AIR POLLUTION HEALTH RISKS: DO CLASS AND RACE MATTER?

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4. Abbreviations: COPD, chronic obstructive pulmonary disease; EPA, Environmental Protection Agency; ETS, environmental tobacco smoke; FEV1, forced expiratory volume in one second; FVC, forced vital capacity; HAPs, hazardous air pollutants; NAAQS, National Ambient Air Quality Standards; NHANES, National Health and Nutrition Examination Survey; TLC, total lung capacity.

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Air pollution is not spread evenly across demographic groups. Exposures and associated health risks appear to fall disproportionately on populations that are poor and nonwhite. Although scientific evidence documenting disparities in air pollution exposures, doses, and health effects is scant, the available data strongly support the contention that disadvantaged groups, many of whom are ethnic and racial minorities, routinely encounter levels of air pollution that are higher than average. The extent to which exposure differentials contribute to observed differences in health status by class and race is unknown, but worthy of further investigation. We recommend several steps, all of them feasible and most of them relatively inexpensive, to improve our understanding and ability to address environmental health disparities.

INTRODUCTION

Exposure to environmental toxicants is sometimes sufficient to cause adverse health effects. The quantitative estimation of such health risks, based on exposure and dose-response information, is fundamental to policy decisions about which risks are unacceptable and how best to manage them. In this paper, we start from the premise that an explicit goal of these societal decisions should be "environmental health equity," a provision of adequate protection for all people, regardless of age, gender, health status, social class, or ethnicity/race.

Recently, concerns have been raised that disadvantaged populations—e.g., those who are poor, have limited formal education, and are either unemployed or work under hazardous conditions—may suffer disproportionate effects from environmental pollution (UCC, 1987; ATSDR, 1988, 1990; Heritage, 1992; U.S. EPA, 1992a). Low-income groups are routinely exposed to elevated levels of certain pollutants in the air they breathe, the water they drink, the food they eat, and the soil and dust they encounter (UCC, 1987; ATSDR, 1988, 1990; Heritage, 1992; U.S. EPA, 1992a). These same groups are also more likely to lack knowledge about environmental health issues, to have a substandard diet, to lack access to adequate health care, to smoke cigarettes and drink alcohol, and, in general, to have more stressful and less healthful lives (DHSS, 1985, 1991a; Heritage, 1992; U.S. EPA, 1992a).

Minorities defined by race (i.e., African-American or black, Asian and Pacific Islander, Native American) and ethnicity (e.g., Hispanic) are statistically more likely to be economically disadvantaged than their white counterparts (DHHS, 1985, 1991a; Goldman, 1991). Bullard (Bullard and Wright, 1986, 1987; Bullard, 1991) and others (Zwerdling, 1973; Neiman and Loveridge, 1980; Van Liere and Dunlap, 1980; UCC, 1987; Goldman, 1991; Bryant and Mohai, 1992) have suggested that some minority groups (e.g., blacks, Hispanics) are subject to "environmental racism;" that is, institutionalized actions, whether intentional or not, that systematically increase environmental health risks.

Air pollution is an important public health problem (CAA, 1990) that may be distributed unevenly across social classes and ethnic/racial groups (Goldman, 1991; Heritage, 1992; U.S. EPA, 1992a; Bryant and Mohai, 1992). This paper surveys some of the available scientific information on disparities in air pollution exposures and related health effects in the United States. We examine what is known about differentials in potential exposures to indoor and outdoor air pollutants, describe important confounding variables that affect the interpretation of exposures and health effects, compare health status across class and race, and survey reasons for possible differential susceptibility to exposures. The discussion concludes by identifying important research needs for improved understanding of equity in environmental health.

FUNDAMENTAL CONCEPTS USED IN STUDYING AIR POLLUTION EXPOSURES

Contact between people and pollution is at the heart of concerns about environmental health risks. This section presents the fundamental ideas and definitions behind environmental exposure analysis, highlights the importance of human time-activity patterns, and discusses criteria for selection of an exposure estimator.

Basic Concepts in Human Exposure Analysis

Environmental exposure is defined as contact between the body envelope and a specific pollutant or mixture of pollutants. Factors that may be important in describing this contact include the route, magnitude, duration, and frequency of exposure. For risk assessment and risk management purposes, it is also important to understand the distribution of individual exposures in the population (e.g., mean, variance, upper tenth percentile), the causes of high exposures (e.g., sources, environmental pathways, locations and settings, time-activity patterns), and the relationship in humans between exposure and dose (e.g., amount of the pollutant or its metabolite that reaches the target tissue) (Sexton and Ryan, 1988; NRC, 1991a,b; Schwab, 1992; Sexton et al., 1992).

Air pollution arises from a variety of sources, both outdoors (e.g., industrial factories, motor vehicles, residential wood burning) and indoors (e.g., combustion appliances, consumer products, tobacco smoke). People are exposed to a wide range of low-level air pollution during the course of their normal daily activities. Depending on factors such as occupation, activity patterns, and place of residence, some individuals can be exposed to certain air pollutants at levels much higher than average (Samet et al., 1987, 1988; Sexton and Ryan, 1988; NRC, 1991a,b; Gold, 1992; Schwab, 1992; Sexton et al., 1992).

It is often impractical (i.e., too costly) or impossible (i.e., lack of suitable methods) to measure air pollution exposures directly for a particular person, subgroup, or population. It is common, therefore, to estimate exposures using surrogate measures (e.g., emission estimates, outdoor concentrations in a community) or mathematical models (Sexton and Ryan, 1988; NRC, 1991a,b; Sexton et al., 1992).

For example, measured ambient (outdoor) concentrations of ozone, nitrogen dioxide, carbon monoxide, sulfur dioxide, and particulate matter are often used to estimate exposures for the residents of a particular community, such as a city or metropolitan area. These estimates may be used as reasonable approximations for people who are outdoors near the monitoring stations. Accuracy is assumed to decrease with distance and with time spent indoors. Estimates can be highly inaccurate for individuals whose daily activities, including work, commuting, and recreation, bring them in contact with pollutant concentrations substantially different from those measured at the central monitoring site (Samet et al., 1987, 1988; Sexton and Ryan, 1988; NRC, 1991a,b; Gold, 1992; Sexton et al., 1992).

Scientists have tried to improve exposure estimates by dividing the space through which people move into "microenvironments," such as outdoors at home, indoors at home, indoors at work, and in transit. By combining information about time spent in each microenvironment and associated activities with knowledge of microenvironmental pollution concentrations, exposures can be estimated with more certainty. Although data are sparse, it is apparent that a person's time-activity pattern (i.e., the time spent in various microenvironments for which we can estimate or measure air pollutant concentrations) is an important predictor of air pollution exposure (Samet et al., 1987, 1988; Sexton and Ryan, 1988; NRC, 1991a,b; Sexton et al., 1992; Schwab, 1992).

Role of Time-Activity Patterns in Exposure Estimation

Systematic variations in activity patterns among socioeconomic and ethnic/racial subgroups could lead to differences in air pollution exposures. Although time-activity data have been used extensively by industrial hygienists, urban geographers and planners, and efficiency experts, application of this approach to nonindustrial air pollution is relatively recent. Few studies of time-activity patterns are directly applicable to exposure assessment, and virtually none focus on disadvantaged or minority populations (Sexton and Ryan, 1988; NRC, 1991a,b; Schwab, 1992).

Results from the few studies that have been conducted consistently show significant differences in where people go, what they do, and how long they spend there by age, gender, and work status (i.e., employed outside the home) (Schwab, 1992). Children have different activity patterns than adults, and some studies suggest that patterns for inner city children are different from those of suburban and rural counterparts. On average, men spend about twice as much time outdoors and traveling as women do, independent of work status. Whether a person works outside of the residence appears to account in large part for the observed differences among adults (Schwab, 1992).

Differences in the use of discretionary time and the amount of travel depend on socioeconomic status. Better educated, more affluent people tend to spend more time in activities away from home and to travel further than those who are less educated and less affluent (Schwab, 1992).

The available evidence suggests that microenvironmental parameters, such as the presence and use of indoor emission sources, depend on employment status. Some gender differences are related to household chores (e.g., kitchen versus garage activities) and personal hygiene habits (e.g., use of hair spray, nail polish, and makeup). There is evidence to suggest that low-income and minority groups are more likely to have certain housing characteristics associated with increased indoor air pollution, like greater use of unvented combustion appliances and higher levels of mite and cockroach allergens in house dust (Gold, 1992). These factors might lead to differential exposures across socioeconomic and ethnic groups (Schwab, 1992).

Selecting an Appropriate Exposure Estimator

As shown in Figure 1 (NRC, 1991b; Sexton et al., 1992), the certainty of exposure estimates depends on the surrogates being used. Some comparatively inexpensive exposure estimators, such as production volumes or emission estimates, are far removed from the point of contact between people and pollution. Their use to estimate human exposures requires assumptions, sometimes heroic, about matters such as transport of the chemical into environmental media, concentrations in both indoor and outdoor air, and contact between people and the pollutant. Though very uncertain, the resulting exposure estimates can be useful in identification of geographical areas where exposures are likely to be elevated (e.g., residential areas in close proximity to important emission sources) (NRC, 1991a,b; Sexton et al., 1992).

More accurate and precise methods are needed to document actual exposures of people living in those areas. Ambient air measurements, such as may be recorded at central monitoring sites, provide better estimates, but can still be very inaccurate for members of the community who spend substantial time either indoors or outdoors in other communities (NRC, 1991a,b; Sexton et al., 1992). The best estimates are those based on direct measurements of "personal" exposure (e.g., real-time concentrations of airborne carbon monoxide obtained from small, portable monitors carried by individuals) or internal dose (e.g., concentrations of carbon monoxide in the blood). Generally (see Figure 1), the cost of estimating exposure is directly proportional to the certainty of the estimate (NRC, 1991a,b; Sexton et al., 1992).

Selection of an exposure estimator should be based on the intended use(s), the required certainty of the estimate, and the costs of the information. For example, if the objective is to identify geographic areas where exposures are likely to be high, then reliance on indirect surrogates like emission estimates may be appropriate. If, however, the goal is to estimate health risks for potentially susceptible subpopulations in an area of high pollutant emissions, then more direct estimators, such as models based on microenvironmental concentration measurements and time-activity pattern data, are needed (NRC, 1991a,b; Sexton et al., 1992).

Informed risk assessment and risk management decisions require accurate exposure information for the general population and for persons who are at the high end of the exposure distribution. Yet despite their obvious importance, reliable exposure data are sparse or lacking entirely for broad segments of the U.S. population (Sexton et al., 1992). Most of the debate about

SEXTON et al., 1992		NATIONAL RESEARCH COUNCIL, 1991b	
EXPOSURE ESTIMATORS	COSTS AND ABILITY TO PREDICT/CLASSIFY EXPOSURES	EXPOSURE ESTIMATORS	APPROXIMATION TO ACTUAL EXPOSURE
• Production Volumes • Emission Inventories • Environmental Concentrations - Models - Measurements • Microenvironmental Concentrations - Models - Measurements • Human Contact - Models - Measurements • Internal Dose - Models - Measurements	Generally Lower Costs and Limited Accuracy → Generally Higher Costs and Better Accuracy	• Residence or Employment in Defined Geographic Area (e.g., county of the emission source/site) • Residence or Employment in Geographic Area in Reasonable Proximity to the Emission Source/Site • Distance from Emission Source/Site or Duration of Residence • Distance from Emission Source/Site and Duration of Residence • Quantified Surrogates of Exposure (e.g., estimates of drinking water use) • Quantified Area or Ambient Measurements in the Vicinity of Residence or Location of Other Significant Activities • Quantified Personal Measurements	Poorest → Best

FIGURE 1. Comparison of the utility of different exposure estimators (adapted from NRC, 1991b and Sexton et al., 1992).

environmental equity has been driven by ecologic (descriptive) studies showing that demographic variables, such as socioeconomic status and race, are positively correlated with proximity to emission sources (UCC, 1987; Goldman, 1991), estimated releases into the environment (Goldman, 1991), or ambient air concentrations (Gelobter, 1986; Wernette and Nieves, 1991; Brajer and Hall, 1992).

OVERVIEW OF INFORMATION ON DIFFERENCES IN AIR POLLUTION EXPOSURES

Available information related to air pollution exposure differentials by class and race is summarized for outdoor and indoor environments.

Outdoor Exposures to Criteria Air Pollutants

The "criteria air pollutants" are those for which National Ambient Air Quality Standards (NAAQS) have been established. At present, these are ozone, nitrogen dioxide, carbon monoxide, sulfur dioxide, particulate matter, and lead. Several studies from the 1970s reported that low income was positively correlated with concentrations of the criteria air pollutants, and that minority populations tended to live in areas where pollution levels were highest (Zupan, 1973; McCaull, 1976; Gianessi et al., 1977; Handy, 1977; Harrison and Rubinfeld, 1977; Asch and Seneca, 1978; Gianessi and Wolff, 1979). Air pollution exposures were likely to be highest for young people who worked in low-paying jobs and lived in low-rent districts, particularly young blacks and Hispanics.

By the 1980s, it was clear that many urban areas were out of compliance with the NAAQS, and would probably remain so for the foreseeable future. Because minority populations are more likely than whites to reside in urban areas (See Table 1) (Census, 1990), they are more likely to have higher exposures.

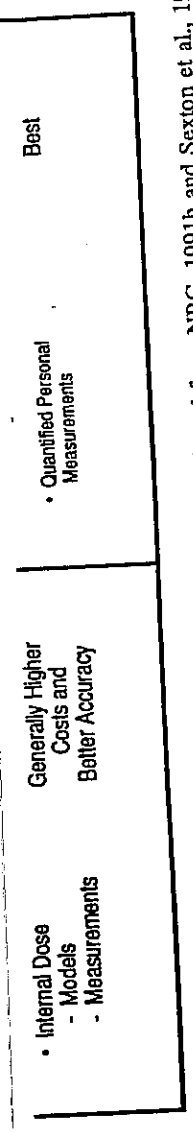


FIGURE 1. Comparison of the utility of different exposure estimators (adapted from NRC, 1991b and Sexton et al., 1992).

TABLE 1. Comparison of the Percentage of Specific Ethnic/Racial Groups Living in Urban and Rural Areas (Adapted from Census, 1990)

Ethnic Group	Urban Areas	Rural Areas (Farms)	Rural Areas (Non-farm)
White	70.3%	2.3%	27.0%
Black	86.1	0.3	13.6
Hispanic	91.2	0.7	8.1
Other	86.5	0.4	12.5

Gelobter (1986) analyzed the distribution of criteria air pollutants by income and race from 1970 to 1984. He found that outdoor air quality had improved nationwide, and exposure disparities by income had diminished over time. Nevertheless, minority exposures still ranged from 40% to 60% higher than for whites. In addition, the proportional decrease in the exposure index for families with incomes greater than $75,000/year was 20% more than the decrease for families with incomes less than $9,000/year.

In 1991, Wernette and Nieves (1991) examined the percentages of whites, blacks, and Hispanics living in geographic areas out of compliance with the NAAQS. As shown in Table 2, they found the proportion of blacks and Hispanics living in such areas to be substantially higher than for whites, with the percentage of Hispanics greater than for blacks, except in areas where sulfur dioxide levels exceeded the NAAQS.

TABLE 2. Percentage of Whites, Blacks, and Hispanics in EPA-designated Air Quality Non-attainment Areas, by Pollutant (Adapted from Wernette and Nieves, 1991)

	Percentage of Population in Non-Attainment Areas		
Air Pollutants	Whites	Blacks	Hispanics
Particulate Matter	14.7%	16.5%	34.0%
Carbon Monoxide	33.6	46.0	57.1
Ozone	52.5	62.2	71.2
Sulfur Dioxide	7.0	12.1	5.7
Lead	6.0	9.2	18.5

Brajer and Hall (1992) developed a statistical model to estimate distributions of exposures to ozone and inhalable particulate matter by demographic group in Southern California. Estimated exposures to particles were above average in younger age groups, in lower income groups, and in blacks and Hispanics. Smaller and less consistent differences were observed across groups for estimated ozone exposures. Because analyses relied on estimates rather than actual (measured) exposures, and because time-activity data for racial/ethnic groups were not used, these findings are suggestive but could not prove significant differences across age, income, and racial/ethnic groups for air pollution exposures.

Outdoor Exposures to Hazardous (Non-Criteria) Air Pollutants

Title III of the 1990 Clean Air Act Amendments (1990) lists 189 substances or groups of substances (e.g., chromium compounds) as Hazardous Air Pollutants (HAPs). Most of the 189 agents are classified as HAPs because they are known or suspected to cause adverse health effects in humans (Hassett-Simple and Cote, 1990; U.S. EPA, 1990; Vandenberg et al., 1993). To protect public health, technology-based emission standards are required for major industrial sources of HAPs. Subsequent to implementation and compliance with these standards, an analysis of the remaining "residual risks" is also required (CAA, 1990).

Although quantitative estimation of health risks is difficult because of inadequate data on exposures, doses, and effects, preliminary estimates suggest that up to 2,000 cancer cases per year may result from outdoor exposures to 45 of the 189 HAPS (U.S. EPA, 1990). Noncancer health effects for exposures to HAPs, including nonmalignant respiratory disease, hematopoietic abnormalities, neurotoxicity, renal toxicity, and reproductive and developmental toxicity, may also be widespread. Approximately 50 million people live near emission sources where estimated concentrations of one or more HAPs exceed "levels of concern" for

noncancer health effects in humans (Hasset-Simple and Cote, 1990). There is, however, a paucity of data available to estimate actual exposures for either the general population or communities potentially at high risk.

Exposures to Indoor Air Pollutants

Most people spend 90% or more of their time indoors (e.g., home, work, public and commercial buildings), and some potentially susceptible subgroups, such as infants, the elderly, and the infirm, are inside virtually all the time (Samet et al., 1987, 1988; Sexton and Ryan, 1988; Gold, 1992). It is now recognized that concentrations of many air pollutants (e.g., benzene, perchloroethylene, nitrogen dioxide, carbon monoxide, inhalable particles, radon, asbestos, formaldehyde, aeroallergens, environmental tobacco smoke, certain pesticides) tend to be substantially higher indoors than out. Air pollution levels inside buildings and vehicles are often a major determinant of human exposures (Samet et al., 1987, 1988; Sexton and Ryan, 1988; Gold, 1992).

Any differences in air pollution exposures by class or race might reflect differences in the levels of air pollution encountered indoors. Data on indoor air pollutant concentrations are sparse, however, and only limited information exists to investigate possible disparities between whites and minority groups.

Goldstein et al. (Goldstein et al., 1986, 1988; Goldstein and Andrews, 1987) have studied indoor air pollution exposures of low-income, inner city residents, most of whom were African-American. In New York City, exposure of residents of Harlem and Washington Heights to both nitrogen dioxide and carbon monoxide in their homes tended to be higher than levels reported in other indoor air studies (Goldstein et al., 1988). Elevated concentrations in kitchens were caused by use of unvented gas-fired cooking stoves, frequently for space heating. Nitrogen dioxide concentrations inside these homes often exceeded 100 micrograms per cubic meter of air (Goldstein et al., 1986, 1988; Goldstein and Andrews, 1987).

Using data from Washington, D.C., a city that is more than 75% African-American, Schwab (1990a) compared personal exposure to carbon monoxide across groups defined by work status and gender. Variability of time spent in important microenvironments showed clear differences, which should lead to exposure differentials, though direct measurements did not demonstrate large differences among groups.

Schwab (1990b) used this same data set to analyze residential carbon monoxide concentrations and demographic variables. She concluded that highest average carbon monoxide exposures were more likely for individuals who were black, below the poverty level, or over 65 years of age. Within the District of Columbia, the highest average carbon monoxide concentrations were observed on the east side, which is also the area with the greatest proportion of economically disadvantaged residents.

Indoor Lead Exposures

Lead exposures often occur through multiple pathways (e.g., air, water, soil/dust, food) and routes (i.e., inhalation, ingestion, dermal contact). As lead has been phased out of both gasoline (once a primary source of inhalation exposures) and solder used for canned food (once a major source of dietary exposures), average blood lead levels have declined substantially in every age, race, gender, and income category for the U.S. population (ATSDR, 1988; Schwartz and Levin, 1992). Children from all socioeconomic and ethnic groups have blood levels high enough to cause adverse health effects, but the percentage of children defined as having unacceptable blood lead levels (currently greater than 15 μg/dl) is higher among African-Americans than whites (see Table 3) (ATSDR, 1988). For both blacks and whites, increasing family income is associated with lower blood lead concentrations, and the difference between blacks and whites is smallest for the highest income level.

TABLE 3. Estimated Percentage of Children 0.5 to 5 Years Old with Blood Lead Levels Greater than 15 μg/dl, Living in Cities with a Population over One Million, by Race and Income (Adapted from ATSDR, 1988)

	Family Income		
Race	< $6,000	$6,000 - $15,000	> $15,000
Black	68%	54%	38%
White	36%	23%	12%

Reasons for the continuing disparity in blood lead levels are not known (ATSDR, 1988; Schwartz and Levin, 1992). However, a possible source of lead exposure for inner city residents, especially children, is lead-based paint, which was used often prior to 1950. If the paint is flaking, peeling, or cracking, building occupants may be exposed through inhalation, ingestion, and/or dermal contact, to lead in common household dust. Children may also be exposed by eating lead-paint chips. For most adults and children, the primary exposure pathway appears to be ingestion of contaminated house dust as a result of normal hand-to-mouth activity (Schwartz and Levin, 1992).

POTENTIAL INTERFERENCES WITH HEALTH STATUS COMPARISONS

Health is the product of many different variables, and it is often difficult to determine unambiguously the contribution of environmental exposures to observed health status for an individual or a population. The following discussion surveys some of the important variables that can interfere with efforts to determine the role of environmental exposures in causing disease or injury.

Socioeconomic Class as a Determinant of Health Status

An important aspect of environmental equity is the relationship of health status to poverty, as well as to race or ethnicity. These factors are correlated and multivariate data are necessary to

sort out their separate effects. The United States, however, has not historically collected morbidity and mortality statistics by class indicators, such as income, education, and occupation, so it is often impossible to obtain reliable health status information categorized by both race and class (U.S. EPA, 1992a; Montgomery and Carter-Pokras, 1993).

Disadvantaged populations (e.g., low income, low educational attainment, blue collar jobs) might be expected to have above average frequency or severity of health-related problems because of factors such as poor nutrition, stressful living and working conditions, higher smoking prevalence, and more limited access to health care. In fact, the available evidence indicates that lower socioeconomic status is strongly related to higher rates of disease and death (DHHS, 1985, 1991a,b; Goldman, 1991; Montgomery and Carter-Pokras, 1993). Moreover, health status seems to be improving more slowly in disadvantaged than in advantaged social classes, so that the gap is widening (Feldman et al., 1989; Pappas et al., 1991; Montgomery, 1992).

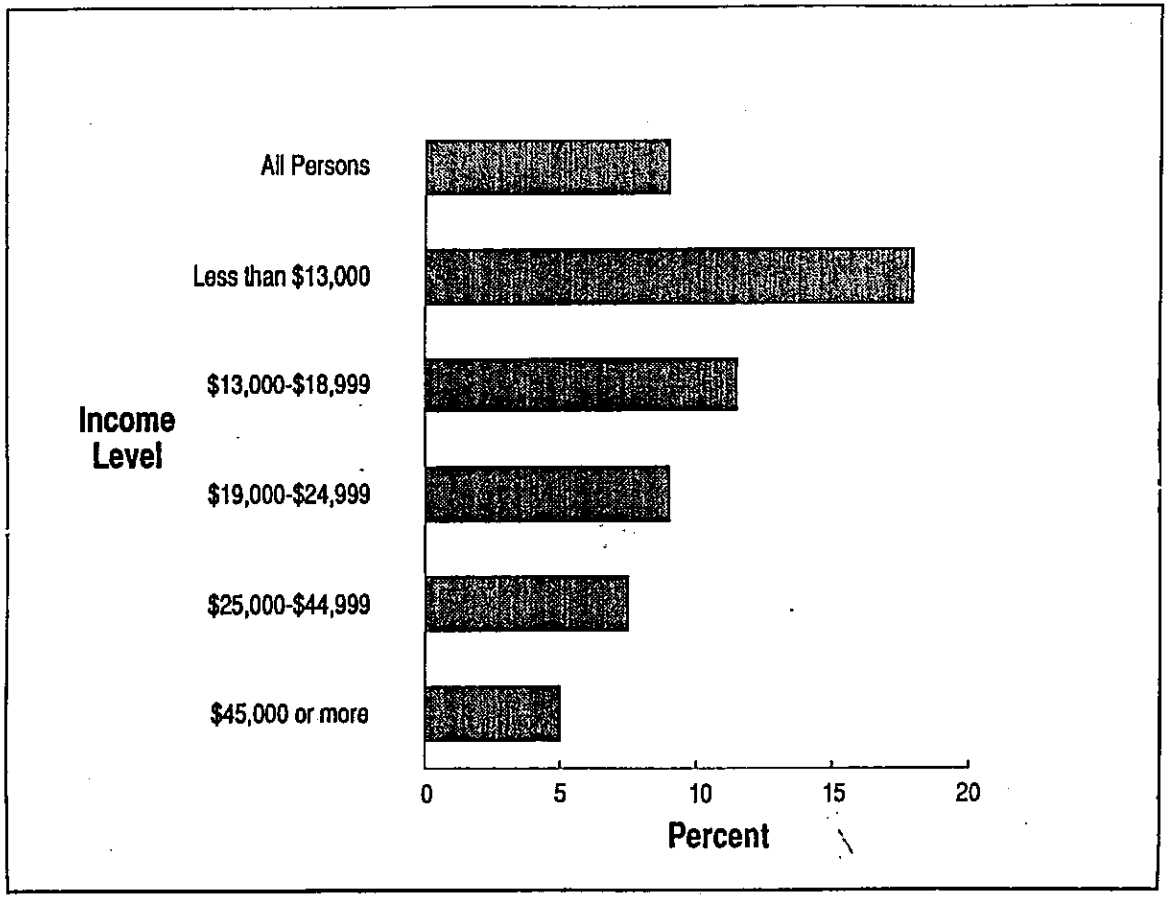
Those in lower income groups are more likely to report limitations in major personal activities (e.g., where they can go, what they can do) because of their health (DHHS, 1991b). As shown in Figure 2 (DHHS, 1991a), individuals living in families with annual incomes less that $13,000 are about twice as likely as the aggregate population to be forced by their health status to limit where they go and what they do. Activity limitations were four times more common among people with eight years or less formal education than among those with 16 years or more (DHHS, 1991b). However, these findings are less than conclusive because possible confounders (e.g., higher income gives more freedom of choice, differences in physical demands) have not been taken into account.

A comparison of selected socioeconomic characteristics by ethnic/racial group is provided in Table 4 (Census, 1990; Montgomery and Carter-Pokras, 1993). Compared to blacks and Hispanics, whites are more likely to earn higher incomes, be better educated, work in a white collar job, own their own home, and have health insurance, and less likely to live below the poverty line (Census, 1990; DHHS, 1991b).

Comparison of Smoking Prevalence and Habits

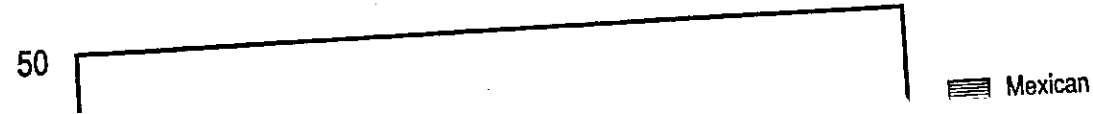
Cigarette smoking is a known risk factor for many diseases and conditions, such as emphysema, lung cancer, and heart disease, as well as low birth weight, and infant mortality (DHHS, 1985, 1991a,b; Montgomery and Carter-Pokras, 1993). The percentage of adult cigarette smokers according to gender and ethnicity for 1985 through 1987 is shown in Figure 3 (DHHS, 1991a).

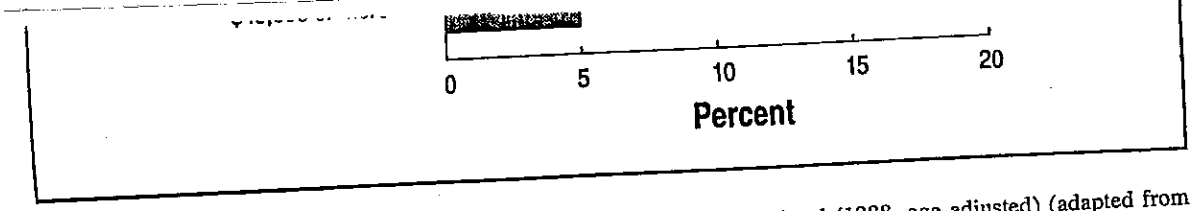
In the years 1985 through 1987, smoking rates were similar among non-Hispanic white males (31%) and females (29%). Compared to white males, age-adjusted rates of cigarette smoking were higher among non-Hispanic black (39%), American Indian (37%), and Puerto Rican



Insert Fig. 2

FIGURE 2. Percentage of people who report limitation of major activity, by income level (1988, age-adjusted) (adapted from *DHHS, 1991a*).





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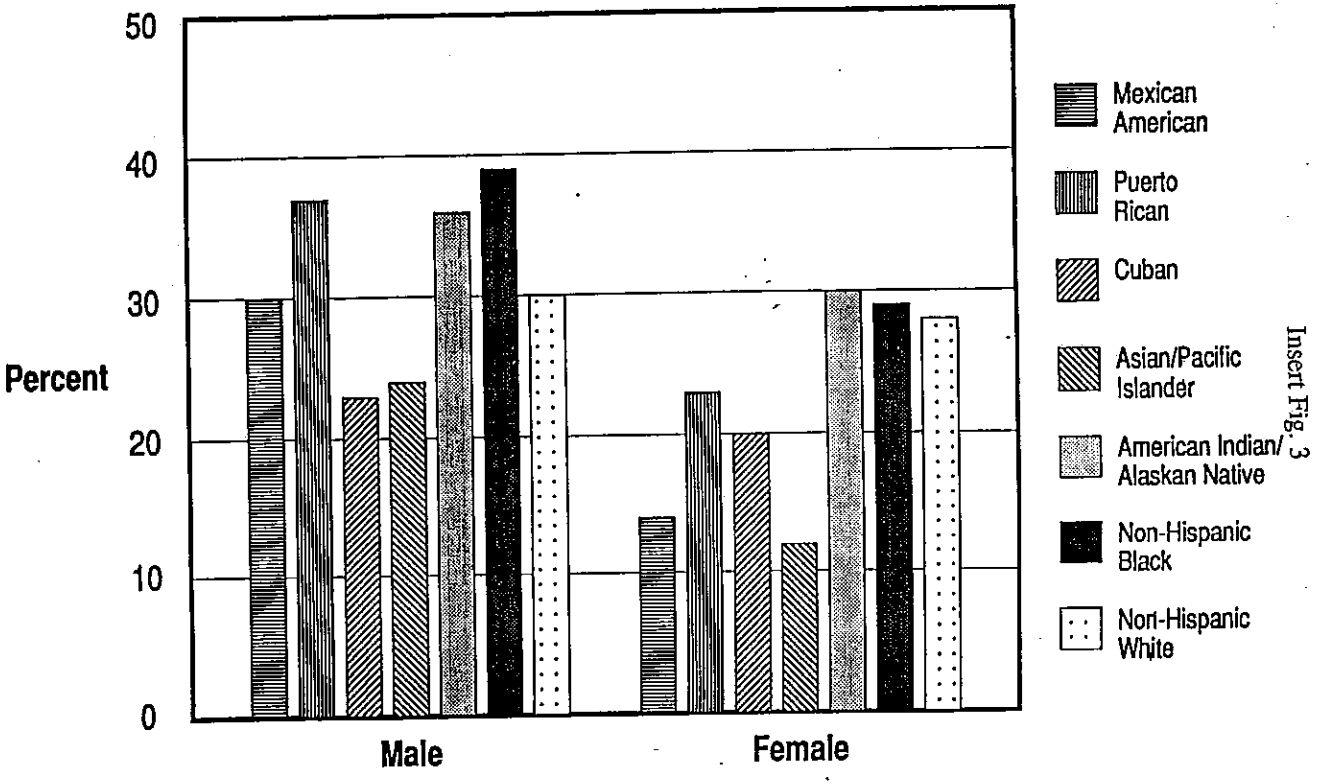

NOTE: Percents are age-adjusted annual averages.

FIGURE 3. Percentage of adult cigarette smokers according to gender and ethnicity for 1985—1987 (adapted from DHHS, 1991a).

Insert Fig. 3

TABLE 4. Comparison of Selected Socioeconomic Characteristics by Ethnic/Racial Group (Adapted from U.S. Bureau of Census, 1990, and from Montgomery and Carter-Pokras, 1993)

Parameter	Total	White	Black	Hispanic
Income (Census, 1990)				
Median household income	$27,225	$28,781	$16,407	$20,359
Households below poverty level	13.1%	10.1%	31.6%	26.8%
Education (Census, 1990)				
Median years of school completed	12.7 yrs	12.7 yrs	12.4 yrs	12.0 yrs
Completed 4+ years of college	20.3%	20.9%	11.3%	10.0%
Completed < 12 years of school	23.8%	22.3%	36.7%	49.0%
Occupation (Montgomery and Carter-Pokras, 1993)				
Managerial and professional specialty	N/A	27.7%	16.3%	13.0%
Technical, sales, and administrative support	N/A	31.2%	28.3%	24.5%
Service	N/A	12.4%	23.3%	20.3%
Precision production, craft and repair	N/A	11.7%	8.6%	12.9%
Operators, fabricators, and laborers	N/A	13.9%	21.7%	23.4%
Farming, forestry, and fishing	N/A	3.1%	1.9%	5.7%
Other (Census, 1990)				
Percent of workers unemployed	5.5%	4.7%	11.7%	8.2%
Percent covered by health insurance	86.2%	87.4%	79.6%	69.9%
Percent who live in own home	64.0%	67.0%	49.0%	40.0%

(37%) males, and lower among Asian (25%) and Cuban (24%) males. Among women, age-adjusted smoking rates were about the same for non-Hispanic whites (29%), non-Hispanic blacks (29%), and American Indians (31%), with lower rates for Puerto Ricans (23%), Cubans (20%), Mexican Americans (16%), and Asians (12%).

Smoking tends to be more prevalent in lower socioeconomic groups (CDC, 1990). Educational level is the major demographic predictor of whether an individual will smoke cigarettes (i.e., lower educational attainment is positively correlated with smoking), although gender (i.e., females more likely to take up smoking), age (i.e., young adults with low educational levels more likely to smoke), and race (i.e., black males more likely to smoke) are also important descriptive variables (CDC, 1989a; Pierce et al., 1989a; Escobedo et al., 1990).

Although smoking prevalence continues to decrease across the U.S. population, the rate of decline is not uniform (Fiore et al., 1989). If current trends persist, only about 40 million Americans (22%) will be smokers in the year 2000 (Pierce et al., 1989b). Approximately 30% of those who have not graduated from high school will smoke, while only 10% of college graduates will be smokers. Smoking prevalence is predicted to decrease to 20% among men and 23% among women, and to 25% among blacks and 20% among whites (Pierce et al., 1989b).

haracteristics by
ureau of Census,
ras, 1993)

Black	Hispanic
6,407	$20,359
1.6%	26.8%
12.4 yrs	12.0 yrs
11.3%	10.0%
36.7%	49.0%
16.3%	13.0%
28.3%	24.5%
23.3%	20.3%
8.6%	12.9%
21.7%	23.4%
1.9%	5.7%
11.7%	8.2%
79.6%	69.9%
49.0%	40.0%

s. Among women, age-
es (29%), non-Hispanic
o Ricans (23%), Cubans

groups (CDC, 1990).
n individual will smoke
with smoking), although
g adults with low educa-
likely to smoke) are also
cobedo et al., 1990).

. population, the rate of
t, only about 40 million
9b). Approximately 30%
ile only 10% of college
ease to 20% among men
ng whites (Pierce et al.,

Potentially significant differences in smoking habits, as well as smoking rates, have been documented between blacks and whites. Compared to whites, blacks smoke fewer cigarettes per day, but tend to smoke cigarettes that are higher in tar, nicotine, and mentholation (Kabat et al., 1991). The significance of these observed differences for smoking-related disease is not well understood.

Smoking exacerbates the health effects of certain carcinogens, as for example asbestos (McDonald and McDonald, 1987) and radon (Nazaroff and Teichman, 1990). Synergistic interactions between smoking and other environmental agents may also occur and should be taken into account when interpreting differentials in environmental health.

Environmental Tobacco Smoke (ETS)

Exposures to tobacco smoke in various environments occupied by smokers can have adverse health effects on nonsmokers (U.S. EPA, 1992b). Environmental tobacco smoke (ETS) has been classified as a "known human" (class A) carcinogen, and is estimated to cause as many as 3,000 lung cancer deaths annually among nonsmokers of both sexes. In addition, ETS is causally associated with a wide range of noncancer diseases and disorders of the respiratory system, including an increased risk of lower respiratory tract infections (e.g., pneumonia, bronchitis, bronchiolitis), additional episodes and increased severity of asthma in children who already have the disease, increased prevalence of respiratory symptoms of irritation (e.g., cough, sputum, wheeze), and a small but statistically significant reduction in lung function (U.S. EPA, 1992b). Epidemiologic evidence suggests that ETS exposure increases the number of new cases of asthma in children who have not previously exhibited symptoms (U.S. EPA, 1992b).

Significant differences in exposure to ETS among socioeconomic and ethnic/racial groups might cause differentials in respiratory health. Although documented variations in smoking prevalence and habits suggest the possibility of systematic variations in ETS exposures, a paucity of data exist to test this hypothesis.

Occupational Cancer Comparisons

In their classic paper, *The Causes of Cancer*, Doll and Peto (Doll and Peto, 1984) estimate that occupational exposures cause approximately 4% (range 2 - 8%) of all cancer deaths. This figure is in agreement with several other published estimates (Wynder and Gori, 1977; Higginson and Muir, 1979; Gough, 1989). Doll and Peto estimated that in 1978, occupational exposures caused 25% of mortality from cancer at sites in the respiratory tract other than the lung (e.g., pleura, nasal sinuses), 15% from both lung cancer and cancer of the mesentery/peritoneum, and 10% respectively from skin cancer (other than melanoma), bladder cancer, and leukemia (Doll and Peto, 1984).

Occupation is a well-documented risk factor for many forms of cancer, and a substantial body of literature exists on cancer mortality by industry type (Wynder and Gori, 1977; Higginson

and Muir, 1979; Doll and Peto, 1984; Gough, 1989; Kipen et al., 1991). However, occupational health data by class and race are sparse. In a review of the occupational cancer epidemiology literature from 1984 through 1987, Kipen et al. (1991) found that only 14 (12%) of 116 studies provided data on a non-white group. Moreover, data on non-whites tended to be based on much smaller numbers of workers than comparable information for white populations. The authors conclude *"that the published literature does not contribute much meaningful data toward understanding the complex relationships among occupation, cancer, and race"* (Kipen et al., 1991).

They go on to argue that it is not appropriate to extrapolate risks identified in white populations to blacks because of some important black-white differences in: 1) potential for confounding interactions among risk factors (e.g., socioeconomic status, smoking, occupation, diet, sexually transmitted diseases, attitudes and awareness toward cancer); 2) historical employment patterns (e.g., fewer blacks in certain industries and more in certain hazardous jobs) that may be related to disparity in exposures, even for workers in the same job classification working in the same plant; 3) size or nature of the "healthy worker effect," which may be larger than average in non-whites and in populations that are poorer and that are in worse health; and 4) the age-structure of the workforce, such that a high proportion of younger workers could cause non-white mortality ratios to be artificially low relative to whites (Kipen et al., 1991). The validity of extrapolating effects in white workers to black workers deserves further study. Research may be hampered, however, by the small size of the minority workforce in some occupations.

DIFFERENCES IN MORBIDITY AND MORTALITY

In this section, we consider differentials in disease and death rates, focusing on those that might be associated with environmental exposures.

Differences in Disease and Death Rates

Rates of disease and death exhibit clear and dramatic differences by race, as do low birthweight and birth defects (DHHS, 1985, 1991a,b; Montgomery and Carter-Pokras, 1993). Ethnic/racial differences for some of the leading causes of death are shown in Table 5 (Montgomery and Carter-Pokras, 1993; Yu et al., 1985; Maurer et al., 1990; Indian Health Service, 1991; NCHS, 1992), while differences in cancer mortality are shown in Table 6 (Pickle et al., 1990). When interpreting these data, it is important to keep in mind that there are substantial reporting problems for race on both census records and death certificates. Differences may be caused by a combination of factors, including economic, social, cultural, biological, and environmental variables.

The contribution of environmental exposures, if any, to these differentials is largely unknown. Establishing a causal relationship between exposures to an environmental agent and subsequent disease or injury in humans is usually difficult unless the link is very strong (e.g.,

TABLE 5. Ratio of Age-adjusted Death Rates (Minority-to-Whites) for Selected Causes of Death by Race (Adapted from Montgomery and Carter-Pokras, 1993)

Cause of Death	Ratio to Whites						
	Black[a]	American Indian and Alaskan Native[b]	Mexican American	Puerto Rican[c]	Chinese American[d]	Japanese American[d]	Filipino American[d]
All causes	1.58	1.13	0.92	0.97	0.63	0.52	0.45
Heart disease	1.43	0.86	0.75	0.88	0.54	0.42	0.42
Malignant neoplasms	1.33	0.70	0.68	0.70	0.76	0.60	0.40
Cerebrovascular diseases	1.89	0.96	0.90	0.69	0.76	0.76	0.66
Chronic obstructive pulmonary disease	0.84	0.70	0.42	0.76	0.50	0.34	0.31
Pneumonia and influenza	1.52	1.38	1.01	1.60	0.81	0.73	0.59
Diabetes mellitus	2.30	2.87	2.11	1.91	0.81	0.64	0.49
Chronic liver disease and cirrhosis	1.67	3.62	1.56	3.64	0.42	0.34	0.29
Atherosclerosis	1.03	0.91	0.96	0.58	0.57	0.41	0.25
Suicide	0.59	1.19	0.73	0.81	0.64	0.62	0.30
Accidents	1.30	2.37	1.27	0.69	0.34	0.44	0.39

[a]National Center for Health Statistics, 1992, for 1989.
[b]Indian Health Service, 1991, for 1988.
[c]Maurer, 1990, for 1979-1981.
[d]Yu, 1985, for 1980.

TABLE 6. Relative Cancer Mortality Rates (1970s) for Males and Females (Adapted from Pickle et al., 1990)

MALES					
	Ethnicity/Race				
Cancer Site(s)	White	Black	American Indian	Chinese	Japanese
All sites	1.00	**1.33**	0.49	0.81	0.65
Lung	1.00	**1.26**	0.36	0.73	0.46
Colon	**1.00**	0.93	0.36	0.88	0.73
Prostate	1.00	**2.05**	0.58	0.34	0.37
Pancreas	1.00	**1.26**	0.52	0.70	0.74
Stomach	1.00	1.95	0.99	1.19	**3.13**
Leukemia	**1.00**	0.83	0.38	0.61	0.47
Bladder	**1.00**	0.83	0.24	0.47	0.43
Non-Hodgkin's lymphoma	**1.00**	0.69	0.39	0.58	0.62
Rectum	1.00	0.95	0.47	1.05	**1.19**
Brain and CNS	**1.00**	0.56	0.27	0.36	0.29

FEMALES					
	Ethnicity/Race				
Cancer Site(s)	White	Black	American Indian	Chinese	Japanese
All sites	1.00	**1.16**	0.61	0.70	0.61
Breast	**1.00**	0.97	0.36	0.45	0.35
Lung	1.00	1.00	0.43	**1.04**	0.48
Colon	1.00	**1.06**	0.41	0.61	0.57
Ovary	**1.00**	0.79	0.42	0.48	0.50
Pancreas	1.00	**1.33**	0.66	0.81	0.83
Leukemia	**1.00**	0.88	0.43	0.66	0.49
Cervix uteri	1.00	**2.78**	1.83	0.91	0.64
Corpus uteri	1.00	**1.74**	0.55	0.62	0.48
Non-Hodgkin's lymphoma	**1.00**	0.59	0.43	0.61	0.68
Stomach	1. 00	1.73	1.04	1.42	**3.25**

Note: Highest mortality rate for each cancer is in bold.

radon-induced lung cancer in uranium miners). The task of linking exposures and effects is typically complicated by several factors, including: 1) incomplete understanding of the etiology of many diseases; 2) the wide range of non-environmental causes of most diseases to which environmental exposures may contribute; 3) exposure of most people to a vast array of environmental agents, few of which are quantified; 4) the long latency period from exposure to effect, often 20 years or more; 5) the multiple health effects caused by some environmental agents; 6) the occurrence of a single health effect after multiple exposures to either a single agent or a mixture of agents; and 7) substantial differences among humans in their biological susceptibility to exposures.

Lung Cancer Comparisons

Lung cancer accounts for about 14% of all cancer incidence and 23% of all cancer deaths (NCHS, 1984). Among black males, lung cancer mortality is 26% higher than in whites, and

more than 50% higher than in American Indians and Chinese- and Japanese-Americans (see Table 6) (Pickle et al., 1990). Similarly, the incidence of lung cancer in black men is higher than in whites, Hispanics, Asians, or Native Americans (Pickle et al., 1990). For women, lung cancer mortality is about the same for whites, blacks, and Chinese-Americans, but more than 50% lower for American Indians and Japanese-Americans (Pickle et al., 1990). Incidence rates for cancer of the lung and bronchus from 1973 through 1986 are presented by race (black/white) and gender in Figure 4 (CDC, 1989b).

Observed differences in cancer incidence/mortality (at many sites, including the lung) between blacks and whites have been linked to socioeconomic factors, such as income, education, and occupation (Devesa and Diamond, 1983; Baquet et al., 1991; Duke, 1991; Freeman, 1991; Gibbons, 1991). For example, Devesa and Diamond (1983) have shown that for black and white males, higher income and educational levels are associated with lower lung cancer incidence rates. As shown in Table 7 (Devesa and Diamond, 1983), lung cancer rates in black men were significantly higher than in white men before adjustment for socioeconomic variables. After adjustment for education, rates for black men were still higher, but not significantly. After adjustment for income, they were non-significantly lower (Devesa and Diamond, 1983).

Approximately 85% of lung cancer cases are attributable to cigarette smoking (NCHS, 1984). Thus, differences in smoking prevalence rates (Figure 3) may in part explain the differentials in lung cancer rates by class and race. It has also been suggested that African-American nonsmokers may be more likely than nonsmoking whites to develop lung cancer (Schneiderman et al., 1990). The apparent elevation in lung cancer in non-smoking African-Americans has not been adequately explained.

Other documented causes of lung cancer include occupational exposures to ionizing radiation, asbestos, radon, and certain chemicals (e.g., chromium) (Wynder and Gori, 1977; Higginson and Muir, 1979; Doll and Peto, 1984; Gough, 1989). Although most of the variability in lung cancer incidence can be explained by smoking, and to a lesser extent by occupational exposures, it is likely that airborne carcinogens (e.g., diesel exhaust, environmental tobacco smoke) in both the ambient environment (e.g., outdoors in urban areas) and indoor non-industrial settings (e.g., inside residences and offices) also contribute to development of lung cancer. Their effect, however, is not likely to exceed 5% of lung cancer rates, and may be much smaller (Doll and Peto, 1984; Gough, 1989).

Relative risks of lung cancer from environmental exposures to air pollution are expected to be less than 2% in most cases, if they exist at all. It is difficult to demonstrate effects of this magnitude, given the technology, methods, and funding limitations of analytic epidemiology. Epidemiologic studies of the relationship between air pollution and lung cancer, either in the general population or in specific subgroups defined by income and race, are complicated by the

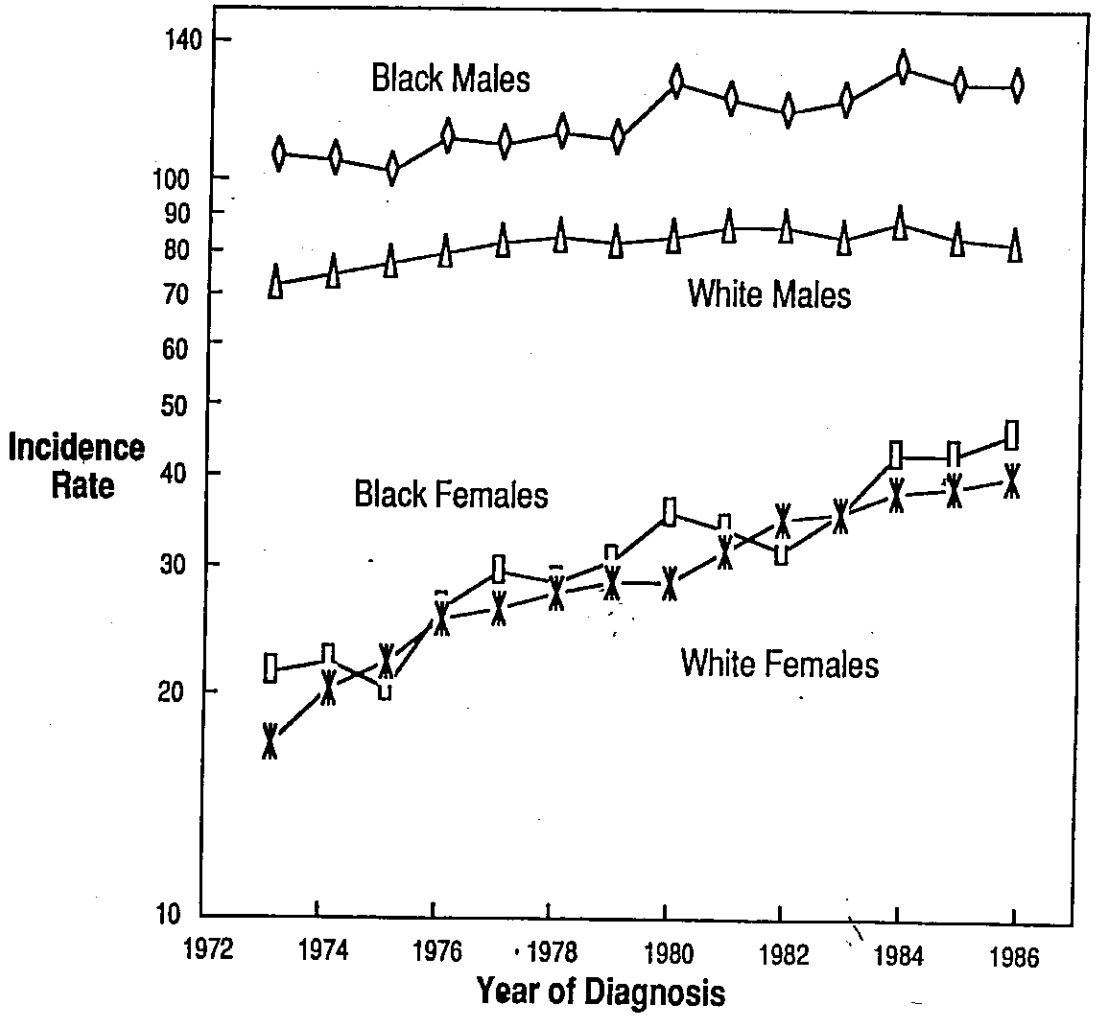

FIGURE 4. Incidence rates (per 100,000 persons, age-adjusted) for cancer of the lung and bronchus from 1973 through 1986 by gender and race (adapted from CDC, 1989b).

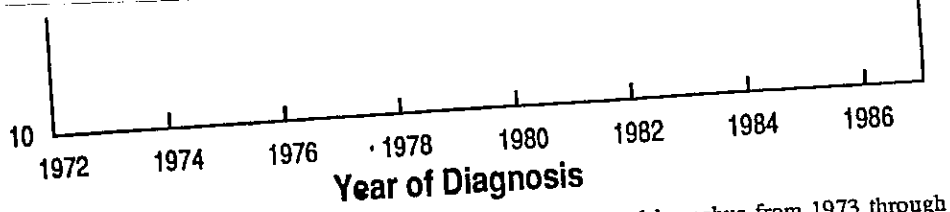

FIGURE 4. Incidence rates (per 100,000 persons, age-adjusted) for cancer of the lung and bronchus from 1973 through 1986 by gender and race (adapted from CDC, 1989b).

TABLE 7. Comparison of Lung Cancer Incidence Rates and Relative Risk by Gender and Race, 1969-1971 (Adapted from Devesa and Diamond, 1983)

Adjustments to Data	Males				Females			
	Incidence Rate[a]		Statistically Significant Difference	Black/White Relative Risk	Incidence Rate[a]		Statistically Significant Difference	Black/White Relative Risk
	White	Black			White	Black		
Adjusted for Age and Geographic Area	73.5	80.6	Yes ($p < 0.001$)	1.10	16.7	15.8	No	0.95
Adjusted for Age, Area, and Education	74.3	77.1	No	1.04	16.8	15.6	No	0.93
Adjusted for Age, Area, and Income	75.5	72.5	No	0.96	17.0	14.6	Yes ($p < 0.05$)	0.86
Adjusted for Age, Area, Education, and Income	75.7	71.8	No	0.95	17.0	14.6	Yes ($p < 0.05$)	0.86

[a]Annual average rates per 100,000 persons in six geographic areas (7 SMSAs).

need to control for the confounding effects of variables such as active and passive smoking, occupation, education, and age. The problem of detecting small increases in relative risks is made even more difficult by misclassification of exposures, a shortcoming inherent in cancer epidemiology, because good measures of cumulative exposures to carcinogens are not usually available.

Chronic Obstructive Pulmonary Disease (COPD)

Chronic obstructive pulmonary disease (COPD), which includes chronic bronchitis and associated conditions, accounted for about 3.5% of all deaths in the United States in 1987, up from 1.6% in 1970 (NCHS, 1990). An estimated 10% of all Americans are afflicted with COPD (NCHS, 1990).

COPD is more prevalent in whites than blacks, and more prevalent in blacks than in other racial and ethnic minorities (Gillum, 1990; NCHS, 1990). Among African-Americans and whites, the incidence of COPD is significantly higher in low-income groups. The prevalence of chronic respiratory conditions by ethnicity and income is given in Table 8 (NCHS, 1990). At incomes both above and below $20,000, whites exhibited a higher prevalence of chronic bronchitis and emphysema than blacks, who, in turn, had higher rates than other minorities.

Asthma Morbidity and Mortality

Asthma affects 10 to 20 million people in the United States. From 1980 to 1987, the prevalence of asthma in the U.S. increased 29%, and death rates increased 31%. The increased incidence of asthma appears in all segments of the population, but is most notable in children and blacks (Hammer et al., 1976; Speizer et al., 1980; Mak et al., 1982; Mullally et al., 1984; Goldstein and Weinstein, 1986; Gergen et al., 1988; Sly, 1988; Gillum, 1990; NCHS, 1990; Schwartz et al., 1990a,b; Weiss and Wagener, 1990a,b; Weitzman et al., 1990; Carr et al., 1992).

In their examination of the 1981 National Health Intervention Survey, Weitzman et al. (1990) found that black children had 1.7 times the asthma rate of white children. Similar estimates of the association between race and asthma were observed by Schwartz et al. (1990a) in their evaluation of the Second National Health and Nutrition Examination Survey (NHANES). African-American children and adults are more likely to be hospitalized and to die from the disease than their white counterparts (Mak et al., 1982; Mullally et al., 1984; Gergen et al., 1988; Gillum, 1990), particularly when asthma onset occurs before the age of six (Schwartz et al., 1990b; Weitzman et al., 1990).

Recent data suggest that asthma death rates are nearly three times as high in blacks as in whites (Gillum, 1990). Nearly 80% of asthma deaths occur in urban areas, with data from New York City and Cook County, Illinois (Chicago area) driving the trends seen in the U.S. (Gillum, 1990; Weiss and Wagener, 1990a). Hospitalization and mortality rates among blacks and Hispanics were 3 to 5.5 times those of whites in a recent study in New York City (Carr et al., 1992).

TABLE 8. Prevalence of Chronic Respiratory Conditions (per 1000 persons) for 1985-1987 by Income and Ethnicity/Race (Adapted from NCHS, 1980)

	Family Income								
	White			Black			Other		
	Total	< $20,000	> $20,000	Total	< $20,000	> $20,000	Total	< $20,000	> $20,000
TOTAL	102.7	125.8	95.0	81.0	97.1	58.0	53.0	51.3	53.3
Chronic Bronchitis	53.5	64.6	50.1	35.3	42.6	28.1	27.1	27.0	24.6
Asthma	39.6	42.7	39.7	42.2	49.9	29.2	22.6	21.1	24.6
Emphysema	9.6	18.5	5.2	3.5	4.6	0.7	3.3	3.2	4.1

Among the reported associations between asthma and specific explanatory variables are the following: race (African-American); gender (male); poverty; maternal smoking (more than 1/2 pack per day); low birth weight; neonatal intensive care; maternal age at birth (< 20 years); maternal education (< high school); living in a large family; not residing with biological parent; living in a small space; and nutritional status (Mullally et al., 1984; Schwartz et al., 1990a; Weiss and Wagener, 1990a). In the Weitzman analysis (Weitzman et al., 1990), only gender, maternal smoking, low birth weight, and living in a large family were found to be significantly predictive of asthma, after controlling for variables relating to poverty. Others have found that perinatal, socioeconomic, and nutritional factors account for some, but not all, of the disparity between black and white children in the prevalence of asthma (Schwartz et al., 1990a; Gold et al., 1993).

Allergy to one or more aeroallergens can be shown in approximately 85% of all asthma cases (Platts-Mills et al., 1987; Chapman et al., 1988). Compared to whites, African-Americans tend to exhibit increased allergic skin reactivity (Gergen and Turkeltaub, 1987), and higher levels of serum IgE antibody (Grundbacher, 1975).

Platts-Mills et al. studied the prevalence of serum IgE antibody to dust mites, cats, cockroaches, grass pollen, and ragweed pollen in 102 patients with acute respiratory obstruction (Platts-Mills et al., 1987; Chapman et al., 1988). Most asthmatic patients, black and white, had high levels of IgE antibody to dust mites, cats, and cockroaches. Serum IgE to cat dander was found to be a risk factor only among white patients, while serum IgE to cockroaches was a risk factor only among black patients. The authors conclude that *"these racial differences were highly significant and appeared to correlate with socioeconomic and housing conditions"* (Chapman et al., 1988). Bernton et al. (1972) and Kang (1976) showed that half of the economically disadvantaged asthmatic patients in large urban centers studied in the United States exhibited specific IgE antibodies and positive skin tests to cockroaches.

Poor housing conditions can lead not only to increased numbers of cockroaches, but also to increased house dampness, which can create conditions conducive to mites, molds, and fungi (Kales et al., 1991; Gold, 1992). Both home dampness and fungi have been associated with an increased prevalence of wheeze, cough, and phlegm in adults and children (Kales et al., 1991; Gold, 1992).

Sporik and his colleagues have suggested that exposure to house-dust mite allergens at an early age is an important determinant of subsequent development of asthma (Sporik et al., 1990). They state that *"increased exposure to dust mites and other indoor allergens may be a factor contributing to the recent increases in morbidity and mortality associated with asthma"* (Sporik et al., 1990). If this is true, then systematic differences in exposures to indoor allergens (e.g., dust mites, cockroaches) may partially explain observed asthma differentials by class and ethnicity/race.

The etiology of asthma is not well understood. Currently, the causes of the general increase in asthma across all segments of the population and the differences between blacks and whites are the subject of much attention and conjecture. It seems likely that the observed trends in asthma prevalence are the result of multiple factors, including genetics, socioeconomic status, indoor and outdoor air pollution, both medicinal and illegal drug use, changes in self-administered treatment, and possibly viruses and other infectious agents.

Differences in Lung Function

Lung volumes vary significantly across race and ethnic groups, and vital capacity is in approximately direct proportion to height (Woolcock et al., 1972; Coultas et al., 1993). For the same standing height, African-Americans have shorter trunks, smaller thoracic diameters, and lower forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) than whites. Blacks also tend to have consistently lower lung function, even after controlling for gender, age, standing height, and body mass (Woolcock et al., 1972; Binder et al., 1976; Schoenberg et al., 1978; Wall et al., 1982; Strope and Helms, 1984; Crapo et al., 1988; Marcus et al., 1988; Schwartz et al., 1988a; Coultas et al., 1993).

The normal ranges of lung function in Hispanics, Asians, and Native Americans have received less attention (Coultas et al., 1993). Differences between whites and Japanese Americans (Marcus et al., 1988; Coultas et al., 1993), Native Americans (Wall et al., 1982; Crapo et al., 1988; Marcus et al., 1988), and Latinos (Hsi et al., 1979; Coultas et al., 1988, 1993) have been reported, but results are inconsistent, especially for Hispanics and Native Americans (Coultas et al., 1993). In general, lung function for these groups tends to be intermediate between those of whites and blacks (Coultas et al., 1993).

It is unlikely that observed ethnic/racial differences in lung function are due to differences in mechanical properties of the lung (Woolcock et al., 1972; Schwartz et al., 1988b). When residual volume and FEV1 are related to total lung capacity (TLC) in different racial groups, similar results are obtained (Woolcock et al., 1972). Higher values of FEV1/FVC have been reported in black as compared to white males, but no difference was found between black and white children (ages 6 to 11) in FEV1/FVC for a given level of FVC (Dockery et al., 1985). After controlling for FVC, another group of investigators found no difference in FEV1/FVC between blacks and whites in any age or gender category, and controlling for FVC virtually eliminated the observed differences in pulmonary function (Schwartz et al., 1988b). Thus, for the same lung volume, spirometric measures of lung function appear to be similar across blacks and whites for both males and females.

Although genetic differences are generally thought to be the primary cause of ethnic/racial differences in lung volumes/lung function, environmental factors may also play a role (Woolcock et al., 1972; Seltzer et al., 1974; Hasselblad et al., 1981; Tager et al., 1983; Dockery et al., 1985; Kaufmann et al., 1989; Schwartz et al., 1990a; Coultas et al., 1993). Maternal smoking (Hasselblad et al., 1981; Tager et al., 1983), use of gas-fired cooking

stoves (Seltzer et al., 1974; Hasselblad et al., 1981; Tager et al., 1983), and exposures to airborne pollutants, such as environmental tobacco smoke (U.S. EPA, 1992b) and allergens in house dust (Sporik et al., 1990; Gold, 1992), have been associated with reduced lung function.

The effect of ethnic/racial differences in lung function on health risks presented by air pollution is unknown. There is evidence, however, that exposures to several of the criteria pollutants (e.g., ozone, nitrogen dioxide, particles) decrease pulmonary function in both children and adults (Shy et al., 1973; Chapman et al., 1976; Dodge et al., 1985). If exposures occur over relatively short time spans (e.g., hours or days), lung function appears to return to normal after exposure ceases. If exposures are persistent, however, lung function values can remain depressed (Chapman et al., 1976; Schoenberg et al., 1978).

Most human clinical studies using controlled exposures to air pollution (e.g., NAAQS pollutants) have not included members of minority groups. A recent clinical study (92 black females, 94 white females; 92 black males, 93 white males) investigated the possibility of gender and race differences in response to ozone exposures (Seal et al., 1993). Findings suggest no statistically significant differences by race or gender for FEV1, specific airway resistance (SRaw), or cough in response to 2.5 hour exposures, with intermittent exercise, at 0.12, 0.18, 0.24, 0.30, and 0.40 ppm ozone. Concentration-response curves for white males were consistent with previous studies.

DIFFERENCES IN SUSCEPTIBILITY TO AIR POLLUTION EFFECTS

Segments of the population could be at increased risk not only because of higher exposures, but also because of enhanced susceptibility to the effects of exposures. This section briefly surveys the scientific evidence bearing on this question.

It is important to acknowledge explicitly the sensitivity of data that purport to show differences in people across cultures and races, especially when based on "genetic" factors. Ethical considerations must always be taken into account in the differentiation of people by cultural and racial attributes (NRC, 1989).

Susceptibility Based on Inherent Genetic Variability

Differences in genetic make-up may increase the likelihood that some individuals will respond biologically to a particular environmental insult. This enhanced susceptibility might be caused by the presence of one or more genetic alleles that "promote" adverse effects from exposures or by the absence of alleles that provide "protection" (e.g., resistance) from environmentally induced disease. It is not clear whether or how these genetic factors are associated with specific ethnic/racial subgroups. Among the genetic differences that may give rise to differential vulnerability to air pollution exposures are: deficiency of the enzyme glucose-6-phosphate dehydrogenase (G-6-PD) in people from Africa, Asia, and the Mediterranean (e.g., possible increased proneness to effects of ozone and nitrogen dioxide); sickle cell anemia in African-

Americans (e.g., may increase risks from carbon monoxide exposures); and slow rate of acetylation in the liver (e.g., risk factor for bladder cancer associated with exposure to aromatic amines) (Calabrese, 1984, 1986; Brain et al., 1988; Polednak, 1989; Utell and Frank, 1989; Grandjean et al., 1991; Rios et al., 1993).

Age and Gender as Susceptibility Factors

Substantial evidence shows that age and gender are important determinants of biological susceptibility to specific environmental agents. It is well recognized that the very young (e.g., fetuses, neonates, infants, children) and the elderly are more vulnerable to the effects of some air pollutants, including aeroallergens (e.g., pollen), microorganisms (e.g., bacteria, fungi, viruses), and pesticides (e.g., organophosphates). Pregnant women, and particularly their fetuses, are an especially sensitive group due to physiologic changes during pregnancy, and may be at increased risk from exposures to pollutants such as carbon monoxide and pesticides that are cholinesterase inhibitors (Calabrese, 1984, 1986; Brain et al., 1988; Polednak, 1989; Utell and Frank, 1989; Grandjean et al., 1991; Rios et al., 1993).

Other Factors Affecting Biological Susceptibility

Circumstances beyond genetics, age, and gender can also affect biological susceptibility to environmental exposures (Calabrese 1984, 1986; Brain et al., 1988; Polednak, 1989; Utell and Frank, 1989; Grandjean et al., 1991; Rios et al., 1993), including those listed below.

- Preexisting disease (e.g., diabetes, asthma) that raises the susceptibility of some individuals to the adverse effects of exposures.
- Inadequate diet (e.g., vitamin and mineral deficiency, low protein) that impairs host defense systems.
- Lifestyle factors, such as alcohol consumption, smoking, and illegal drug use, that damage the respiratory and cardiovascular systems.
- Exposure to multiple environmental hazards, at work and at home, which may cause additive or synergistic health effects.
- Inadequate access to health care, which may delay recognition, diagnosis, and treatment of environmentally induced disease.
- Possibly pyschosocial-induced stress, as might be caused by unemployment, under-employment, poverty, inadequate living and working conditions, and language problems, although these matters have not been rigorously investigated.

CONCLUSIONS

The possibility that environmentally related health risks fall disproportionately on those who are disadvantaged, including racial and ethnic minorities, has been the subject of much recent debate and concern. Despite the absence of systematically collected data, the weight of the available evidence supports two observations. First, many disadvantaged and minority groups live in areas with poor outdoor air quality, and these same groups may encounter high levels of certain indoor air contaminants (e.g., aeroallergens, environmental tobacco smoke) in their residences and/or workplaces. Second, national data on health status provide clear proof that disease and death rates are related to social class and ethnicity/race. The extent to which exposures to air pollutants and other environmental agents contribute to these observed differences, however, is uncertain.

Typically, it is difficult to establish a causal link between environmental exposures and resulting disease or injury in the general population. The difficulties arise from: 1) the complexity of environmentally induced chronic disease (e.g., multiple exposures, multiple causative agents, long latency periods, variability in response to exposures); and 2) the lack of appropriate data on human exposures, doses, and health effects. The problem is compounded for disadvantaged and minority populations because of their smaller numbers and the paucity of information about disparities in environmental exposures and related effects.

Despite the lack of proof that the impaired health status of minority and disadvantaged populations is caused in part by non-occupational air pollution, there is ample reason for concern. In the few cases where we have sufficient information to make informed estimates of national exposures (e.g., outdoor air pollutants like ozone and carbon monoxide, blood lead levels in children), there is strong presumptive evidence that disadvantaged minority communities routinely experience above-average environmental exposures.

Still, fundamental questions remain. Are environmental health risks (e.g., cancer, respiratory disease, neurotoxic effects, birth defects) systematically higher for disadvantaged and minority populations? And, if so, is the excess a result of higher environmental exposures? In our opinion, the answer to both questions is a qualified "Yes."

More definitive resolution of these important questions must be high on the nation's environmental health research agenda. Adequate resources should be devoted to conducting well-designed scientific studies that will aid decision makers in answering the following questions:

- Are disadvantaged and minority populations at increased risk of adverse health effects from air pollution and other environmental exposures?
- If so, what are the relative contributions of class and race to the observed differences?

- Are health risk disparities caused by differences in exposures, in susceptibilities, or both?

- What are the relative costs and benefits of societal strategies to prevent or reduce unacceptable environmental health risks in disadvantaged and minority communities?

This need for additional research is pressing, and cannot be reconciled with further delay in addressing issues of environmental equity. Available evidence is more than sufficient to justify prompt initiation of needed studies. Areas (e.g., inner cities) and populations (e.g., occupational cohorts) that seem to be at high risk should be identified and special emphasis placed on understanding and reducing exposures.

RECOMMENDATIONS

- Expand efforts to determine the environmental health risks experienced by disadvantaged populations, including ethnic/racial minorities, and set priorities for research and risk prevention/reduction activities based on relative risks.

- Reanalyze existing sources of data, including large field studies and databases, to investigate differences according to class and race. Scientists with relevant information should be encouraged to share their data.

- Modify survey methods and techniques for studying different ethnic/race, income, and linguistic groups to improve the quality of information collected, improve response rates, and enhance comparability across studies.

- Collect, analyze, and report environmental and health data by cross-tabulated socioeconomic indicators (e.g., income, education, occupation) and ethnic/racial group. New databases should emphasize acquisition of information relevant to differential exposures and/or environmentally induced diseases in important population subgroups (e.g., communities near major sources, occupational cohorts, poor and disadvantaged groups, ethnic/racial minorities).

- Focus research on improved understanding of cumulative health risks from multiple exposures to the same agent, as well as from multiple exposures to different agents.

- Define and understand important determinants of exposure, dose, and effects in population subgroups (e.g., occupational cohorts, disadvantaged groups) that are at high risk from air pollution exposures.

- Examine the variability in time-activity patterns by socioeconomic status and ethnicity/race to determine whether significant and systematic differences exist, and, if they do, to document ramifications for air pollution exposures.

- Assess relevant non-industrial, indoor microenvironments, such as residences and offices, to ascertain whether significant and systematic differences exist in pollutant concentrations (e.g., ETS, aeroallergens) by socioeconomic status and ethnicity/race.

- Undertake disease-specific studies (e.g., COPD, asthma, lung cancer) to document the role of air pollution, if any, in morbidity and mortality, with special emphasis on understanding differentials by socioeconomic status and race.

- Conduct special studies of environmental risks to ethnic groups that have not traditionally received much attention, such as immigrants from Southeast Asia, Eskimos, Pacific Islanders, Puerto Rican Americans, Cuban Americans, Mexican Americans, and Japanese- and Chinese-Americans.

- Examine ethnic/racial differences in controlled exposure studies of acute (reversible) responses to specific environmental agents.

- Identify biological and non-biological factors that increase susceptibility to environmental agents and determine how much any systematic differences are related to socioeconomic status, and how much to ethnicity/race.

- Increase research emphasis on understanding the effects of genetics, nutritional status, preexisting health conditions, smoking, cultural and social behaviors, and activity levels/patterns on differential susceptibility to air pollution effects.

- Investigate differential air pollution effects on the quality of life (e.g., odors, reduced visibility, property values) for all important demographic groups.

- Develop a productive dialogue between public health agencies and disadvantaged and minority communities about the design, implementation, and reporting of environmental health research.

- Implement appropriate mitigation strategies once the critical cultural and social factors affecting environmental exposures for specific socioeconomic and ethnic/racial groups are understood.

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