291f.pdf

Mortality counts for the White Hispanic and Black Hispanic groups were estimated by subtracting the tabulated number of deaths for White (or Black) non-Hispanic from the tabulated number of deaths for White (or Black). This ignores the small error due to the number of deaths for the Origin Not Stated group. Populations for 1999 by race/ethnicity were obtained from the Census Bureau at www.census.gov. These data were obtained from the "1990–1999 Intercensal State and County Characteristics Population Estimates Files for Internet Display." For 2000–2002, populations by race/ethnicity were obtained from the National Center for Health Statistics "Bridged-Race Vintage 2004 (July 1, 2000–July 1, 2004) Postcensal

Population Estimates for Calculating Vital Rates.”¹ The age adjusted mortality rates were calculated by direct standardization.

A2. Air quality measure for $PM_{2.5}$

These measures for $PM_{2.5}$ for Figs. 6–8 were constructed by obtaining ambient air quality monitoring data from EPA’s Aerometric Information Retrieval System (AIRS) Air Quality Subsystem (AQS) at the website www.epa.gov/air/data/reports.html. For each county and year, the maximum 24-h average concentration across all monitors and days was compared to $65 \mu\text{g}/\text{m}^3$, the National Ambient Air Quality Standard for 24-h average $PM_{2.5}$. If the county maximum exceeds the standard, then the entire county population for that year is assumed to be living in a county exceeding the standard. Otherwise, the entire county population for that year is assumed for this analysis not to be living in a county exceeding the standard, even if there were no monitoring sites or no reported measurements made for that county and year. For co-located monitors, only the lowest numbered Pollutant Occurrence Code (POC) was used, following EPA recommendations. County populations for 1999 by race/ethnicity were obtained from the Census Bureau at www.census.gov. These data were obtained from the “1990–1999 Intercensal State and County Characteristics Population Estimates Files for Internet Display.” For 2000–2004, county populations by race/ethnicity were obtained from the National Center for Health Statistics “Bridged-Race Vintage 2004 (July 1, 2000–July 1, 2004) Postcensal Population Estimates for Calculating Vital Rates.”²

It should be noted that on average approximately 35% of American Indians or Alaska Natives, 9% of Asian or Pacific Islanders, 9% of Black Hispanics, 19% of Black Non-Hispanics, 14% of White Hispanics, and 34% of White Non-Hispanics live in counties where PM air quality was not monitored during the period 1999–2003. This is a limitation of air quality monitoring programs in the US. County populations for 1999 by race/ethnicity were obtained from the Census Bureau.

A3. Blood mercury measures

Laboratory data on measured mercury concentrations in blood for women between 16 and 49 years of age were

obtained from the National Health and Nutrition Examination Survey (NHANES) carried out by the Centers for Disease Control and Prevention, National Center for Health Statistics. <http://www.cdc.gov/nchs/nhanes.htm>. The survey is designed to assess the health and nutritional status of the non-institutionalized civilian population with direct physical examinations and interviews, using a complex multi-stage, stratified, clustered sampling design. Interviewers obtain information on personal and demographic characteristics, including age, household income, and race and ethnicity by self-reporting or as reported by an informant. Data were obtained from NHANES 1999–2000, and NHANES 2001–2002. Blood mercury data were provided by 4084 women between 16 and 49 years of age. The surveys were weighted to represent the national population. This analysis used the race/ethnicity categories (RIDRETH2) recoded so that persons who indicated multi-racial status but who reported their main race/ethnicity as either White Non-Hispanic or Black Non-Hispanic were re-assigned to their main race.

References

- Acevedo-Garcia, D., 2001. Zip code-level risk factors for tuberculosis: neighborhood environment and residential segregation in New Jersey, 1985–1992. *Am. J. Public Health* 91 (5), 734–741.
- Akinbami, L.J., Schoendorf, K.C., 2002. Trends in childhood asthma: prevalence, health care utilization, and mortality. *Pediatrics* 110 (2, Part 1), 315–322.
- American Lung Association, A., 2001. Urban air pollution and health inequities: a workshop report. *Environ. Health Perspect.* 109 (Suppl 3), 357–374.
- Bhopal, R., Rankin, J., Bennett, T. (Eds.), 2000. Editorial role in promoting valid use of concepts and terminology in race and ethnicity research. *Sci. Editor.* 23, 75–80.
- Brown, P., 1995. Race, class, and environmental health: a review and systematization of the literature. *Environ. Res.* 69 (1), 15–30.
- Bullard, R.D., 1990. *Dumping in Dixie: Race, Class and Environmental Quality*. Westview Press, Boulder.
- Bullard, R.D., Wright, B.H., 1993. Environmental justice for all: community perspectives on health and research needs. *Toxicol. Ind. Health* 9 (5), 821–841.
- Burger, J., 2002. Consumption patterns and why people fish. *Environ. Res.* 90 (2), 125–135.
- Burger, J., Pflugh, K.K., Lurig, L., Von Hagen, L.A., Von Hagen, S., 1999a. Fishing in urban New Jersey: ethnicity affects information sources, perception, and compliance. *Risk Anal.* 19 (2), 217–229.
- Burger, J., Stephens Jr., W.L., Boring, C.S., Kuklinski, M., Gibbons, J.W., Gochfeld, M., 1999b. Factors in exposure assessment: ethnic and socioeconomic differences in fishing and consumption of fish caught along the Savannah River. *Risk Anal.* 19 (3), 427–438.
- Burger, J., Gaines, K.F., Gochfeld, M., 2001. Ethnic differences in risk from mercury among Savannah River fishermen. *Risk Anal.* 21 (3), 533–544.
- Burger, J., Gaines, K.F., Boring, C.S., Stephens, W.L., Snodgrass, J., Dixon, C., et al., 2002. Metal levels in fish from the Savannah River: potential hazards to fish and other receptors. *Environ. Res.* 89 (1), 85–97.
- Carter-Pokras, O.D., Gergen, P.J., 1993. Reported asthma among Puerto Rican, Mexican-American, and Cuban children, 1982 through 1984. *Am. J. Public Health* 83 (4), 580–582.

¹National Center for Health Statistics. Estimates of the July 1, 2000–July 1, 2004, United States resident population from the Vintage 2004 postcensal series by year, county, age, sex, race, and Hispanic origin, prepared under a collaborative arrangement with the US Census Bureau. Available on the Internet at: <http://www.cdc.gov/nchs/about/major/dvs/popbridge/popbridge.htm>, September 9, 2005.

²National Center for Health Statistics. Estimates of the July 1, 2000–July 1, 2004, United States resident population from the Vintage 2004 postcensal series by year, county, age, sex, race, and Hispanic origin, prepared under a collaborative arrangement with the U.S. Census Bureau. Available on the Internet at: <http://www.cdc.gov/nchs/about/major/dvs/popbridge/popbridge.htm>, September 9, 2005.

- Centers for Disease Control, 2003. Environmental Public Health Indicators. National Center for Environmental Health, Division of Environmental Hazards and Health Effects, Atlanta.
- Collins, C.A., Williams, D.R., 1999. Segregation and mortality: the deadly effects of racism? *Sociol. Forum* 14 (3), 493–521.
- Corburn, J., 2002. Combining community-based research and local knowledge to confront asthma and subsistence-fishing hazards in Greenpoint/Williamsburg, Brooklyn, New York. *Environ. Health Perspect.* 110 (Suppl 2), 241–248.
- Cutler, E., Glaeser, E., 1997. Are ghettos good or bad? *Quart. J. Econom.* 112, 827–872.
- Delfino, R.J., Gong Jr., H., Linn, W.S., Pellizzari, E.D., Hu, Y., 2003. Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. *Environ. Health Perspect.* 111 (4), 647–656.
- Diez Roux, A.V., 2002. A glossary for multilevel analysis. *J. Epidemiol. Community Health* 56 (8), 588–594.
- Environmental Justice and Health Union, 2004. Environmental Exposure and Racial Disparities, Oakland.
- Evans, G.W., 1994. The psychological costs of chronic exposure to ambient air pollution. In: RL Isaacson, K.J. (Ed.), *The Vulnerable Brain and Environmental Risks*. Plenum, New York, pp. 167–182.
- Evans, G.W., Colome, S.D., Shearer, D.F., 1988. Psychological reactions to air pollution. *Environ. Res.* 45 (1), 1–15.
- Executive Order 12898, 1994. In: Clinton, P.W.J. (Ed), *Federal Actions to Address Environmental Justice in Minority Populations and Low Income Populations*.
- Fang, J., Madhavan, S., Bosworth, W., Alderman, M.H., 1998. Residential segregation and mortality in New York City. *Soc. Sci. Med.* 47 (4), 469–476.
- Gee, G.C., 2002. A multilevel analysis of the relationship between institutional and individual racial discrimination and health status. *Am. J. Public Health* 92 (4), 615–623.
- Gee, G.C., Payne-Sturges, D.C., 2004. Environmental health disparities: a framework integrating psychosocial and environmental concepts. *Environ. Health Perspect.* 112 (17), 1645–1653.
- Gergen, P.J., Mullally, D.I., Evans III, R., 1988. National survey of prevalence of asthma among children in the United States, 1976–1980. *Pediatrics* 81 (1), 1–7.
- Geronimus, A.T., Bound, J., Waidmann, T.A., 1999. Poverty, time, and place: variation in excess mortality across selected US populations, 1980–1990. *J. Epidemiol. Community Health* 53 (6), 325–334.
- Geronimus, A.T., Bound, J., Waidmann, T.A., Colen, C.G., Steffick, D., 2001. Inequality in life expectancy, functional status, and active life expectancy across selected black and white populations in the United States. *Demography* 38 (2), 227–251.
- Gordis, L., 2000. *Epidemiology*. Saunders, Philadelphia.
- Gwynn, R.C., Thurston, G.D., 2001. The burden of air pollution: impacts among racial minorities. *Environ. Health Perspect.* 109 (Suppl. 4), 501–506.
- Hart, K.D., Kunitz, S.J., Sell, R.R., Mukamel, D.B., 1998. Metropolitan governance, residential segregation, and mortality among African Americans. *Am. J. Public Health* 88 (3), 434–438.
- Hightower, J.M., O'Hare, A., Hernandez, G.T., 2006. Blood mercury reporting in NHANES: identifying Asian, Pacific Islander, Native American, and multiracial groups. *Environ. Health Perspect.* 114 (2), 173–175.
- Institute of Medicine, 1999. *Toward Environmental Justice: Research, Education, and Health Policy Needs*. National Academy Press, Washington, DC.
- Jargowsky, P., 1997. *Poverty and Place: Ghettos, Barrios, and the American City*. Russell Sage Foundation, New York.
- Kaplan, J.B., Bennett, T., 2003. Use of race and ethnicity in biomedical publication. *J Am. Med. Assoc.* 289, 2709–2716.
- Kjellstrom, T., Corvalan, C., 1995. Framework for the development of environmental health indicators. *World Health Stat. Quart.* 48 (2), 144–154.
- Landrigan, P.J., Graham, D.G., Thomas, R.D., 1994. Environmental neurotoxic illness: research for prevention. *Environ. Health Perspect.* 102 (Suppl. 2), 117–120.
- Laveist, T.A., 1989. Linking residential segregation to the infant-mortality race disparity in US cities. *Soc. Sci. Res.* 73 (90), 94.
- Laveist, T.A., 1993. Segregation, poverty, and empowerment: health consequences for African Americans. *Milbank Q* 71 (1), 41–64.
- LaVeist, T.A., 1994. Beyond dummy variables and sample selection: what health services researchers ought to know about race as a variable. *Health Serv. Res.* 29 (1), 1–16.
- Lee, C., 1993. Beyond toxic wastes and race. In: Bullard, R.D., Chavis, B. (Eds.), *Confronting Environmental Racism: Voices from the Grassroots*. South End Press, Boston, pp. 41–52.
- Lee, C., 2002. Environmental justice: building a unified vision of health and the environment. *Environ. Health Perspect.* 110 (Suppl. 2), 141–144.
- Lopez, R., 2002. Segregation and black/white differences in exposure to air toxics in 1990. *Environ. Health Perspect.* 110 (Suppl. 2), 289–295.
- Lundberg, A., 1996. Psychiatric aspects of air pollution. *Otolaryngol. Head Neck Surg.* 114 (2), 227–231.
- Maantay, J., 2001. Zoning, equity, and public health. *Am. J. Public Health* 91 (7), 1033–1041.
- Maldonado, G., Greenland, S., 2002. Estimating causal effects. *Int. J. Epidemiol.* 31 (2), 422–429.
- Mannino, D., Homa, D., Akinbami, L., Moorman, J., Gwynn, C., Redd, S., 2002. *Surveillance for Asthma—United States 1980–1999*. Centers for Disease Control and Prevention, Atlanta.
- Maroni, M., Colosio, C., Ferioli, A., Fait, A., 2000. Biological monitoring of pesticide exposure: a review. *Introduction. Toxicology* 143 (1), 1–118.
- Massey, D., 2001. Residential segregation and neighborhood conditions in US metropolitan areas. In: Smelser, N.J., Wilson, W.J., Mitchell, F. (Eds.), *America Becoming: Racial Trends and their Consequences*. National Academy Press, Washington, DC.
- Massey, D., Denton, N.A., 1993. *American Apartheid: Segregation and the Making of the Underclass*. Harvard University Press, Cambridge.
- McConnell, R., Berhane, K., Gilliland, F., London, S.J., Vora, H., Avol, E., et al., 1999. Air pollution and bronchitic symptoms in Southern California children with asthma. *Environ. Health Perspect.* 107 (9), 757–760.
- McConnell, R., Berhane, K., Gilliland, F., London, S.J., Islam, T., Gauderman, W.J., et al., 2002. Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359 (9304), 386–391.
- Morello-Frosch, R., Jesdale, B.M., 2006. Separate and unequal: residential segregation and estimated cancer risks associated with ambient air toxics in US metropolitan areas. *Environ. Health Perspect.* 114 (3), 386–393.
- Morello-Frosch, R., Lopez, R., in press. The riskscape and the color line: examining the role of segregation in environmental health disparities. *Environ. Res.*, in press.
- National Center for Environmental Health, 2003. *National Report on Human Exposure to Environmental Chemicals*. CDC. Available at <<http://www.cdc.gov/exposurereport/>>.
- Northridge, M.E., Stover, G.N., Rosenthal, J.E., Sherard, D., 2003. Environmental equity and health: understanding complexity and moving forward. *Am. J. Public Health* 93 (2), 209–214.
- O'Neill, M.S., Jerrett, M., Kawachi, I., Levy, J.I., Cohen, A.J., Gouveia, N., et al., 2003. Health, wealth, and air pollution: advancing theory and methods. *Environ. Health Perspect.* 111 (16), 1861–1870.
- Ostro, B., Lipsett, M., Mann, J., Braxton-Owens, H., White, M., 2001. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiology* 12 (2), 200–208.
- Pastor, M., 2001. Geography and opportunity. In: Smelser, N., Wilson, W.J., Mitchell, F. (Eds.), *America Becoming: Racial Trends and Their Consequences*. National Academy Press, Washington, DC, pp. 435–468.
- Pew Environmental Health Commission, 2000. *America's environmental health gap: why the country needs a nationwide health tracking*

- network. Technical Report. Pew Environmental Health Commission, Johns Hopkins School of Hygiene and Public Health, Baltimore.
- Polednak, A.P., 1991. Black-white differences in infant mortality in 38 standard metropolitan statistical areas. *Am. J. Public Health* 81 (11), 1480–1482.
- Polednak, A.P., 1993. Poverty, residential segregation, and black/white mortality ratios in urban areas. *J. Health Care Poor Underserved* 4 (4), 363–373.
- Pope III, C.A., Burnett, R.T., Thurston, G.D., Thun, M.J., Calle, E.E., Krewski, D., et al., 2004. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation* 109 (1), 71–77.
- Potter, L.B., 1991. Socioeconomic determinants of white and black males' life expectancy differentials, 1980. *Demography* 28 (2), 303–321.
- Rios, R., Poje, G.V., Detels, R., 1993. Susceptibility to environmental pollutants among minorities. *Toxicol. Ind. Health* 9 (5), 797–820.
- Rothwell, C.J., Hamilton, C.B., Leaverton, P.E., 1991. Identification of sentinel health events as indicators of environmental contamination. *Environ. Health Perspect.* 94, 261–263.
- Schwartz, J., 1999. Air pollution and hospital admissions for heart disease in eight US counties. *Epidemiology* 10 (1), 17–22.
- Schwartz, J., Morris, R., 1995. Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. *Am. J. Epidemiol.* 142 (1), 23–35.
- Schwartz, J., Slater, D., Larson, T.V., Pierson, W.E., Koenig, J.Q., 1993. Particulate air pollution and hospital emergency room visits for asthma in Seattle. *Am. Rev. Respir. Dis.* 147 (4), 826–831.
- 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., Gong Jr., H., Bailar III, J.C., Ford, J.G., Gold, D.R., Lambert, W.E., et al., 1993. Air pollution health risks: do class and race matter? *Toxicol. Ind. Health* 9 (5), 843–878.
- Shepard, P.M., 2002. Preface: advancing environmental justice through community-based participatory research. *Environ. Health Perspect.* (110 (Suppl. 2)), 139–140.
- Shihadeh, E.S., Flynn, N., 1996. Segregation and crime: the effect of Black social isolation on the rates of Black urban violence. *Soc. Forces* 74 (4), 1325–1352.
- Silbergeld, E., Tonat, K., 1994. Investing in prevention: opportunities to prevent disease and reduce health care costs by identifying environmental and occupational causes of noncancer disease. *Toxicol. Ind. Health* 10 (6), 675–827.
- Soobader, M.-J., Cubbin, C., Gee, G.C., Rosenbaum, A., Laurenson, J., in press. Levels of analysis for the study of environmental health disparities. *Environ. Res.*, in press.
- Subramanian, S.V., Acevedo-Garcia, D., Osypuk, T.L., 2005. Racial residential segregation and geographic heterogeneity in black/white disparity in poor self-rated health in the US: a multilevel statistical analysis. *Soc. Sci. Med.* 60 (8), 1667–1679.
- Thacker, S.B., Berkelman, R.L., 1988. Public health surveillance in the United States. *Epidemiol. Rev.* 10, 164–190.
- Thacker, S.B., Stroup, D.F., Parrish, R.G., Anderson, H.A., 1996. Surveillance in environmental public health: issues, systems, and sources. *Am. J. Public Health* 86 (5), 633–638.
- Thacker, S.B., Parrish, R.G., Trowbridge, F.L., 1988. A method for evaluating systems of epidemiological surveillance. *World Health Stat. Q.* 41 (1), 11–18.
- United Church of Christ, 1987. *Toxic Wastes and Race in the United States: A National Report on the Racial and Socio-Economic Characteristics With Hazardous Waste sites.* United Church of Christ, Commission for Racial Justice, New York.
- US Department of Health and Human Services, 2004. *Healthy People 2010.* Available: <<http://www.healthypeople.gov/Publications/>> (accessed May 22, 2004).
- US Environmental Protection Agency, 2000. *Guidelines for Preparing Economic Analyses.* Office of the Administrator, Washington, DC.
- US Environmental Protection Agency, 2004. *Draft Report on the Environment.* Available: <www.epa.gov/indicators/roe/index.htm>.
- US Environmental Protection Agency, 2005. *Particle pollution and your health.* Available: <<http://www.epa.gov/airnow/particle/airborne.html>> (accessed April 11, 2005).
- US Office of Management and Budget, 1997. *Statistical Policy Directive No. 15. Race and ethnic standards for federal statistics and administrative reporting.*
- Wernette, D., Nieves, L.A., 1992. Breathing polluted air. *EPA J.* 18 (1), 16–17.
- Williams, D.R., 1999. Race, socioeconomic status, and health. The added effects of racism and discrimination. *Ann. NY Acad. Sci.* 896, 173–188.
- Williams, D.R., Collins, C., 2001. Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Rep.* 116 (5), 404–416.
- Williams, D.R., Yu, Y., 1997. Racial differences in physical and mental health: socioeconomic status, stress, and discrimination. *J. Health Psychol.* 2 (3), 335–351.
- Wilson, W.J., 1987. *The Truly Disadvantaged.* University of Chicago Press, Chicago.
- Wilson, W.J., 1996. *When Work Disappears: the World of the New Urban Poor.* Alfred A. Knopf, New York.
- Woodruff, T.J., Axelrad, D.A., Kyle, A.D., 2000. *America's children and the environment: a first view of available measures.* EPA 240-R-00-006. US Environmental Protection Agency, Office of Policy, Economics and Innovation and Office of Children's Health Protection, Washington, DC.
- Woodruff, T.J., Axelrad, D.A., Kyle, A.D., Nweke, O., Miller, G., 2003. *America's children and the environment: measures for contaminants, exposures, and diseases.* EPA 240-R-03-001. US Environmental Protection Agency, Office of Policy, Economics and Innovation and Office of Children's Health Protection, Washington, DC.
- Zanobetti, A., Schwartz, J., Dockery, D.W., 2000. Airborne particles are a risk factor for hospital admissions for heart and lung disease. *Environ. Health Perspect.* 108 (11), 1071–1077.