SUSCEPTIBILITY TO ENVIRONMENTAL POLLUTANTS AMONG MINORITIES

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Susceptibility to environmental pollutants involves both biological and nonbiological factors. Individuals belonging to minority groups are much more likely to be subject to a number of these factors. This paper examines biological susceptibility of minorities to environmental pollutants and provides specific examples of susceptibility resulting from: genetic makeup; occupation; other factors such as compromised health status, exposure to mixtures of pollutants, substance abuse, and unemployment; and social inequality of access to health care, education, and communication skills. Recommendations are made for specific actions and for additional studies.

INTRODUCTION

The ultimate objective of pollution control programs is to reduce exposure to pollutants to levels that are not harmful, even to persons who are particularly sensitive. To achieve this long-range goal, it is necessary to identify harmful pollutants and their sources as well as factors that increase susceptibility to specific pollutants. These include both biological and nonbiological factors. Nonbiological factors that may increase susceptibility to environmental toxicants include: diet and substance abuse; occupational exposures; and multiple environmental exposures due to poverty, quality and location of housing, and employment and recreation in undesirable areas. Biological factors that increase susceptibility include both "genetic susceptibility" and a greater frequency of diseases.

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4. Abbreviations: AIDS, acquired immunodeficiency syndrome; CDC, Centers for Disease Control; G-6-PD, glucose-6-phosphate dehydrogenase; HIV, human immunodeficiency virus.

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Certain diseases may increase the likelihood that an individual will respond adversely to specific environmental and occupational exposures. The presence of these diseases may be due to genetic factors, to social, economic, environmental, and occupational factors, or to a combination of these factors. Because the distribution of these factors in society is also not random, the distribution of diseases associated with them is not random. Minorities and economically disadvantaged individuals are much more likely to be subject to multiple social, economic, environmental, and occupational conditions that lower their threshold to certain pollutants.

In this paper we examine biological susceptibility and provide specific examples of increased susceptibility resulting from: genetic makeup; occupation; other factors, such as compromised health status, exposure to mixtures of pollutants, substance abuse, and unemployment; and social inequality in access to health care, education, and communications skills. Finally, we recommend additional studies to examine factors that may increase biological susceptibility of minorities and the economically disadvantaged.

GENETIC SUSCEPTIBILITY: DIFFERENTIAL CAPABILITY TO RESPOND TO EXPOSURES

Genetic makeup is the most obvious inherent biological factor that increases susceptibility of some individuals to adverse effects of environmental and occupational exposures. A simple genetic trait is determined by a pair of separable factors, called the alleles of a gene. Because of the presence of specific gene alleles, some individuals experience adverse health effects when exposed to certain pollutants, while the same exposures are benign for individuals without the allele. Conversely, some alleles may be associated with biological resistance to pollutant effects. Individuals without this specific "protective" allele may be at greater risk from exposure to these pollutants than individuals who have it. While some alleles that affect health outcomes are randomly distributed in the population, others are associated with certain areas of the world and with specific ethnic groups.

Human variation is, in part, a result of genetic variation. Human genetic variation is accounted for by approximately 20,000 polymorphic proteins. Currently, approximately 50 polymorphic traits are thought to significantly influence susceptibility to environmental agents. A subset of these traits (sickle-cell trait is one) interacts with environmental factors (see Table 1). The interaction of biological and environmental factors can result in adverse health effects making ecogenetic research difficult.

The concept of "race" implies that, within a particular species, subpopulations possess distinctive hereditary traits that permit biologically relevant subclassifications. While "race" was a dominant paradigm of science in certain periods of history and parts of the world, in modern times the biological concept of "race" is not useful for classifying human diversity (Montague, 1964). Detailed biochemical analyses of human populations, due to the intermixing

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TABLE 1. Identification and Quantification of Genetic Factors Affecting Susceptibility to Environmental Agents

Genetic Factor	Estimated Occurrence	Environmental Agent(s) to Which the Group is (may be) at Increased Risk
	Red Blood Cell Conditions	
G-6-PD deficience	16% of American black males	Environmental oxidants such as ozone, nitrogen dioxide, and chlorite
Sickle-cell trait	7-13% of American blacks are heterozygotes	Aromatic amino and nitro compounds; carbon monoxide, cyanide
	Liver Metabolism	
Acetylation, slow vs. fast	Slow: 50% Caucasian 50% blacks 10% Japanese	Aromatic amine-induced bladder cancer; numerous drugs, e.g., isoniazid; peripheral neuropathies
	Fast: 50% Caucasian 50% blacks 90% Japanese	Numerous drugs, e.g., isonizaid, hepatitis
Alcohol dehydrogenase variant	70% Japanese	Metabolize alcohol (e.g., ethanol) faster than normal
	Dermatological Conditions	
Albinism Tyrosinase +	1/14,000 blacks 1/16,000 Caucasians; very high frequency in Native Americans	UV radiation

Adapted from Calabrese, 1984: 2-3.

of genetic alleles through marriage, show very complex and varied combinations of genetic traits within and among the physical features that have historically been used to classify race (e.g., skin color, hair characteristics, facial features). For this reason, the United Nations does not recommend using the term "race;" it favors the term "ethnic group," which is a comprehensive descriptor for classifying culturally and socially allied populations (UNESCO, 1975).

Nevertheless, United States health statistics have traditionally included self-identification by race in health surveys (Polednak, 1989). Health data stratified by race establish current status and historical social trends in minority health. They help researchers identify population groups who can benefit from intensive efforts to promote health and to prevent diseases that, when coupled with certain environmental exposures, increase the host's risk of adverse effects.

Ethnic differences in disease rates, however, may be due to numerous factors, including genetic makeup, socioeconomic status, environmental factors associated with that status, as well as

prejudices and social inequalities. Assessing the genetic component of disease susceptibility in ethnic minorities is difficult because of the problems inherent in correlating biological variability with social and cultural classifications.

Basic research into inborn errors of metabolism has increased our understanding of the role of genetics in human disease. Genetic researchers have also devoted considerable effort to understanding why people have very different drug tolerances and responses. Building upon this pharmacogenetic research, ecogenetic researchers seek to uncover the underlying mechanisms in human populations that account for variable susceptibilities to environmental agents. Examples of three genetic traits that increase susceptibility to environmental pollutants are glucose-6-phosphate dehydrogenase (G-6-PD) deficiency, sickle-cell trait, and acetylation phenotype. In addition, chemically induced hypersensitivity is also mediated by genetic makeup. These four conditions are discussed in the following subsections.

Glucose-6-Phosphate Dehydrogenase (G-6-PD) Deficiency

Glucose-6-phosphate dehydrogenase (G-6-PD) is an enzyme that contributes to the integrity of the red blood cell membrane. G-6-PD deficiency has been identified as the most common enzyme abnormality in human beings, and there are more than 120 G-6-PD variants of this gene (Kahn, 1978). The worldwide distribution is not random: G-6-PD deficiency is found almost exclusively in tropical or subtropical countries and in populations from these areas. Persons with this deficiency in the heterozygous form (the allele pair consists of one normal and one abnormal allele) may be less likely to contract malaria than persons with the deficiency in homozygous form (both alleles of the pair are identical and abnormal). The condition is linked to the X-chromosome, and so it is inherited from women but mostly expressed in males, who have no allele for this characteristic to counterbalance the expression of the X gene.

About half of the G-6-PD variants are clinically innocuous because the enzymes of the persons with these deficiencies function almost as well as the normal enzyme. A few kinds of G-6-PD deficiency, however, have great medical significance. Many forms of the G-6-PD gene and its respective product, both abnormal and normal, are known. Two major variants of the normal G-6-PD enzyme are A, which is confined to black Africans and their descendants, and B, which is common throughout the world. The amino acid sequences of these two variants are identical except that A has aspartic acid whereas B has asparagine.

Favism, which is characterized by anemia subsequent to eating fava beans (especially raw fava beans), is associated with a mutation of the B allele. This mutant allele is common in Greece, Sardinia, northwest India, southern Italy, and among the Oriental and Sephardic Jews of Israel. In blacks of African descent, a mutation of the A allele results in acute hemolytic anemia for 1 week after the administration of primaquine, an antimalarial drug. It was the testing of this drug in the 1950s that led researchers to recognize that individuals with G-6-PD deficiency are unable to maintain blood cell membrane integrity during hemolytic stress.

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Subsequent research also showed clinically significant hemolysis with exposure to naphthalene and trinitrotoluene. G-6-PD deficiency has been implicated as a risk factor with exposures to pervasive pollutants such as ozone, nitrogen oxide, copper, and chlorite. However, epidemiological research on the effect of pollutants on individuals with G-6-PD deficiency is lacking. Nor have attempts to develop a predictive animal model for G-6-PD deficiency been successful, despite an extended effort (Calabrese, 1991a).

Sickle-Cell Anemia

Sickle-cell anemia results from a genetic mutation causing the hemoglobin molecule to be distorted. Red blood cells assume a crescent (sickle) shape, thereby impeding blood flow and oxygenation of cells. The disease occurs almost exclusively in African blacks and their descendants (Williams, 1975). The single gene responsible for this disorder has a worldwide distribution corresponding to the distribution in the eastern hemisphere of malaria: equatorial Africa, the Mediterranean, parts of the Middle East, India, and Southeast Asia.

In parts of the world plagued with malaria, heterozygotes (those with one normal gene and one sickle-cell gene) are more likely to reach reproductive age because they are less susceptible to malaria, and when they do contract it, their symptoms are milder. In Africa, the majority of heterozygotes survive to reproductive age. By contrast, only 85% of individuals homozygous for the normal gene survive to reproductive age in Africa. Of individuals homozygous for the sickle-cell trait, only 10% survive into adulthood throughout the world.

Although the red blood cells in heterozygotes do not sickle within their own bodies, sickling can occur under unusual circumstances. When individuals heterozygous for the sickle-cell trait are exposed to lower levels of oxygen in the air or to elevated levels of airborne pollutants, such as carbon monoxide (which displaces oxygen in hemoglobin), a sickle-cell crisis may be induced. Experimental and epidemiological evidence to explore this concern is needed (Calabrese, 1984).

Acetylation Phenotype

The liver metabolizes drugs, proteins, and carbohydrates from food. A primary function of the liver is to remove from the body those chemicals with primary amino groups using an acetylation pathway mediated through n-acetyl transferase enzymes, which transfer acetyl groups from intracellular cofactors onto the amine sites of foreign compounds. Acetylation is an autosomal (nonsex chromosomal) recessive trait. Human beings can be characterized as either slow or fast acetylators. Fast acetylators can remove these toxic substances more rapidly than slow acetylators. In both North American blacks and whites, about half the population are fast acetylators and half are slow acetylators. Fast acetylators can have 9 to 10 times higher amounts of acetylase activity than their slow counterparts. Slow acetylators are at much greater risk of peripheral neuropathies when exposed to drugs such as: isoniazid, used to treat tuberculosis; phenelzine, used to treat depression; and hydralazine, used to treat hypertension (Calabrese, 1991a).

Slow acetylation is also a risk factor for recurrence of bladder cancer related to exposure to aniline (also known as arylamine). (Aniline is an aromatic amine widely used as an intermediate in the synthesis of dyestuffs and in the manufacture of such products as organic chemicals and pharmaceuticals.) Given the greater proportion of the African-American population exposed to aniline (McMicheal et al., 1976; Friedman-Jimenez, 1989), additional epidemiological studies are needed to determine the relationship of aniline exposure, acetylation speed, and bladder cancer. Research is also needed to investigate other health outcomes in slow acetylators, especially among blacks and generally among groups with relatively high exposures to toxicants.

Chemically Induced Hypersensitivities

Hypersensitivity, an exaggerated, harmful response to chemicals, is a disorder that can be mediated by genetics and the immune system. It develops over time in individuals who are repeatedly exposed to toxicants in the environment. Although the role that hypersensitivity plays in determining susceptibility to environmental pollutants is, for the most part, unknown, genetically mediated hypersensitivities may account for as much as 25% of all drug reactions, with severity ranging from mild inconvenience such as a rash to life-threatening conditions such as severe asthma.

Most drugs and environmental chemicals are small molecules that must combine (conjugate) with large molecules within the body to elicit an immune response. Industrial chemicals that form conjugated complexes include diisocyanates, formaldehyde, ethylene oxide, and phthalic anhydrides (Calabrese, 1991a). These chemicals are produced in large volumes in the United States. Diisocynates and formaldehyde are well-known for their ability to cause sensitization reactions in susceptible individuals. This reaction was the reason that use of urea formaldehyde foam insulation in homes was banned in the United States.

The wide spectrum of hypersensitivities mediated by genes and the relatively low frequency of their occurrence in populations have made it difficult to conduct epidemiological studies. Immunochemically reactive metabolites are generally unstable and are minor components of the byproducts of chemical reactions. They are, therefore, difficult to quantify in human beings. Based on proposed animal models, however, a battery of immunotoxicological tests has been developed (Luster et al., 1988). Using these tests, a variety of compounds has been evaluated, and the results for 50 selected chemicals were entered into a database and analyzed. The results have established the capability of each test and combination of tests for detecting immunotoxic compounds (Luster et al., 1992). Minorities are more likely to be repeatedly subjected to occupational pollutants that cause hypersensitive disorders (Friedman-Jimenez, 1989).

The application of genetics to environmental problems is in its infancy. Premature application of genetic research to screening for genetic traits that are related to hypersensitivity would have extensive legal and ethical ramifications (Ashford et al., 1990). Discriminatory practices could

result. The *hypothesis* that the sickle-cell trait (carried by approximately 8% of U.S. blacks) and G-6-PD deficiencies (carried by approximately 10% of U.S. blacks) may lead to health hazards under certain conditions, such as mild hypoxia, precipitated the recommendation that carriers be barred from service in the U.S. armed forces. Since the military has been an important route of training and employment for African Americans, up to 18% of the otherwise eligible population might be refused this opportunity (assuming that there is no overlap in sickle-cell and G-6-PD deficiency traits). In addition, use of such screening by the military could influence pre-employment screening practices by private military contractors and the broader public and private sectors of the economy. Such situations are particularly egregious in light of evidence that every person is likely to carry one or more deleterious genetic traits, yet current detection methods can identify only a few traits (Motulsky, 1978).

Recognizing these dilemmas, participants at a conference held by the World Health Organization recommended expanded epidemiological and biomedical research into ecogenetics. Their recommendations emphasize the following points (World Health Organization, 1991):

1. Primary preventive measures to eliminate hazardous exposures are the preferred means for reducing risks to susceptible individuals.

2. Standards for occupational and environmental exposure should protect the hypersusceptible individual.

3. Screening programs should be set up to be consistent with maintaining and promoting basic human rights such as good health, confidentiality, autonomy, and equity; and they should be conducted only where:
   a. genetic predisposition to susceptibility is significant,
   b. health risks to susceptible individuals are significantly greater than to similarly exposed nonsusceptible people, and
   c. screening techniques are both sensitive and specific.

Future Research

Screening tools for assessing genetic susceptibilities to environmental agents are one component of the larger scientific domain of biomarker research. Biomarker research also includes developing cellular or molecular indicators of exposure (such as concentrations of blood lead or of urinary phenol, which indicates exposure to lead and to phenol, respectively), and developing indicators of adverse health effects (such as liver function assays and determination of sperm concentration and morphology) (National Research Council, 1991).

While ecogenetic research has been limited to date, the development of increasing numbers of DNA probes, the polymerase chain reaction, and other technological innovations will increase the likelihood of discovering additional hypersusceptibility traits relevant to minority populations.

OCCUPATIONAL RISK FACTORS THAT INCREASE SUSCEPTIBILITY TO ENVIRONMENTAL EXPOSURES

Identifying the nature and extent of occupational factors that increase susceptibility of minority workers to environmental exposures is essential to understanding the overall impact of environmental exposures on low-income and minority communities. At the same time, unemployment and underemployment, which occur more frequently among minorities, deny individuals and families access to basic human needs, which can increase their susceptibility to environmental pollutants.

While significant progress in identifying and controlling occupational illness and accidents is evident in some workplaces, many Americans still face serious hazards to their health and safety in their jobs. Minority workers tend to encounter disproportionately greater numbers of these hazards, because they are often employed in especially dirty and dangerous jobs, such as mining (Cherniack, 1986) or harvesting produce (Wright, 1990). Recent trends have indicated a narrowing of the disparity in occupational disability (work-related injuries and/or acute illnesses identified in Bureau of Labor Statistic surveys), although the overall rates of disability have been increasing (Robinson, 1987). However, health problems of minority workers continue to receive little attention despite the magnitude of their occupational morbidity and mortality (Lapin and Hoffman, 1981).

The disproportionately high rates of occupational health and safety problems experienced by minorities are related to: high on-the-job exposure; the illegal use of children as laborers, especially minority children; and secondary exposure of family members to pollutants. These problems are discussed in the following subsections.

Differential Occupational Exposures

Disproportionate numbers of minority workers are engaged in low paying and potentially high-exposure jobs. These exposures may be additive to environmental exposures or may increase susceptibility to some environmental exposures. According to data from the U.S. Department of Labor, Bureau of Labor Statistics data, Latino and African American workers are under-represented in managerial, professional, and technical jobs, which have low exposures to health risks. They are over-represented (relative to their proportion in the population) in "blue-collar" jobs such as those performed by operators, fabricators, laborers, and agricultural workers, which have high exposures to health and safety risks (Friedman-Jimenez, 1989). Collective bargaining units do not exist for the vast majority of these high-risk jobs, so workers have little control over their working conditions, and that in turn reduces incentives for management to improve health and safety in the workplace. Two groups of minority workers experience especially high exposures to toxicants—coke oven workers and farm workers.

Coke Oven Workers. Even within hazardous industries, some jobs are more dangerous than others. In the steel industry, working near coke ovens is associated with excess cancer risk. In operating coke ovens, coal is heated under a reduced concentration of oxygen to produce coke. During this process, many chemicals are released into the air, especially when the oven is opened to remove the coke or to add coal. The highest exposure to coke oven byproducts is experienced by those workers who work "topside," on top of the oven.

In examining excess cancer deaths in coke oven workers at 12 steel mills throughout Canada and the United States, Redmond et al. (1972) found a significant ($p < 0.05$) excess of malignant neoplasms of the lung, trachea, and bronchus in full-time topside workers, with most of this excess occurring among nonwhite workers. When deaths were analyzed by time spent at the coke ovens, a significant ($p < 0.01$) increase in malignant neoplasms of the same three sites (lung, trachea, bronchus) for workers having worked 5 years or more was found, with most of this excess among nonwhite workers.

Farm Workers. Farm work poses severe hazards to laborers. Frequent acute health problems among farmworkers include respiratory infections, allergies, dermatitis, sun- and heat-stroke, urinary tract infections, and musculoskeletal injuries. Chronic health impairments induced by occupational exposures include cancer, reproductive and developmental problems, systemic poisoning, and chronic respiratory disease. EPA has estimated that farmworkers experience as many as 300,000 acute illnesses and injuries each year due to pesticide exposure. Agricultural workers have an annual death rate five times greater than the national rate for all occupations combined (University of California, 1992).

An estimated 1.5 to 2.5 million people are identified as farmworkers. However, the actual number engaged in migrant farm work may be as high as 4 million, including dependents of hired farmworkers and undocumented aliens (U.S. General Accounting Office, 1992). In the West, Midwest, and Southwest, migrant farmworkers are predominantly young, married Hispanic men with families; whereas on the East Coast, inner city poor and families or males from Jamaica, Puerto Rico, and Haiti predominate in agriculture. Regardless of geographic location, minorities are more likely to be subjected to adverse agricultural exposures than nonminorities.

The General Accounting Office concluded that farmworkers are not protected by federal statutes, regulations, and programs (U.S. General Accounting Office, 1992). The magnitude of pesticide exposures and their impact on the health of farmworkers are not known. Protective work practices and field sanitation are inadequate, posing greater risks from pesticide exposures. Minority children, who have greater susceptibility to pesticides than adults (U.S. General Accounting Office, 1992), are more frequently engaged in hazardous farm work (U.S. General Accounting Office, 1992). Access to health care is denied to many farmworkers since they do not receive assistance through Medicaid or the Migrant Health Program (U.S. General Accounting Office, 1992). In addition, because many employers do not report all workers'

earnings to the Social Security Administration, many farmworkers do not receive full benefits when they retire or are disabled. Finally, adequate housing is generally unavailable, with limited government assistance for construction and rehabilitation.

The implications for increased susceptibility to environmental pollutants are obvious. One measure of increased environmental susceptibility is shown by the enhanced rates of tuberculosis among farmworkers in North Carolina (Ciesielski et al., 1991). While the rate of tuberculosis is 3.6% in U.S. blacks and 0.47% in Hispanics, among migrant farmworkers (n = 543) the tuberculin-positive test rate is 33% in Hispanics, 54% in U.S.-born blacks, and 76% in Haitians. Individuals with tuberculosis have a higher susceptibility to respiratory pollutants in particular.

Child Labor

Child labor is defined as employment of children under the age of 18 years. Recent data show that the number of legally and illegally employed children has steadily increased over the past 5 years (U.S. General Accounting Office, 1989b). The increased susceptibility of young children to the adverse effects of environmental exposures has been documented as shown in Table 2. Young children who work in certain occupations are at increased risk to adverse effects due to their increased job-related exposures. A combination of economic and social factors is responsible for the fact that minority children are more likely to enter the work force. These minority children are also more likely to be given jobs that have higher levels of occupational exposures, such as "helping out" in factory sweatshops or picking vegetables in fields still wet with pesticides (Pollack et al., 1992). Data on the incidence or severity of work-related illness among minority children are difficult to collect; however, children are known to be more susceptible than adults to the adverse effects of environmental pollutants and toxins. Further, the earlier these exposures begin, the greater will be their lifetime burden.

Secondary Exposure to Occupational Pollutants

Some occupational exposures associated with poor work conditions and procedures also result in inadvertent transport of toxic chemical hazards to workers' homes, or, in the case of lead paint removal, improper procedures create health hazards for future occupants (Savitz and Chen, 1990; Zirschky, 1991).

The Department of Housing and Urban Development estimates that more than 57 million housing units contain paint having higher percentages of lead than the federal action level of 0.5% lead by weight, or 1 mg/cm^2. Inexperienced lead abatement workers, renovators, and painters, who improperly sand and scrape lead-contaminated surfaces, can increase levels of lead in the home, causing increased blood lead levels among children living there (Moroni, 1992).

TABLE 2. Developmental Processes That Enhance Susceptibility to Environmental Pollutants

High-Risk Groups	Developmental Processes	Pollutant(s) to Which High-Risk Group is (may be) at Increased Risk
Embryos, fetuses, and neonates to the ages of 2-3 months	Immature enzyme detoxification systems	Pesticides, polychlorinated biphenyls (PCBs)
Infants	Infant stomach acidity	Nitrates
Infants and children under 10-12 years old	Immature immune system (lower levels of IgA than in adults)	Respiratory irritants
Infants and young children	Differential absorption of pollutants as a function of age	Barium, lead, radium, strontium
Pregnant women	Physiological modifications due to pregnancy	Anticholinesterase insecticides, carbon monoxide, lead

Adapted from Calabrese, 1984: 3.

An estimated 13 million children are at significant risk of suffering developmental deficits due to ingesting and inhaling lead from all sources. As a result of these exposures, the children are educationally disadvantaged as well, and as adults they will either be unemployed, underemployed, or employed in high-hazard occupations, continuing the cycle of living in poor quality housing with the adverse health effects because of lack of economic opportunity.

Similar scenarios where job tasks increase overall community exposures have been identified for farmworkers who bring pesticide-laden work clothing into their homes, thereby exposing their families; for construction workers who inadvertently carry asbestos home; and for primary and secondary smelter and battery workers who transport toxic metals into their homes.

Recommendations

Despite strong evidence of health problems and of large populations at risk, exposures and diseases are rarely documented. Thus, effective programs for preventing disease have not been developed or implemented. EPA's Pesticide Farm Safety Center Advisory Panel recommended several steps for documenting exposures and diseases (University of California, 1992):

1. Expand medical surveillance, including mandatory reporting of injuries and illnesses, and extend record keeping;
2. Improve pesticide labeling to include all ingredients (both active and inert) and their acute and chronic health effects in language understandable to all workers;

3. Mandate training about pesticide health and safety for farmworkers, farmers, health officials, and regulators;

4. Expand human health research, including epidemiological studies and the development of biomarkers.

These recommendations are applicable to workers in other hazardous occupations as well.

ADDITIONAL RISK FACTORS MORE PREVALENT IN MINORITY POPULATIONS

In addition to genetic susceptibility and occupational exposures, other factors increase the risk of adverse outcomes to environmental exposures and they, too, occur with greater frequency among minority populations. Diseases such as hypertension and diabetes are more prevalent, leaving afflicted individuals less able to cope with adverse environmental exposures. Nutritional deficiencies and use of drugs, tobacco, and alcohol, which reduce resistance to environmental exposures, also occur with higher frequency. Minority populations are more likely to be exposed to multiple environmental hazards, which can act additively or synergistically, increasing the impact of the exposures. In addition, poverty causes minority populations to be more exposed to environmental pollutants. In this section we discuss the role that these factors play in increasing susceptibility to environmental pollutants.

Diseases That Increase Susceptibility

Hypertension, diabetes, chronic liver disease, chronic respiratory diseases, sickle-cell anemia, and AIDS occur more frequently among minority populations (Polednak, 1989). Persons having these diseases are more susceptible to environmental exposures. While some of these diseases may be related to an increased genetic susceptibility, others are probably related to greater environmental exposures (Calabrese, 1984; Sweeny et al., 1988; Warren and Weinstock, 1988). The following subsections present several examples of diseases that may increase susceptibility to certain pollutants, but the examples presented are not exhaustive; many more diseases may also increase susceptibility.

Hypertension. Hypertension occurs much more frequently among blacks and is much less likely to be controlled by medication than in the majority of the population (U.S. Dept. of Health and Human Services, 1986a). Few researchers have studied the relationship between hypertension and environmental exposures, but some studies suggest that cadmium exposures may induce high blood pressure (Schroeder, 1965, 1974). Whether cadmium exposures would exacerbate blood pressure in individuals with pre-existing high blood pressure has not been determined. The major disease outcomes of persistent high blood pressure include kidney and heart disease. Since the kidney is one of the major organs that filters toxic substances from the blood, individuals with hypertension-induced kidney disease may be less able to eliminate toxic substances than healthy individuals.

Diabetes. Diabetes occurs more frequently among some groups of native Americans, African-Americans, and Hispanics (U.S. Dept. of Health and Human Services, 1986b; Indian Health Service, 1991; U.S. Dept. of Health and Human Services, 1991). Diabetes decreases cardiovascular function, resulting in a wide range of physical and metabolic impairments. Some evidence suggests that diabetics are less capable of detoxifying harmful substances, particularly organic solvents, which are common pollutants in indoor air, the environment, and the workplace (Calabrese, 1984).

Chronic Liver Disease. Chronic liver disease occurs with greater frequency among blacks, some Hispanic groups, and some groups of American Indians (U.S. Dept. of Health and Human Services; 1990b; Indian Health Service, 1991; U.S. Dept. of Health and Human Services, 1991). Because the liver is the major organ for metabolizing toxic substances, individuals with impaired liver function are more susceptible to toxic substances, such as pesticides, poly-chlorinated biphenyls, metals, or hydrocarbons (Calabrese, 1984). Minority workers are often exposed to high concentrations of pesticides and polychlorinated biphenyls at work, as well as to persistent chlorinated hydrocarbons occurring in urban air, water, and food (Davies et al., 1972).

Chronic Respiratory Disease. Individuals with chronic respiratory diseases, particularly tuberculosis, which occurs with greater frequency in many Asian groups, blacks, and Hispanics (CDC, 1986b, 1987b,c; Jereb et al., 1991), are more susceptible to a number of environmental exposures. Tuberculosis, for example, impairs the ability of the lung to eliminate particulates and the airborne toxicants on particulate surfaces. Thus, individuals with tuberculosis are probably more susceptible to lower levels of airborne pollutants than individuals with normal lung function.

Hyper-reactive airways disease (e.g., asthma), which is more common and less likely to be treated in blacks (Evans et al., 1987; CDC, 1990; Wiess, 1992a,b), also increases susceptibility to environmental exposures. In studies conducted in Los Angeles and other heavily polluted urban areas, researchers found that individuals with hyper-reactive airways disease suffer asthmatic attacks when the air contains high levels of pollutants (Frezieres et al., 1982; Brain et al., 1988). Thus, pollutants may function as allergens in individuals with hyper-reactive airways disease.

Sickle-Cell Anemia. Since carbon monoxide competitively displaces oxygen in hemoglobin, sickle-cell anemia may make individuals with this genetic trait more susceptible to high levels of carbon monoxide, which frequently pollutes urban air (Williams, 1975).

Human Immunodeficiency Virus Disease (HIV) and AIDS. The immune systems of individuals with HIV gradually lose their ability to function. Thus, these individuals are more susceptible to any exposure against which the body mobilizes the immune response, including exposure to pollutants. Although HIV infection is considered by many in the United States to

be a disease of white male homosexuals, the incidence is actually higher in African-American and Hispanic populations (CDC, 1986a; CDC, 1992).

These are only a few of the diseases that both increase the host's susceptibility to environmental pollutants and occur more frequently among one or more of the minority groups in the United States. Public health professionals need to consider the impact of these diseases on the susceptibility of minorities to environmental and occupational exposures. Although the long-term goal of public health is to reduce the frequency of these diseases in minority populations, an interim goal must be to protect those individuals who have these diseases from environmental exposures to which they are particularly sensitive.

Increased Susceptibility Due to Multiple Pollutant Exposures

Minority populations are subjected to multiple environmental exposures, and each exposure is often to higher levels than those experienced by nonminorities. For example, minorities are more likely to live near toxic dump sites from which toxic substances leach into the water supply; near factories that emit pollutants into air, water, and soil; and in areas of cities with high levels of air pollutants (United Church of Christ, 1987; U.S. Dept. of Health and Human Services/ATSDR, 1990; Bullard, 1992; Mohai and Bryant, 1992; Perfecto and Velásquez, 1992; Wernette and Nieves, 1992). Multiple pollutants having the same site of action in the human body may act additively or synergistically, increasing the impact of a low-level exposure. Most studies of environmental pollutants have examined the impact of individual pollutants on the human body. Pollutants in the real world, however, seldom occur alone.

Consider the hypothetical case of an inner-city minority child as an illustrative scenario. This child is born to a cigarette-smoking father employed in a lead battery plant, and she or he lives with many siblings and other family members in substandard housing adjacent to a freeway and several factories. This child would receive multiple pollutant exposures, including lead from the father's clothes and lead in paint, dust, and drinking water; air pollutants from adjacent freeways and industrial factories; and indoor air pollution from the father's cigarette smoke and from that of other family members who share the same overcrowded residence. This child's diet may also be deficient in vitamins, iron, and protein, which would also increase her/his susceptibility to a number of conditions. These childhood exposures would affect her/his growth, intellectual development, and health status and, thus, her/his later employment potential as an adult. As an adult, this person is likely to be employed in a high-exposure job and live in a neighborhood subject to multiple exposures. The cycle begins again with her/his children. This hypothetical example underscores the need to consider the impact of exposure to multiple pollutants in the environment.

Lifestyle Factors

Various studies indicate that alcohol, tobacco, and drug use are more frequent in minority populations, who are heavily targeted by manufacturers' advertising campaigns (Caetano, 1983; Leland, 1984; U.S. Dept. of Health and Human Services, 1986a,b, 1990a; Davis,

1987). These habits result in impaired respiratory, cardiovascular, and metabolic processes (Calabrese, 1978, 1991b; Collins and Schenker, 1988; Greaves and Schenker, 1988), thereby increasing the susceptibility of minorities to the toxic substances that are normally metabolized or eliminated. Although public health campaigns have successfully reduced the use of these destructive substances in most of the population, additional programs are needed to help minority populations reduce their use of these substances (CDC, 1987a).

Unemployment

Unemployment poses significant health risks. For more than 20 years, researchers have known that elevated blood pressure and mortality from heart disease are associated with job loss and unemployment (Kasl and Cob, 1970; Brenner, 1971). Unemployment rates are significantly higher among minority populations. For example, based upon the 1980 census, 13.3% of Native Americans, Eskimos, and Aleuts were unemployed as compared to the overall unemployment rate in the United States of 6.5% (Indian Health Service, 1991). Similarly, the proportions of the work force unemployed were 15.1% for black, and 10.5% for Hispanic workers (on the U.S. mainland), respectively (U.S. Department of Labor, 1987).

Reasons for unemployment are complex and varied but are directly related to the following: educational levels; language barriers; geographic proximity and access to workplaces; overall strength of the local, national, and international economies; and, in some instances, outright discrimination. Regardless of the reason, higher rates of unemployment in low-income and minority communities add stress that results in increased morbidity and mortality from stress-related diseases. Further, high unemployment compels minorities to accept low paying, high-risk jobs.

Social/Demographic Factors

A higher proportion of minority populations in the United States live below the official poverty level as defined by the Social Security Administration (Rose, 1992). In addition, the birth rate in most minority populations is higher than in the majority of the population (U.S. Dept. of Health and Human Services, 1990a,b, 1991; Indian Health Service, 1991). Thus, compared to the population as a whole, most minority populations have a higher proportion of young persons and women of childbearing age (U.S. Dept. of Health and Human Services, 1990a,b; Indian Health Service, 1991). Fetuses, neonates, infants, children, and pregnant women are all more susceptible to the adverse effects of some pollutant exposures (see Table 2) (Calabrese, 1978, 1986; Beck and Weinstock, 1988b). Pollutant exposures will more severely impact minority populations, because of the higher proportion of these susceptible individuals among them.

Dietary Factors

Minority populations have inadequate diets due to poverty, which is associated with insufficient intake of protein, calories, vitamins, and minerals (Mayer, 1990). In addition, some minority diets are high in fats and salt for cultural or historical reasons (Kerr, 1982; U.S. General

Accounting Office, 1989a). Many of the host defense systems are dependent on vitamins and micronutrients, which are essential for the immune system to function normally. Individuals with deficiencies in these dietary elements may be less capable of defending themselves against pollutants (Beck and Weinstock, 1988a). Minorities are especially vulnerable because they lack essential vitamins, which can potentiate the effects of several pollutants, and because they lack iron, which can, for example, decrease the effects of lead toxicity (Chase, 1980; Looker, 1989; Moss, 1989). Table 3 presents a list of nutritional factors affecting susceptibility to environmental pollutants.

TABLE 3. Partial List of Nutritional Factors Affecting Susceptibility to Environmental Pollutants

Nutritional Deficiency	Estimated Proportion of Individuals in United States Affected	Pollutant(s) to Which High-Risk Group is (may be) at Increased Risk
Vitamin A	25% of children between 7 and 12 have lower than recommended dietary allowance (RDA). Percent is slightly higher among lower income groups.	Aflatoxin, dichlorodiphenyl-trichlorethane (DET), hydrocarbon carcinogens, PCBs
Vitamin C	10-30% of infants, children, and adults of low-income groups receive less than the RDA	Arsenic, cadmium, carbon monoxide, chromium, DDT, dieldrin, lead, mercury, nitrites, ozone
Vitamin E	7% of the general population is "physiologically deficient."	Lead, nitrite, nitrogen dioxide, ozone
Calcium	65% of children between the ages of 2 and 3 receive less than the RDA.	Cadmium, fluoride, lead, strontium
Iron	98% of children between the ages of 2 and 3 receive less than the RDA	Cadmium, hydrocarbon, carcinogens, lead, manganese
Magnesium	Most U.S. males have a partial deficiency.	Fluoride
Phosphorus	Deficiency in people with various kidney diseases.	Lead
Selenium	Unknown: deficience thought to be rare.	Cadmium, mercury, ozone
Zinc	Unknown: deficiency in association with various diseases, but not thought to be widespread.	Cadmium, ethanol, lead, nitrosamines
Riboflavin	30% of women and 10% of men aged 30-60 ingest less than 2/3 of the RDA.	Hydrocarbon, carcinogens, lead, ozone
Dietary protein	10% of women and 5% of men aged 30-60 ingest less than 2/3 of the RDA for protein.	DDT and other insecticides, industrial solvents

Adapted from Calabrese, 1984: 4.

SOCIAL INEQUALITIES

Minority groups are subjected to social inequalities in many aspects of life including education, employment, housing, and health status (Calabrese, 1984; Sweeny et al., 1988; Polednak, 1989). The combined impact of lower job status, unemployment, and poor and overcrowded living conditions is important when assessing ethnic inequalities or "ethnic differences" in health and disease. Studies indicate that stress from direct racist abuse and social structural discrimination adversely impacts health (Warren and Weinstock, 1988). Furthermore, because minorities are more likely to be poor, less fluent in English, and unfamiliar with the political and cultural modes of social function in mainstream America, they are more likely to work in high-exposure occupations without proper safety and health protection, to have jobs not covered by adequate safety regulations, and to live in neighborhoods in which pollutant levels are high (Schroeder, 1965, 1974; U.S. Dept. of Health and Human Services, 1986b). In addition to being subject to multiple pollutant exposures, minorities are also less likely to be able to reduce their exposures.

Poverty and discrimination often result in low self-esteem. When this deficit is added to the above social inequities, minorities are less likely or able to play a major role in the political process (Schroeder, 1965; U.S. Dept. of Health and Human Services, 1986b; Indian Health Service, 1991). Thus, minorities often face many difficulties in attempting to change the social inequities that cause them to be at higher risk for pollutant exposures, and these difficulties reduce their ability to lessen these exposures.

Access to Health Care

Numerous studies have documented the limited access to health care by individuals with low income (Calabrese, 1984; U.S. Dept. of Health and Human Services, 1990b). Access to health care is limited because many have no health insurance, inadequate child care, and/or no transportation to health care facilities (Calabrese, 1984; U.S. Dept. of Health and Human Services, 1991). Because of these difficulties, minority patients are more likely to have advanced disease when they seek health care. As a result, treatment is more difficult and potentially less effective.

Further, some studies suggest that minorities receive poorer quality health care than that received by most of the population (Calabrese, 1984). For example, in a study in New York, physicians with 50% or more minority patients were less likely to follow national guidelines for promoting health and for preventive services than physicians with a small proportion of minority patients (CDC, 1987c).

Studies have found that minorities are less likely to seek preventive health care than whites (Calabrese, 1984). For example, a rising percentage of minority mothers receive little or no prenatal care (CDC, 1986b, 1987b). Inadequate access to high-quality health care permits the impact of exposures to pollutants to be greater.

Educational and Communications Skills

Fewer minority individuals complete high school education, and speak English fluently (Sweeny et al., 1988; Polednak, 1989). They, therefore, have difficulty reading and understanding safety and health instructions at work and are unfamiliar with actions that can be taken to decrease exposures to environmental pollutants. Further, they are less likely to effectively communicate their concerns to employers and government officials (Schroeder, 1965; U.S. Dept. of Health and Human Services, 1986b; Indian Health Service, 1991).

RECOMMENDATIONS

Action to assure protection from harmful levels of pollutants will require the cooperation of both scientists and politicians. The environmental research community must provide the scientific underpinning needed to convince the public and politicians that public health action is necessary to assure that all Americans are equally free from exposure to pollutants and from the effects of those exposures.

The following specific actions can be taken:

- Increase public awareness of inequities in exposures to pollution. Act politically to reduce these inequities.
- Develop improved methods for identifying adverse health effects of pollutants.
- Develop more and better biomarkers of the harmful effects of pollutant exposures.
- Continue to identify pollutants harmful to human beings.
- Document more fully the nature and impact of pollutant exposures.
- Continue to identify biologic and nonbiologic factors that increase the susceptibility of individuals to specific pollutants, especially among minorities.
- Identify commonly occurring mixtures of pollutants.
- Identify interactions among pollutants that enhance their toxicity.
- Identify occupational exposures that enhance the toxic effects of common environmental exposures.
- Implement better occupational health and safety programs to improve protection from hazards, including instructing workers in languages they understand.

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- Stress the
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Books/Reports

Ashford, N.A.
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Academic Pres

Brain, J.D., Be
to Inhaled Poll

Calabrese, E.J.
1986.

Calabrese, E.J
New York: Jo

Calabrese, E.J
Wiley & Sons

Calabrese, E.J
New York: Jo

Calabrese, E.J

Calabrese, E.

Chavkin, W.

Children's De
Book, 1989.

- Provide incentives to industry to reduce exposures to occupational and environmental pollutants.
- Educate the public to motivate them to take action to reduce exposures to harmful pollutants.
- Stress the need for education from within the minority populations so that they can improve their ability to communicate and obtain the necessary political power to induce change.

RECOMMENDED READING

Books/Reports

Ashford, N.A. and Miller, C.S. *Chemical Exposures: Low Levels and High Stakes.* New York: Van Nostrand Reinhold, 1991.

Berg, K. (ed). *Genetic Damage in Man Caused by Environmental Agents.* New York: Academic Press, 1979.

Brain, J.D., Beck, B.D., Warren, A.J., and Shaikh, R.A. (eds.). *Variations in Susceptibility to Inhaled Pollutants.* Baltimore: John Hopkins University Press, 1988.

Calabrese, E.J. *Age and Susceptibility to Toxic Substances.* New York: John Wiley & Sons, 1986.

Calabrese, E.J. *Ecogenetics: Genetic Variation in Susceptibility to Environmental Agents.* New York: John Wiley & Sons, 1984.

Calabrese, E.J. *Nutrition and Environmental Health: Vol 1. The Vitamins.* New York: John Wiley & Sons, 1980.

Calabrese, E.J. *Nutrition and Environmental Health: Vol 2. Minerals and Macronutrients.* New York: John Wiley & Sons, 1981.

Calabrese, E.J. *Multiple Chemical Interactions.* Chelsea: Lewis Publishers, 1991.

Calabrese, E.J. *Pollutants and High-Risk Groups.* New York: John Wiley & Sons, 1978.

Chavkin, W. (ed.). *Double Exposure.* New York: Monthly Review Press, 1984.

Children's Defense Fund. *The Health of America's Children: Maternal and Child Health Data Book,* 1989.

Cohen, B.H., Lilienfeld, A.M., and Huang, P.C. (eds.). *Genetic Issues in Public Health and Medicine.* Springfield: Charles C. Thomas, 1978.

Cruickshank, J.K. and Beevers, D.G. (eds.). *Ethnic Factors in Health and Disease.* London: Wright, 1989.

Grandjean, P. (ed.). *Eco-genetics: Genetic Predisposition to the Toxic Effects of Chemicals.* New York: Chapman & Hall, 1991.

Klein, S.D. "Class, culture, and health." In: Last, J. (ed.). *Maxcy-Rosenau Public Health and Preventive Medicine.* New York: Appleton-Century-Crofts, 1980. pp. 1021-1034.

Polednak, A.P. *Racial and Ethnic Differences in Disease.* New York: Oxford University Press, 1989.

United States Department of Health and Human Services. *Report of the Secretary's Task Force on Black and Minority Health,* vol. I-VIII, 1986.

United States Department of Health and Human Services. *Health Status of the Disadvantaged: Chartbook 1990.* DHHS Pub. No. (HRSA) HRS-P-DV 90-1, 1990.

United States Department of Health and Human Services. *Health Status of Minorities and Low Income Groups,* 3rd ed., 1991.

United States Department of Health and Human Services, Indian Health Service. *Trends in Indian Health - 1991.*

Williams, R.A. (ed.). *Textbook of Black-Related Diseases.* New York: McGraw-Hill, 1975.

Woodhead, A.D., Bender, M.A., and Leonard, R.C. (eds.). *Phenotypic Variation in Populations Relevance to Risk Assessment,* Symposium-Brookhaven National Laboratory. New York: Plicum Press, 1986.

Articles

Blum, K., Noble, E.P., Sheridan, P.J., Finley, O., Montgomery, A., Ritchie, T., Ozkaragoz, T., Fitch, R.J., Sadlack, F., Sheffield, D., Dahlmann, T., Halbardier, S., and Nogami, H. "Association of the A1 allele of the D-sub-2 dopamine receptor gene with severe alcoholism." *Alcohol* 1991; 8(5): 409-416.

Fleming, I., O'Keeffe, M.K., and Baum, A. "Chronic stress and toxic waste: The role of uncertainty and helplessness." *J. Appl. Soc. Psychol.* 1991; 2(23): 1889-1907.

Jacobson, J.L., Jacobson, S.W., Fein, G.G., Schwartz, P.M., and Dowler, J.K. "Prenatal exposure to environmental toxin: A test of the multiple effects model." *Developmental Psychology* 1984; 20(4): 523-532.

Rosenbaum, S. "Child health and poor children." Special Issue: The Impact of Poverty on Children. *American Behavioral Scientist* 1992; 35(3): 275-289.

REFERENCES

ASHFORD, N.A., SPADAFOR, C.J., HATTIS, D.B., and CALDART, C.C. (1990). Monitoring the Worker for Exposure and Disease: Scientific, Legal and Ethical Considerations in the Use of Biomarkers. Johns Hopkins University Press, Baltimore, MD.

BECK, B.D. and WEINSTOCK, S. (1988a). "Age and nutrition." In: Variations in Susceptibility to Inhaled Pollutants (J.D. Brain, B.D. Beck, A.J. Warren, and R.A. Shaikh, eds.). John Hopkins University Press, Baltimore, MD. pp. 104-126.

BECK, B.D. and WEINSTOCK, S. (1988b). "Gender." In: Variations in Susceptibility to Inhaled Pollutants (J.D. Brain, B.D. Beck, A.J. Warren, and R.A. Shaikh, eds.). John Hopkins University Press, Baltimore, MD. pp. 132-141.

BRAIN, J.D., PIKUS, A.A., and GREAVES, I.A. (1988). "Asthma and airway reactivity." In: Variations in Susceptibility to Inhaled Pollutants (J.D. Brain, B.D. Beck, A.J. Warren, and R.A. Shaikh, eds.). John Hopkins University Press, Baltimore, MD. pp. 159-181.

BRENNER, M.H. (1971). "Economic changes and heart disease mortality." Am. J. Pub. Hlth. 61(3): 606-610.

BULLARD, R.D. (1992). "In our backyards." EPA Journal 18(1): 11-12.

CAETANO, R. (1983). "Drinking patterns and alcohol problems among Hispanics in the U.S.: A review." Drug and Alcohol Dependence 12: 37-59.

CALABRESE, E.J. (1978). Pollutants and High-risk Groups. John Wiley & Sons, New York, NY. pp. 4-26; 135-149.

CALABRESE, E.J. (1984). Ecogenetics: Genetic Variation in Susceptibility to Environmental Agents. John Wiley & Sons, New York, NY. pp. 1-12; 39-48; 125-130; 235-255.

CALABRESE, E.J. (1986). Age and Susceptibility to Toxic Substances. John Wiley & Sons, New York, NY.

CALABRESE, E.J. (1991a). "Genetic predisposition to occupationally related diseases: Current status and future directions." In: Ecogenetics: Genetic Predisposition to the Toxic Effects of Chemicals (Proceedings of WHO meeting, Krefeld, Federal Republic of Germany, 17-20 October, 1989) (P. Grandjean, D. Kello, G. Rohrborn, and S. Tarkowski, eds.). Chapman & Hall, New York, NY. pp. 21-55.

CALABRESE, E.J. (1991b). Multiple Chemical Interactions. Lewis Publishers, Chelsea. pp. 467-544.

CENTERS FOR DISEASE CONTROL. (1986a). "Acquired immunodeficiency syndrome (AIDS) among blacks and Hispanics—United States." Morbidity and Mortality Weekly Report 35: 655-666.

CENTERS FOR DISEASE CONTROL. (1986b). "Tuberculosis—United States, 1985." Morbidity and Mortality Weekly Report 35: 699-703.

CENTERS FOR DISEASE CONTROL. (1987a). "Cigarette smoking among blacks and other minority populations." Morbidity and Mortality Weekly Report 36(25): 404-407.

CENTERS FOR DISEASE CONTROL. (1987b). "Tuberculosis in minorities." Morbidity and Mortality Weekly Report 36: 77-80.

CENTERS FOR DISEASE CONTROL. (1987c). "Tuberculosis in blacks." Morbidity and Mortality Weekly Report 36: 212-220.

CENTERS FOR DISEASE CONTROL. (1990). "Asthma—United States, 1980-1987." Morbidity and Mortality Weekly Report 39: 493-497.

CENTERS FOR DISEASE CONTROL. (1992). "The second 100,000 cases of Acquired Immunodeficiency Syndrome—United States." Morbidity and Mortality Weekly Report 41(2): 28-29.

CHASE, H.P., HAMBIDGE, K.M., BARNETT, S.E., HOUTS-JACOB, M.S., LENZ, K., and GILLESPIE, J. (1980). "Low vitamin A and zinc concentrations in Mexican-American children with growth retardation." Am. J. Clin. Nutr. 33(11): 2346-2349.

CHERNIACK, M. (1986). The Hawk's Nest Incident. Yale University Press, New Haven, CT.

CIESIELSKI, S.D., SEED, J.R., ESPOSITO, D.H., and HUNTER, N. (1991). "The epidemiology of tuberculosis among North Carolina migrant farmworkers." J. Am. Med. Assoc. 235(13): 1715-1719.

COLDITZ, G.A., STAMPFER, M.J., and GREEN, L.C. (1988). "Diet." In: Variations in Susceptibility to Inhaled Pollutants (J.D. Brain, B.D. Beck, A.J. Warren, and R.A. Shaikh, eds.). John Hopkins University Press, Baltimore, MD. pp. 314-331.

COLLINS, M. and SCHENKER, M. (1988). "Susceptibility to neoplasia altered by tobacco smoke." In: Variations in Susceptibility to Inhaled Pollutants (J.D. Brain, B.D. Beck, A.J. Warren, and R.A. Shaikh, eds.). John Hopkins University Press, Baltimore, MD. pp. 269-294.

DAVIES, J.E., EDMUNDSON, W.F., RAFFONELLI, A., CASSADY, J.C., and MORGADE, C. (1972). "The role of social class in human pesticide pollution." Am. J. Epidemiol. 96: 334-341.

DAVIS, R.M. (1987). "Current trends in advertising and marketing methods." N. Engl. J. Med. 316(12): 725-732.

EVANS, R., MULLALLY, D.I., WILSON, R.W., GERGEN, P.J., ROSENBERG, H.M., GRAUMAN, J.S., CHEVARLEY, F.M., and FEINLEIB, M. (1987). "National trends in the morbidity and mortality of asthma in the U.S.: Prevalence, hospitalization and death from asthma over two decades: 1965-1984." Chest 91: 65S-74S.

FREZIERES, R.G., COULSON, A.H., KATZ, R.M., DETELS, R., SIEGEL, S.C., and RACHELEFSKY, G.S. (1982). "Response of individuals with reactive airway disease to sulfates and other atmospheric pollutants." Ann. Allergy 48(3): 156-165.

FRIEDMAN-JIMENEZ, G. (1989). "Occupational disease among minority workers: A common and preventable public health problem." AAOHN Journal 37(2): 64-70.

GREAVES, I.A. and SCHENKER, M. (1988). "Tobacco smoking." In: Variations in Susceptibility to Inhaled Pollutants (J.D. Brain, B.D. Beck, A.J. Warren, and R.A. Shaikh, eds.). John Hopkins University Press, Baltimore, MD. pp. 182-203.

INDIAN HEALTH SERVICVE. (1991). Trends in Indian Health—1991. U.S. Department of Health and Human Services, Public Health Service.

JEREB, J.A., KELLY, G.D., DOOLEY, S.W., CAUTHEN, G.M., and SNIDER, D.E. (1991). "Tuberculosis morbidity in the United States: Final data, 1990." Morbidity and Mortality Weekly Report 40(SS-3): 23-27.

KAHN, A. (1978). "G6PD variants." In: Human Genetic Variation in Response to Medical and Environmental Agents: Pharmacogenetics and Ecogenetics, Human Genetics, Suppl. 1 (Proceedings of the International Titisee Conference, Titisee, Federal Republic of Germany, 13-15 October, 1977) (F. Vogel, W. Buselmaier, W. Reichert, G. Kellermann, and P. Berg, eds.). Springer-Verlag, New York, NY. pp. 37-44.

KASL, S.V. and COBB, S. (1970). "Blood pressure changes in men undergoing job loss: A preliminary report." Psychosom. Med. 32(1): 19-38.

KERR, G.R., AMANTE, P., DECKER, M., and CALLEN, P.W. (1982). "Ethnic patterns of salt purchase in Houston, Texas." Am. J. Epidemiol. 115: 906-916.

LAPIN, N. and HOFFMAN, K. (1981). "Occupational disease among minority workers: An annotated bibliography." U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, Cincinnati, OH.

LELAND, J. (1984). "Alcohol use and abuse in ethnic minority women." In: Alcohol Problems in Women (S.C. Wilsnack and L.J. Beckman, eds.). Gilford Press, New York, NY. pp. 69-96.

LOOKER, A.C., JOHNSON, C.L., McDOWELL, M.A., and YETLEY, E.A. (1989). "Iron status: Prevalence of impairment in three Hispanic groups." Am. J. Clin. Nutr. 49(3): 553-558.

LUSTER, M.I., MUNSON, A.E., THOMAS, P.T., HOLSAPPLE, M.P., FENTERS, J.D., WHITE, K.L., Jr., LAUER, L.D., GERMOLEC, D.R., ROSENTHAL, G.J., and DEAN, J.H. (1988). "Development of a testing battery to assess chemical-induced immunotoxicity." Fundam. Appl. Toxicol. 10: 2-19.

LUSTER, M.I., PORTIER, C., GAYLA, P.D., WHITE, K.L., Jr., GENNINGS, C., MUNSON, A.E., and ROSENTHAL, G.J. (1992). "Risk assessment in immunotoxicology: I. Sensitivity and predictability of immune tests." Fundam. Appl. Toxicol. 18: 200-210.

MAYER, J. (1990). "Hunger and undernutrition in the United States." J. Nutr. 120: 919-923.

McMICHEAL, A.J., SPIRTAS, R., GAMBLE, J.F., and TOUSEY, P.M. (1976). "Mortality among rubber workers: Relation to specific jobs." J. Occup. Med. 18(3): 178-185.

MOHAI, P. and BRYANT, B. (1992). "Race, poverty, and the environment." EPA Journal 18(1): 6-8.

MONTAGU, A. (ed.). (1964). The Concept of Race. Collier Books, Collier-Macmillian Limited, London, England.

MORONI, R. (1992). "The Department of Housing and Urban Development perspective." (Presentation at Worker Protection Practices for Lead Abatement). In: Workshop Proceedings. Society for Occupational and Environmental Health, McLean, VA. July 22-24.

MOSS, A.J., LEVY, A.S., KIM, I., and PARK, Y.K. (1989). "Use of vitamin and mineral supplements in the United States: Current uses, types of products and nutrients." Advance Data from Vital and Health Statistics, No. 174. DHHS Publication No. (PHS) 89-1250. pp. 1-19.

MOTULSKY, A.G. (1978). "Bioethical problems in pharmacogenetics and ecogenetics." In: Human Genetic Variation in Response to Medical and Environmental Agents: Pharmacogenetics and Ecogenetics, Human Genetics, Suppl. 1 (Proceedings of the International Titisee Conference, Titisee, Federal Republic of Germany, 13-15 October, 1977) (F. Vogel, W. Buselmaier, W. Reichert, G. Kellermann, and P. Berg, eds.). Springer-Verlag, New York, NY. pp. 185-192.

NATIONAL RESEARCH COUNCIL. (1991). Environmental Epidemiology: Public Health and Hazardous Wastes, Vol 1. National Academy Press, Washington, DC.

PERFECTO, I. and VELÁSQUEZ, B. (1992). "Farm workers: Among the least protected." EPA Journal 18(1): 13-14.

POLEDNAK, A.P. (1989). Racial and Ethnic Differences in Disease. Oxford University Press, New York, NY.

POLLACK, S.H., RUBENSTEIN, H., and LANDRIGAN, P.J. (1992). "Child labor." In: Maxcy-Rosenau-Last: Public Health and Preventative Medicine, 13th ed. (J.M. Last and R.B. Wallace, eds.). Appleton and Lange, Norwalk, CT. pp. 576-581.

REDMOND, C.K., CIOCCO, A., LLOYD, J.W., and RUSH, H.W. (1972). "Long-term mortality study of steelworkers. VI: Mortality from malignant neoplasms among coke oven workers." J. Occup. Med. 14(8): 621-629.

ROBINSON, J.C. (1987). "Trends in racial inequality and exposure to work-related hazards, 1968-1986." Milbank Q. 65 (Suppl 2): 404-419.

ROSE, S.J. (1992). Social Stratification in the United States. The New Press, New York, NY.

SAVITZ, D.A. and CHEN, J. (1990). "Parental occupation and childhood cancer: Review of epidemiologic studies." Environ. Health Perspect. 88: 325-337.

SCHROEDER, H.A. (1965). "Cadmium as a risk factor in hypertension." J. Chron. Dis. 18: 647.

SCHROEDER, H.A. (1974). "The role of trace metals in cardiovascular diseases." Med. Clin. N. Amer. 58(2): 381-396.

SWEENY, T.D., BRAIN, J.D., and GODLESKI, J.J. (1988). "Preexisting diseases." In: Variations in Susceptibility to Inhaled Pollutants (J.D. Brain, B.D. Beck, A.J. Warren, and R.A. Shaikh, eds.). John Hopkins University Press, Baltimore, MD. pp. 142-158.

UNITED NATIONS EDUCATIONAL, SCIENTIFIC, AND CULTURAL ORGANIZATION (UNESCO). (1975). Race, Science, and Society. (L. Kuper, ed.). UNESCO Press, Paris, France.

UNITED STATES DEPARTMENT OF HEALTH AND HUMAN SERVICES. (1986a). Report of the Secretary's Task Force on Black and Minority Health, Vol. I, IV. O. Carter-Pokras and L. Montgomery, personal communication, 1992.

UNITED STATES DEPARTMENT OF HEALTH AND HUMAN SERVICES. (1986b). Report of the Secretary's Task Force on Black and Minority Health, Vol. I, VII. O. Carter-Pokras, and L. Montgomery, personal communication, 1992.

UNITED STATES DEPARTMENT OF HEALTH AND HUMAN SERVICES. (1990a). Health Status of the Disadvantaged: Chartbook 1990. DHHS Pub. No. (HRSA) HRS-P-DV 90-1.

UNITED STATES DEPARTMENT OF HEALTH AND HUMAN SERVICES. (1990b). Deaths of Hispanic Origin, 15 Reporting States, 1978-8. National Center for Health Statistics, Vital Statistics Series 20, No. 18, DHHS Pub. No. PHS 91-1855, December. pp. 1-30.
UNITED STATES DEPARTMENT OF HEALTH AND HUMAN SERVICES. (1991). Health Status of Minorities and Low Income Groups, 3rd ed.
UNITED STATES DEPARTMENT OF HEALTH AND HUMAN SERVICES, AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY. (1990). Proceedings from the National Minority Health Conference: Focus on Environmental Contamination. Atlanta, GA. December.
UNITED STATES DEPARTMENT OF LABOR, BUREAU OF LABOR STATISTICS. (1987). Statistical Abstracts.
UNITED STATES GENERAL ACCOUNTING OFFICE. (1989a). Food Assistance Programs: Recipient and Expert Views on Food Assistance at Four Indian Reservations. Report No. GAO/RCED-89-177: 4-6.
UNITED STATES GENERAL ACCOUNTING OFFICE. (1989b). "Sweatshops and child labor violations: A growing problem in the United States." Testimony of W. Gainer before the Capital Hill Forum on the Exploitation of Children in the Workplace. November.
UNITED STATES GENERAL ACCOUNTING OFFICE. (1992). Report to Congressional Requesters: Hired Farmworkers' Health and Well-Being at Risk. Pub. No. GAO/HRD-92-46. February.
UNITED CHURCH OF CHRIST, COMMISSION FOR RACIAL JUSTICE. (1987). "Toxic wastes and race in the United States." Public Data Access.
UNIVERSITY OF CALIFORNIA AGRICULTURAL HEALTH AND SAFETY CENTER AT DAVIS AND THE WESTERN CONSORTIUM FOR PUBLIC HEALTH. (1992). Final Report to the U.S. Environmental Protection Agency from the Pesticide Farm Safety Center Advisory Panel. Western Consortium for Public Health, Berkeley, CA.
WARREN, A.J. and WEINSTOCK, S. (1988). "Age and preexisting disease." In: Variations in Susceptibility to Inhaled Pollutants (J.D. Brain, B.D. Beck, A.J. Warren, and R.A. Shaikh, eds.). John Hopkins University Press, Baltimore, MD. pp. 253-268.
WERNETTE, D.R. and NIEVES, L.A. (1992). "Breathing polluted air." EPA Journal 18(1): 16-17.
WIESS, K.B. (1992a). "Asthma among minority children," Chest. June supplement. 101(6): 368S-371S.
WIESS, K.B. (1992b). "Inner-city asthma." Chest. June supplement. 101(6): 362S-367S.
WILLIAMS, R.A. (ed.). (1975). Textbook of Black-Related Diseases. McGraw Hill, New York, NY. pp. 234-236.
WORLD HEALTH ORGANIZATION. (1991). "Report on a WHO Consultation." In: Ecogenetics: Genetic Predisposition to the Toxic Effects of Chemicals (Proceedings of WHO meeting, Krefeld, Federal Republic of Germany, 17-20 October, 1989) (P. Grandjean, D. Kello, G. Rohrborn, and S. Tarkowski, eds.). Chapman & Hall, New York, NY. pp. 3-18.
WRIGHT, A. (1990). The Death of Ramon Gonzales: A Modern Agricultural Dilemma. University of Texas Press, Austin, TX.
ZIRSCHKY, J. (1991). "Employee transported contaminant releases." Haz. Waste & Haz. Mat. 7(2): 201-209.