"ENVIRONMENTAL JUSTICE": THE CENTRAL ROLE OF RESEARCH IN ESTABLISHING A CREDIBLE SCIENTIFIC FOUNDATION FOR INFORMED DECISION MAKING

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Although much of the evidence is anecdotal and circumstantial, there are mounting concerns that environmental health risks are borne disproportionately by members of the population who are poor and nonwhite. We examine the central role of environmental health research in defining the dimensions of the problem, understanding its causes, and identifying solutions. Environmental health sciences, including epidemiology, exposure analysis, pharmacokinetics, toxicology, and surveillance monitoring, must be employed to determine the extent to which society has achieved "equity" and "justice" in safeguarding the health and safety of its citizens. By improving our ability to identify, evaluate, prevent, and/or reduce risks for all members of society, environmental health research can contribute directly to fair and equitable protection for everyone, regardless of age, ethnicity, gender, race, or socioeconomic status.

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4. Abbreviations: ATSDR, Agency for Toxic Substances and Disease Registry; CEQ, Council on Environmental Quality; EPA, Environmental Protection Agency; GAO, General Accounting Office; NIEHS, National Institute of Environmental Health Sciences; OEE, Office of Environmental Equity; PCB, polychlorinated biphenyls; UCC, United Church of Christ.

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INTRODUCTION

The premise that environmental health risks are distributed unequally across different segments of society has recently gained national prominence (Gore, 1992; Satchell, 1992; Weisskopf, 1992a; Baucus, 1993; BNA, 1993; Clinton, 1993; Lewis, 1993). There is mounting evidence that a disproportionate share of environmental hazards is borne by disadvantaged communities, including those who are poor, have limited formal education, and are either unemployed or work under hazardous conditions (UCC, 1987; EPA, 1992a; Johnson et al., 1992). Concerns that possible disparities in environmentally induced illness are related to socioeconomic class and ethnicity/race have made this issue a top priority on the environmental health agenda of the United States (ATSDR, 1988; EPA, 1992a; Baucus, 1993; BNA, 1993; Clinton, 1993; Lewis, 1993).

Most of the studies cited as evidence of environmental inequities are observational. That is to say, these studies document disparities by relying on statistical associations between demographic characteristics of populations, primarily race and income, and indirect surrogates for exposure, such as residential proximity to pollution sources. These investigations have been consistent in finding that members of disadvantaged groups, including many African Americans and Hispanics, are more likely than affluent whites to: (1) live near sources of environmental pollution, such as waste sites (Gould, 1986; UCC, 1987; Bullard, 1990; Goldman, 1991); (2) reside in urban areas where ambient levels of certain pollutants, such as lead and carbon monoxide, are elevated (Gelobter, 1986; Wernette and Nieves, 1991; Bryant and Mohai, 1992a; Sexton et al., 1993); (3) eat significantly greater amounts of contaminated fish (EPA, 1992a; Calderon et al., 1993); and (4) be employed in potentially dangerous occupations, such as migrant farm work (EPA, 1992a; Moses et al., 1993).

Because many racial minorities are more likely than whites to be disadvantaged in terms of education, income, and occupation, some researchers have pointed to "environmental racism" as the cause of disparities in environmental risks (Bullard and Wright, 1986, 1987; UCC, 1987; Russell, 1989; Bullard, 1991; Satchell, 1992). Typically, however, those concerned about the issue refer to it more positively as the need for all citizens to attain "environmental equity" (EPA, 1992a) or "environmental justice" (Ferris, 1992; Gore, 1992; Lewis, 1993; Taylor, 1992). The terms "equity" and "justice" focus attention on the underlying principle that fairness and equality are inherent in society's efforts to protect the health of all citizens from the adverse effects of environmental agents.

Although a substantial amount of anecdotal and circumstantial evidence suggests that environmental health risks vary by class and race, there is little scientific information available to help risk assessors determine the magnitude, extent, and causes of risk differentials. Characterization of exposures, doses, and effects in both the general population and in groups potentially at greater risk is an important step toward better estimation and

comparison of risks for all citizens. By collecting relevant, scientifically sound data to fill important gaps in knowledge, as well as by improving understanding of fundamental mechanisms of environmentally related disease, research provides a solid scientific foundation on which to make more informed choices about appropriate strategies to prevent or reduce unacceptable risks.

The need for additional research does not imply that efforts to redress environmental disparities should be held in abeyance. Research must not be used as an excuse to delay remedial actions when problems and solutions are apparent, as for example when low-income communities need improved sanitation and safe drinking water. If a more equitable outcome is to be achieved, existing environmental laws and regulations must be actively and equally enforced in all communities.

Shared concerns about the environmental health risks experienced by disadvantaged populations have mobilized civil rights activists, public interest groups, Congress, state and local governments, federal agencies, and the general scientific community. Appropriately, these groups focus on effective actions that protect at-risk groups from environmental hazards. Research is a necessary adjunct to these activities because it allows us to identify at-risk populations, to recognize strategies that effectively and efficiently mitigate risks, and to measure the success of efforts to prevent or reduce unacceptable risks.

This paper examines how science can offer a credible basis from which to make informed and equitable decisions about environmental health risks. The discussion is divided into four sections. First, we present a brief survey of environmental justice from a historical perspective, tracing the evolution of this issue in the United States and highlighting its emergence as a national priority.

Second, we examine some of the important questions regarding the roles of class and race in determining health status, and present three conceptual models that attempt to explain qualitatively how class, race, and environmental factors might cause health disparities.

Third, we outline a risk-based framework for analyzing issues of environmental justice. We review some of the literature calling for a "risk-based" approach to priority setting, briefly describe the interrelationships among risk assessment, risk management, and environmental health research, underscore the critical importance of identifying and evaluating groups potentially at greater risk, and propose a conceptual model to help generate testable hypotheses about environmental justice.

Finally, we provide a short description of the ways research can be effective in strengthening the scientific credibility of decisions about environmental equity and justice. We compare and contrast the "community perspective" and the "risk-based perspective" on research goals,

describe the two generic causes of uncertainties in health risk assessment (lack of data and lack of scientific understanding), and suggest general research approaches for reducing these scientific uncertainties.

One major theme runs throughout the paper. Scientific research is an essential component of 1) efforts to better understand the extent to which we have attained environmental equity and justice, and 2) informed decisions about how best to address documented instances of environmental inequity and injustice.

"ENVIRONMENTAL JUSTICE" FROM A HISTORICAL PERSPECTIVE

Evolution of Environmental Justice as a National Issue (1971-1991)

Although the terms "environmental equity" and "environmental justice" are relatively new, the underlying issues are not. Inequitable distribution of the costs and benefits associated with environmental regulations has been the topic of discussion and study for more than 20 years (Gelobter, 1986; Mohai and Bryant, 1992). The 1971 annual report of the President's Council on Environmental Quality (CEQ) presented information on disparities in environmental factors by demographic groups (Mohai and Bryant, 1992). Several studies published during the 1970s showed that disadvantaged groups are more likely to encounter above average levels of air pollution (Gelobter, 1986; Mohai and Bryant, 1992).

During the 1980s, hundreds of grass roots, community action groups brought attention to the environmental problems facing disadvantaged communities (EPA, 1992a). In 1982, demonstrations by members of a low-income, predominately black community against the proposed site for a polychlorinated biphenyl (PCB) landfill in Warren County, North Carolina garnered national media coverage (Lee, 1990). The following year, a General Accounting Office (GAO) study found that three of four hazardous waste sites investigated in the southern United States were located in primarily African American communities (GAO, 1983). In 1985, the first national African American environmental organization, the Center for Environment, Commerce, and Energy, was established (EPA, 1992a). That same year, the National Council of Churches' Eco-Justice Working Group began to address environmental issues (EPA, 1992a).

The United Church of Christ's (UCC's) Commission for Racial Justice released a nationwide study in 1987 on the demographics of populations living near waste sites (UCC, 1987). The report found that in communities with one or more commercial hazardous waste facilities, the proportion of racial minorities was significantly greater than in communities without such facilities. The UCC report heightened concerns about inequitable environmental risks and eventually spawned the Conference on Race and the Incidence of Environmental Hazards, held in Ann Arbor, Michigan in January 1990. At the conference, a group of social scientists, community activists, and civil rights leaders formed the Michigan Coalition, a group devoted to increasing the visibility of environmental justice issues (Bryant and Mohai, 1992b).

In the early 1990s, mostly as a result of the Coalition's successful efforts, the issue of environmental justice gained national prominence (Gladwell, 1990; Lancaster, 1990; Milloy, 1990; Winslow, 1990; Bryant and Mohai, 1992b; EPA, 1992a). In 1990, *Race, Poverty and the Environment*, "a newsletter for social and environmental justice," was launched in San Francisco, and Robert Bullard's book, *Dumping in Dixie: Race, Class and Environmental Quality* was published (Bullard, 1990). In December 1990, the Agency for Toxic Substances and Disease Registry (ATSDR), along with other federal agencies and several national organizations, sponsored the National Minority Health Conference: Focus on Environmental Contamination (Johnson et al., 1992). The conference was the first attempt to take a comprehensive look at issues of environmental justice from a scientific perspective.

Media coverage accelerated in 1991 (Gibbons, 1991; Hager et al., 1991; Hunt, 1991; Ina, 1991; Okie, 1991; Stout, 1991) as equity and justice became mainstream environmental issues. A symposium on Environmental Issues in Ethnic Communities, sponsored by the Environmental Protection Agency (EPA) and other public and private groups, was held in July 1991. It examined the environmental problems facing Colorado's ethnic communities. In October, the First National People of Color Environmental Leadership Summit was held in Washington, D.C. and received widespread media attention (Summit, 1991).

Emergence of Environmental Justice as a National Priority (1992-1993)

The pace of activities related to environmental justice picked up considerably in 1992. The first "Directory for People of Color Environmental Groups" was published; it identified 205 organizations in 35 states, the District of Columbia, and Puerto Rico (Bullard, 1992). The *EPA Journal* devoted the March/April issue to the subject of "Environmental Protection—Has It Been Fair?" (Heritage, 1992). A workshop on "Equity in Environmental Health: Research Issues and Needs," sponsored jointly by the EPA, ATSDR, and the National Institute of Environmental Health Sciences (NIEHS), was held in August in North Carolina. The purpose was to discuss initial drafts of the manuscripts that comprise this issue of *Toxicology and Industrial Health.*

In July, then Senator Albert Gore introduced in the United States Senate the "Environmental Justice Act of 1992" (Gore, 1992). Because the 102d Congress never acted on the bill, it was reintroduced in the 103d Congress in the House of Representatives by Representative John Lewis (D-Georgia) (Lewis, 1993) and in the Senate by Senator Max Baucus (D-Montana) (Baucus, 1993). The Act calls for a "program to ensure nondiscriminatory compliance with environmental, health, and safety laws and to ensure equal protection of the public health." Among its provisions are requirements to collect environmental health data so that geographical areas subject to the "highest loadings of toxic chemicals, through all media," can be identified, and to "assess the health effects that may be caused by emissions in those areas of highest impact."

The EPA's 1992 report, "Environmental Equity: Reducing Risks For All Communities" (EPA, 1992a), sparked a flurry of press articles (Satchell, 1992; Weisskopf, 1992a) and prompted Representative Henry Waxman (D-California) to hold public hearings (Weisskopf, 1992b). The EPA report found a paucity of data that related environmental risk to race and class. However, the report stated that the available information was "highly suggestive" of disparities: "The evidence indicates that racial minority and low-income populations are disproportionately exposed to lead, selected air pollutants, hazardous waste facilities, contaminated fish tissue, and agricultural pesticides in the workplace" (EPA, 1992a). Among other things, the report recommended that EPA raise the priority given to environmental equity, identify and target opportunities to reduce high concentrations of risk to specific population groups, and increase efforts to involve racial minorities and low-income communities in environmental policy making (EPA, 1992a).

In September 1992, a special report by the *National Law Journal*, "Unequal Protection: The Racial Divide in Environmental Law" (Nat. Law J., 1992), argued that environmental laws, including statutes that set standards for air, water, and waste disposal, were not enforced equally. The authors' analysis of census data, EPA's civil court case docket, and the record of EPA's performance at Superfund sites showed that federal compliance and enforcement actions were taken less often and with less force in communities inhabited by "people of color." Based on this report, Representative John Conyers (D-Michigan) held public hearings in March 1993 to investigate allegations of environmental discrimination.

The EPA formally established the Office of Environmental Equity (OEE) in November 1992 to deal with issues of environmental equity and justice. The OEE has responsibility for coordinating communication, outreach, education, and training of the public on equity issues; for providing technical and financial assistance to outside groups on equity concerns; and for serving as a central repository of information on environmental equity.

At the request of Senator John Glenn (D-Ohio), the GAO began a study in December 1992 to examine EPA's activities relating to environmental equity, emphasizing its methods for characterizing demographics around polluting sites and its techniques for data collection and application. The GAO study is ongoing (Gaylord, 1993).

In 1992, results from the 1990 Census showed that the U.S. poverty rate (14.2%) was the highest in a decade. Poverty rates were found to vary substantially across ethnic and racial groups: 11.3% for whites, 13.8% for Asian Americans, 28.7% for Hispanics, and 32.7% for blacks (Census, 1990; Pear, 1992). Population projections indicated that non-Hispanic whites, currently 75% of the U.S. population, will make up only about 53% of the total by the year 2050. Within this time-frame, the Hispanic population was projected to increase from 9.0% of the total population to 21.1%, blacks from 11.8% to 15.0%, Asians from 3.0% to 10.7%, and Native Americans from 0.7% to 1.2% (Census, 1990; Price, 1992; Vobejda, 1992). The

evidence indicates that the United States is rapidly becoming more diverse ethnically and racially. At the same time, the percentage of U.S. residents who live in poverty is increasing, a disproportionate share of whom are minorities.

In 1993, environmental justice is at the forefront of the nation's environmental agenda. On Earth Day, President Clinton issued a statement on environmental justice:

> I have asked the Environmental Protection Agency and the Department of Justice to begin an inter-agency review of federal, state and local regulations and enforcement that affect communities of color and low income communities with the goal of formulating an aggressive investigation of the inequalities in exposure to environmental hazards. As part of this evaluation, the Department of Justice and the Environmental Protection Agency—in coordination with the Department of Housing and Urban Development and the Department of Labor—will identify examples of communities in which the distributional inequalities of environmental decision making have adversely affected minority and low income populations. This process will be the basis for legislative and enforcement reforms if necessary (Clinton, 1993).

The White House is currently drafting an Executive Order on Environmental Justice to ensure "that management of federal facilities, establishment of federal policies, and the implementation of federal actions promote fair and proportionate environmental protection for all."

> Under the order, all federal agencies would be required to adopt a regulation stating that they intend to 'administer, interpret, and enforce all regulations and conduct all programs affecting health or the environment, including facility siting or permitting, in a manner that addresses the exposure of minority and low-income populations to environmental hazards.' The executive order also would direct federal agencies—as required by NEPA—to include analyses of social and economic impacts when conducting major federal actions that affect the human environment. An interagency task force on environmental justice, chaired by the White House, also would be set up to receive reports from agencies on their strategy for implementing the order and revising procedures to reflect environmental justice priorities. The task force would report to the president on federal environmental justice activities that are underway (BNA, 1993).

The recently released *Report of the National Performance Review*, under the direction of Vice President Gore, recommended that "EPA should develop a blueprint of actions that will

incorporate environmental justice consideration into all aspects of EPA operations" (Gore, 1993). In line with this recommendation, Carol Browner, EPA Administrator, has made environmental equity one of her top strategic priorities, emphasizing the need to incorporate equity into EPA's mission and programs (Browner, 1993; Inside EPA, 1993a).

As of 1993, six EPA Regions have issued policy statements on environmental equity, created equity offices and work groups, or announced equity strategies. At the state level, New York, California, and South Carolina have introduced environmental justice bills, while Texas, Louisiana, and Kentucky announced environmental justice task forces, programs, or work groups (Gaylord, 1993).

Representative Cardiss Collins (D - Illinois) introduced in the U.S. House of Representatives the "Environmental Equal Rights Act of 1993." The Act seeks "to amend the Solid Waste Disposal Act to allow petitions to be submitted to prevent certain waste facilities from being constructed in environmentally disadvantaged communities" (Collins, 1993). The Act has been referred to the House Committee on Energy and Commerce.

The U.S. Senate and the House of Representatives have explicitly called for increased emphasis on environmental justice in the pending bills that would elevate EPA to cabinet-level status. Although the House and Senate versions differ, both propose creating an Office of Environmental Justice in the new Department of the Environment (Gaylord, 1993; Inside EPA, 1993b; Wellstone, 1993).

The bottom line message is loud and unequivocal. Environmental justice—adequate protection for all people, regardless of age, ethnicity, gender, socioeconomic class, or race—is an important and explicit goal of environmental health programs in the United States, and will be for the foreseeable future.

CLASS, RACE, AND ENVIRONMENT AS DETERMINANTS OF HEALTH STATUS

Class and Race as Causes of Differences in Health Status

It is well established that rates of disease and death in the United States vary significantly by class and race (DHHS, 1985, 1991a,b; Montgomery and Carter-Pokras, 1993). However, the exact nature of the interactions between socioeconomic status and ethnicity/race, and their relative roles in affecting health status are a matter of debate (Freeman, 1989, 1991; Gladwell, 1990; Navarro, 1990; Baquet et al., 1991; Gibbons, 1991).

Regardless of its relationship to ethnicity/race, there is general consensus that socioeconomic status (i.e., class as determined by education, income, and/or occupation) is an important factor in morbidity and mortality (Graham et al., 1992). Recently, Pappas et al. (1993)

examined changes in mortality rates from 1960 through 1986 according to income and education among persons 25 to 64 years of age in the United States. They found that, despite important declines in death rates, there were greater disparities between people of different incomes and educational levels in 1986 than in 1960. As death rates declined, poor and less educated members of society did not benefit equally in comparison with those who were wealthier and better educated.

> The inverse relation between mortality and socioeconomic status persisted in 1986 and was stronger than in 1960. The disparity in mortality rates according to income and education increased for men and women, whites and blacks, and family members and unrelated persons. Over the 26-year period, the inequalities according to educational levels increased for whites and blacks by over 20 percent in women and over 100 percent in men. In whites, absolute death rates declined in persons of all educational levels, but the reduction was greater for men and women with more education than for those with less (Pappas et al., 1993).

Guralnik et al. (1993) studied a representative sample of elderly people in the Piedmont region of North Carolina. They found that 65-year-old men and women with 12 or more years of education can expect 2.4 to 3.9 more years of life without disability than can persons of the same age and sex with less education. They summarize the results as follows:

> Our finding that educational attainment has a strong influence on total life expectancy and active life expectancy among both blacks and whites is of great importance, because educational level, and socioeconomic status in general, are alterable risk factors. At least part of the disadvantage associated with low socioeconomic status relates to poorer lifelong health practices in this group, and efforts must continue to improve these practices. At the population level, however, raising the general level of socioeconomic status may have even more profound effects on health-related behavior and health outcomes (Guralnik et al., 1993).

Yet despite the apparent importance of socioeconomic class for determining health status, the mechanisms by which it exerts its influence are not well described.

> Income, education, and profession are not likely to influence health directly. Instead, these factors are almost certainly proxies for other variables that have a direct impact on health. But what are these variables? Most relevant studies attempt to control for such obvious ones as cigarette smoking and heavy alcohol consumption, both of which are more frequent among the disadvantaged. And the increased frequency of trauma and substance abuse

> among the poor cannot explain the increased mortality and morbidity from other causes. One can imagine a host of other influences—such as diet, stress, exposure to infectious agents or toxic chemicals—that are related to socioeconomic status, but there is very little evidence to point to any of them as the major cause of the health difference between the advantaged and the disadvantaged (Angell, 1993).

The influence of ethnicity and race on health is also well known and, as with socioeconomic status, the causal relationships are poorly understood. While some researchers maintain that ethnicity/race exerts an influence separate from class (Gladwell, 1990; Gibbons, 1991; Bullard and Wright, 1993), others believe that the apparent effects of this variable are due primarily to differences in socioeconomic status (Freeman, 1989, 1991; Navarro, 1990; Baquet et al., 1991). There are data suggesting that ethnicity and race are important factors in explaining, among other things, disparities in health care and residential living patterns. Both of these factors may have significant ramifications for health.

Several recent studies have shown that inequalities in health and in access to health services persist for certain ethnic and racial groups (Bergner, 1993). Escarce et al. (1993) examined Medicare data to document use of 32 medical procedures and diagnostic tests. They found that whites were more likely than blacks to receive 23 of the selected services, while blacks were more likely to receive 7 of the services. Whites had a particular advantage in their access to newer services and to services using higher technology. The authors conclude "there are pervasive racial differences in the use of medical services by elderly Americans that cannot be explained by differences in prevalence of specific clinical conditions. Financial barriers to care do not fully account for these findings. Race may exacerbate the impact of other barriers to access" (Escarce et al., 1993).

In a study of access to health care among adolescents, Lieu et al. (1993) found that in the United States a higher proportion of blacks (16%) and Hispanics (28%) were uninsured than were whites (11%). Moreover, blacks and Hispanics made fewer visits to doctors than whites did, despite reporting worse health status. Greater increases in access to and usage of health care were associated with having health insurance for both whites and minorities; however, racial differences persisted even after adjusting for health insurance, family income, need, and other factors (Lieu et al., 1993).

Denton and Massey (1988) examined the effect of socioeconomic status on residential segregation of blacks, Hispanics, and Asians in 60 U.S. metropolitan areas. They found that blacks are highly segregated from non-Hispanic whites at all socioeconomic levels. In contrast, Hispanic and Asian segregation from whites is lower than that for blacks at all socioeconomic levels, and it declines markedly as these groups move from low to high

socioeconomic status. Their results suggest that, although both Hispanics and Asians seem to be following a process of integration and geographic assimilation comparable to that of earlier European immigrants, blacks continue to confront significant barriers to integration.

> Because blacks continue to be residentially segregated, in spite of their strong desires for integration . . . , middle-class blacks are forced to live in neighborhoods of much poorer quality than whites with similar class backgrounds. No matter what their education or occupational achievements, and whatever their incomes, blacks are exposed to higher crime rates, less effective educational systems, higher mortality risks, more dilapidated surroundings, and a poorer socioeconomic environment than whites, simply because of the persistence of strong barriers to residential integration. As long as black race overshadows class in the allocation of people to residences, it remains a fundamental basis of stratification in U.S. society (Denton and Massey, 1988).

Although disparities in health status by ethnicity and race are substantial, the causes, which undoubtedly include differences in access to health care and residential living patterns, are not well delineated. Polednak (1989) has identified general categories of possible explanations for observed differences in frequency of disease among ethnic and racial groups (see Table 1).

Conceptual Models Relating Class, Race, and Environmental Factors to Health Status

From the preceding discussion, it is clear that health status is the product of multiple variables, many of which are poorly understood. The issue of environmental justice raises difficult but important questions about the role of environmental agents in causing disparities in health status by class and race. Do environmental health risks vary by race and class? If so, to what extent do differences contribute to higher rates of morbidity and mortality among disadvantaged groups? In most instances, there is a shortage of scientific evidence to answer these questions definitively.

Historically, it has been difficult to establish the existence of a causal relationship between exposures to environmental agents and subsequent disease or injury unless the link is strong (e.g., radon-induced lung cancer in uranium miners). The problems that can complicate attempts to verify causality are abundant:

- Incomplete understanding of the etiology of many diseases;
- The wide range of nonenvironmental causes of most diseases to which environmental agents contribute, and the fact that environmental agents often enhance or exacerbate, rather than cause, disease or dysfunction;

TABLE 1. General Categories of Explanations for Reported Differences in the Frequency of Disease Among Racial and Ethnic Groups (Adapted from Polednak, 1989)

Errors of Measurement

- Inadequate data or insufficient information, based on "clinical impressions" or hospital admissions
- Differential access to medical care and diagnostic facilities
- Differential use of available facilities; differences in reporting due to cultural factors or to differences in the severity of disease
- Differing fashions of diagnosis (e.g., in death certification)
- Problems of diagnosis associated with racial or ethnic groups

Differences Between Groups With Respect to More Directly Associated Demographic Variables

- Age differences in the groups compared
- Differences in socioeconomic class and occupation, and secondary factors associated with these differences

Differences in Environment

- Climatic differences and their effects; geographic variation in disease frequency (as in multiple sclerosis)
- Nutrition or diet
- Differences in personal customs or habits (e.g., reproductive and nursing habits, use of tobacco and alcohol, differences in sexual practices)
- Differences with respect to personality development, family dynamics, interpersonal relationships, and role behavior; differences in patterns of psychosocial "stress" or acculturation

Differences in Body Constitution

- Anatomical differences; differences related to rates of growth and development
- Physiologic and biochemical differences: these may be influenced by environmental factors (e.g., diet, exercise) and genetic factors

Genetic Differences (e.g., blood groups)

- Genetic differences among races or ethnic groups may be due to consanguinity, differences in mutation rate, natural selection (differing selection pressures), or random genetic differences due to founder effect and isolation

- Lack of methods, measurements, and models to 1) estimate accurately exposures and absorbed doses for important environmental agents and mixtures of agents, and 2) characterize variability (e.g., within and among individuals, over time and space) in exposures/doses;
- Lack of surveillance (e.g., disease, exposure) and reporting systems related to environmental hazards;
- The long latency period from exposure to adverse health effect, often 20 years or more, for many environmentally induced diseases (e.g., cancer);
- Multiple health effects caused by some environmental agents;
- Occurrence of a single health effect after multiple exposures to either a single agent or a mixture of agents;
- Multiple exposures, both sequential and simultaneous, to a diversity of environmental agents (e.g., chemical, biological, physical);
- The observed health end point (e.g., lung damage) may not be the primary target system (e.g., immune system);
- Lack of methods that are sufficiently sensitive and specific to detect subtle biological changes that portend clinical manifestations of disease or injury;
- Inherent variability among individuals in biological susceptibility to environmentally induced illness.

Therefore, informed discussion and analysis of issues of environmental justice are hampered not only by the complexity and uncertainty surrounding the major determinants of health and the variations in these determinants by class and race, but also by the difficulties associated with establishing the relative role of environmental agents in affecting health status. An important step toward better and more constructive communication, as well as more critical examination of key interrelationships, is the development and refinement of models.

Models provide an intellectual framework that enables researchers to generate testable hypotheses about how and under what circumstances environmental risks might cause differences in health status by class or race. They are conceptual constructs that describe, either qualitatively or quantitatively, the interactions and interdependencies among selected variables. Development and refinement of models is a continuous process of generating, testing, and modifying hypotheses (see Figure 1). It is this logical sequence that forms the basis of the scientific method of investigation.

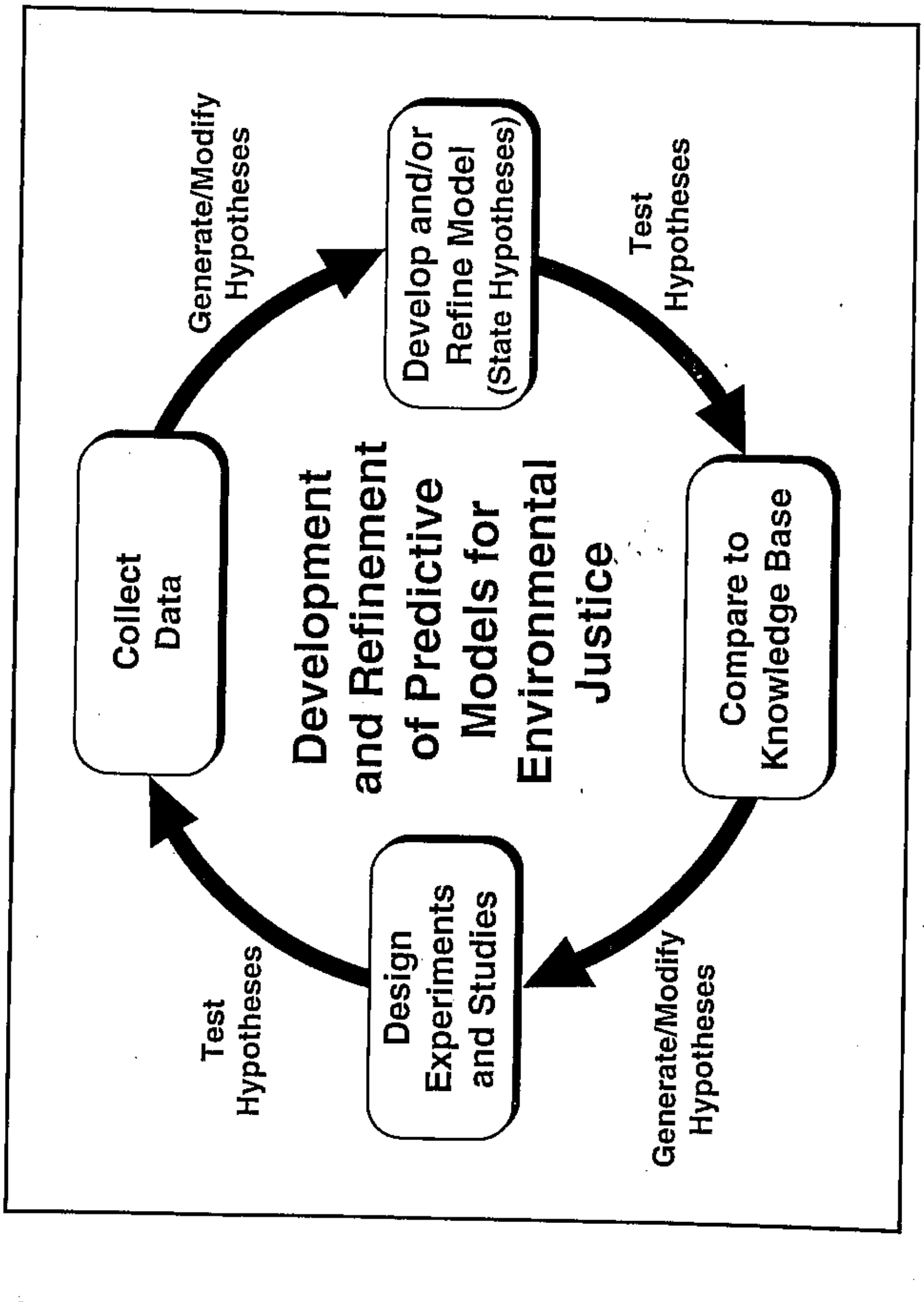

FIGURE 1. Iterative process for generating, testing, and modifying hypotheses as part of model development and refinement.

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Proposing a plausible, testable hypothesis is the first step in designing appropriate research to address issues of environmental justice. Two different conceptual models that have been proposed to explain the relative roles of class, race, and environmental risks in determining health status are summarized in Figure 2.

The two models, one by Freeman (1989, 1991) and the other by Wagener and Williams (1993), attempt to provide a framework to explain the dramatic differences in health status among socioeconomic and racial groups. Freeman focused on the interrelationships among race, poverty, and cancer, and suggested "that poverty acts through the prism of culture" to affect cancer survival rates (Freeman, 1989, 1991). In his model, "race may be seen as a gross variable for culture," where "a population designated by race has common ancestors, similar social and physical environment, and a shared communication system" that tend to promote "a similar tradition, value system, belief system, and world view." He argues that "these shared elements lead to common lifestyle, attitudes, and behavior," which have a powerful effect on health status.

Wagener and Williams (1993) find that "race is a composite measure of a broad range of variables that may affect health status." They suggest "that the term 'race' encompasses biological, cultural, socioeconomic, and sociopolitical factors, as well as racial discrimination." Furthermore, these factors are interrelated and may, either individually or interactively, "affect health through intermediary mechanisms and processes that include health practices, psychosocial stress, environmental stress, psychosocial resources, and medical care."

Both models provide useful constructs with which to visualize the complex and interrelated factors that affect health status. Neither, however, tries to quantify or prioritize the relative importance of these explanatory variables. The specific effect of differential environmental health risks on increased prevalence of disease and decreased life expectancy is left unresolved. Accordingly, both models are considered to be qualitative.

Additional insight into differences in health status is provided by a model proposed by Polednak (1989), which describes the possible relationship between "acculturation" and health (Figure 3). Acculturation is defined as "... those phenomena which result when groups of individuals having different cultures come into continuous first-hand contact with subsequent changes in the original culture patterns of either or both groups" (Polednak, 1989). His model attempts to describe a composite of the kinds of effects that have been observed in ethnic/racial groups from "developing" cultures that come into contact with industrialized societies (e.g., blacks, Hispanics, Pacific Islanders in the United States).

As Polednak observes, "it is clear from numerous studies in numerous populations undergoing various rates of acculturation that the disease pattern changes over time and at varying rates, depending on the length of time that acculturation has occurred and numerous other factors

FIGURE 1. Iterative process for generating, testing, and modifying hypotheses as part of model development and refinement.

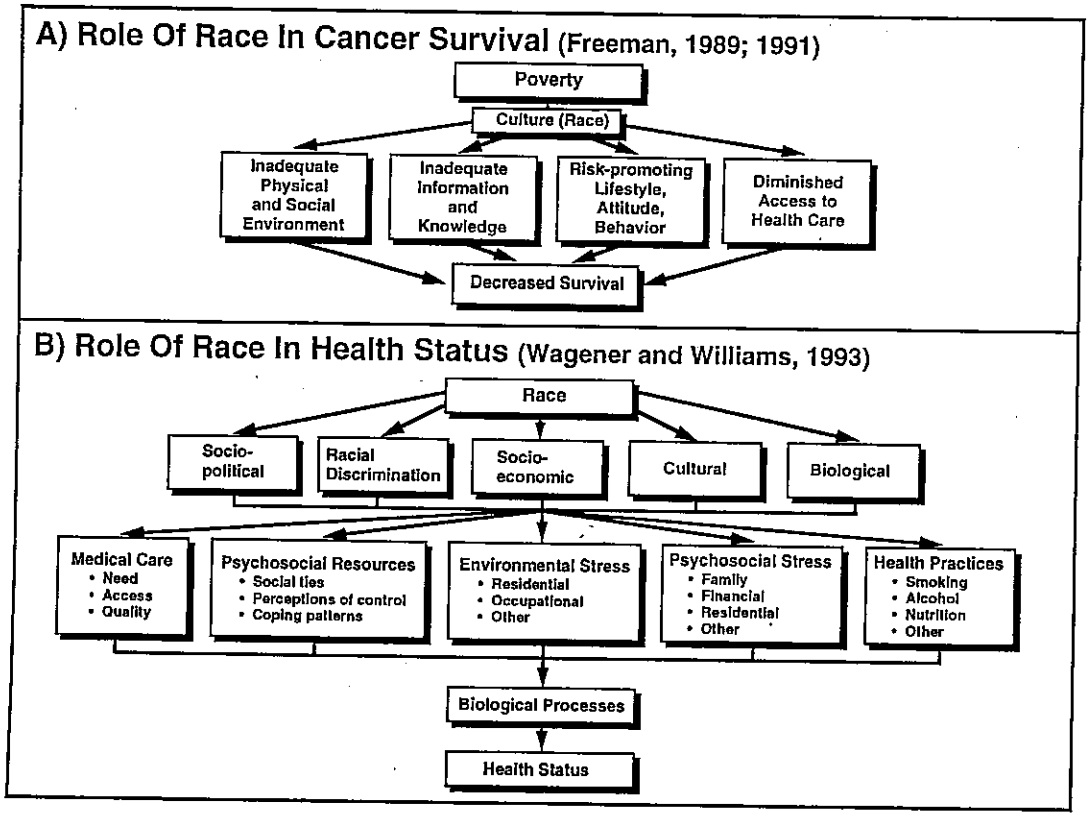
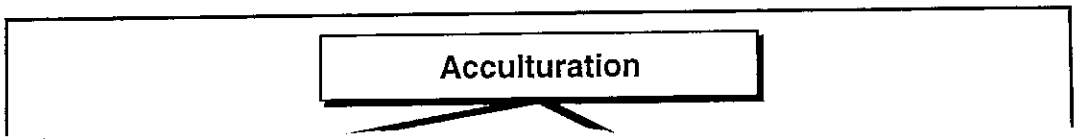


FIGURE 2. Conceptual models of A) the role of race in cancer survival (Freeman, 1989, 1991) and B) the role of race in health status (Wagener and Williams, 1993).



Health Status

FIGURE 2. Conceptual models of A) the role of race in cancer survival (Freeman, 1989, 1991) and B) the role of race in health status (Wagener and Williams, 1993).

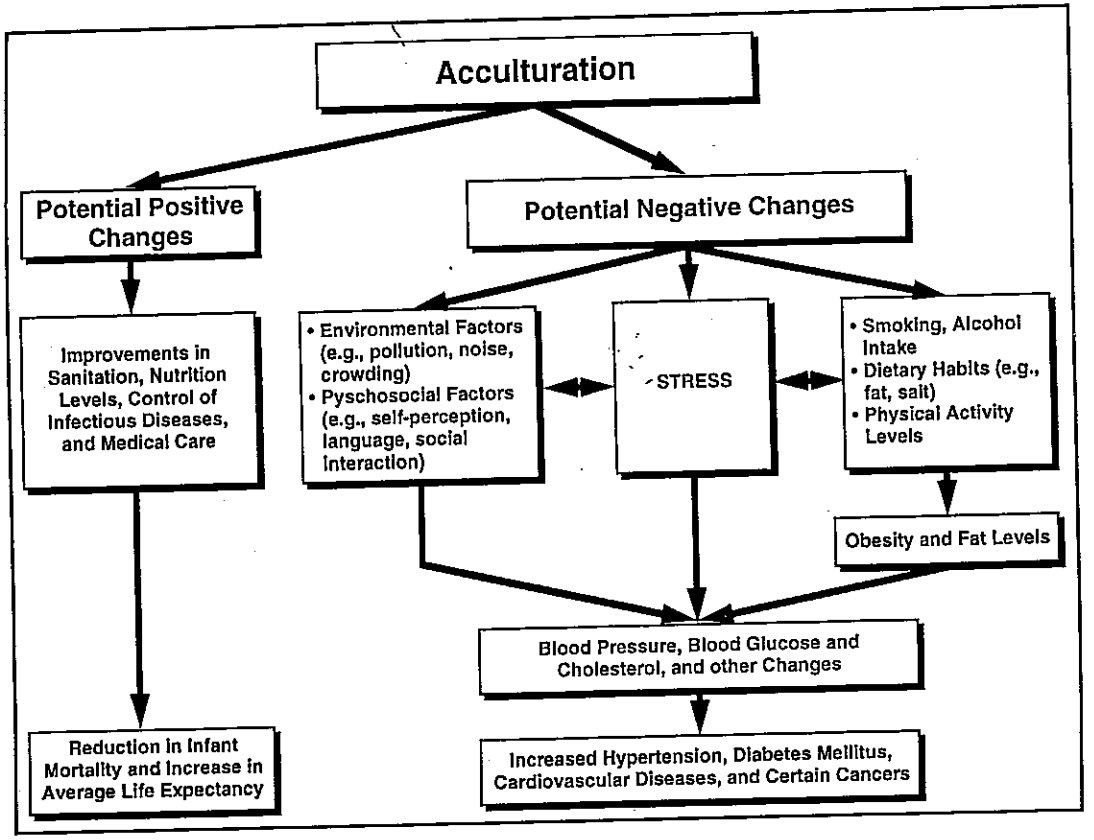


FIGURE 3. Conceptual model of the potential positive and negative effects on health from acculturation (adapted from Polednak, 1989).

such as the degree of segregation of the previously unacculturated group (especially with regard to quality of medical care)" (Polednak, 1989). He suggests that the "double-edged sword of acculturation" is evident in the decline in infectious diseases, after long contact, but also in the increases in blood pressure, certain cardiovascular diseases, and some cancers (Polednak, 1989). In his opinion, "Even in such long-standing minority groups as U.S. blacks, recent changes in mortality from cardiovascular diseases probably reflect in part the continuing process of acculturation" (Polednak, 1989).

Polednak's model provides another perspective on the possible explanations for ethnic/racial differences in health status. He suggests that acculturation may cause significant alterations in patterns of disease and death for ethnic and racial groups. Yet there is still substantial uncertainty about the relative contributions of various factors, including environmental, psychosocial, and lifestyle variables. Like the models proposed by Freeman (1989, 1991) and Wagener and Williams (1993), Polednak postulates a simplified, qualitative construct to describe some of the interrelationships among major determinants of health status.

Conceptual models such as these explicitly state a theory about the causal relationships between important explanatory variables (e.g., medical care, diet, environmental agents) and health status. They are a necessary first step in formulating hypotheses that can then be subjected to scrutiny and testing through scientific research.

FRAMING THE CONCEPT OF "ENVIRONMENTAL JUSTICE" IN TERMS OF RISK

Risk-Based Priority Setting for Environmental Health Problems

The magnitude and extent of environmental problems, the associated costs of remediation and mitigation, and the need to balance the nation's budget are forcing hard societal choices about strategic directions and how resources will be allocated among competing needs. A broad-based consensus seems to be emerging that "risk-based priority setting" is the method of choice to ensure that scarce resources are used to address the "worst" problems first. In essence, risk-based priority setting compares and ranks risks to establish priorities for resource allocations (Sexton, 1993).

Comparison of health risks is not new. In the late 1970s and early 1980s, for example, several groups examined and compared the relative cancer risks in the United States attributable to various factors, including diet, tobacco use, occupation, and environmental agents (Wynder and Gori, 1977; Higginson and Muir, 1979; Doll and Peto, 1984). Moreover, it has been recognized for some time that there is a need for "comparative risk assessment," that is, we need to provide a context for interpreting health risks by comparing risks from exposures to environmental agents (e.g., ambient air pollution) with commonplace risks, such as those from auto accidents (Wilson and Crouch, 1987).

What is new about risk-based priority setting is its insistence, indeed its demand, that resources be targeted on those environmental health problems that represent the greatest risks to the public health and welfare (Sexton, 1993). Among the advocates for this approach are some members of Congress (Baucus, 1993; Lewis, 1993; Moynihan, 1993), the EPA (Reilly, 1990; Sexton, 1993), EPA's board of outside scientific advisors (SAB, 1990), and the Carnegie Commission (Carnegie, 1993). Although there are lingering concerns about the feasibility and utility of risk-based priority setting (Finkel, 1993; Finkel and Golding, 1993; RFF, 1993; Sexton, 1993), it is already being implemented at local, state, and federal levels of government (Sexton, 1993).

One potential problem with the risk-based approach is that it may not result in a fair and equitable outcome (Comparative Risk Bulletin, 1993; Finkel and Golding, 1993; RFF, 1993). For example, Robert Bullard, professor of sociology at the University of California, Riverside, has argued that environmental protection is not a privilege to be doled out according to a process of "environmental triage" but a right for all individuals. Bullard faults the comparative risk approach for helping to institutionalize a system of unequal environmental protection across racial and class lines. As an alternative, he calls for an "environmental justice paradigm," which puts priority on all of the "obvious" geographic areas where minorities and the poor face multiple risks from many sources (Finkel and Golding, 1993).

Clearly, issues of equity and justice involve value judgments that cannot be addressed adequately by relying exclusively on quantitative risk assessments (Sexton, 1993). We recognize that a risk-based approach has limitations, and that it is not sufficient by itself to deal adequately with the relevant social, ethical, and moral issues. Nevertheless, concerns about disparities in environmental health risks across different segments of society are basically what environmental justice is all about. We believe, therefore, that "environmental health risk" is a useful intellectual construct for analyzing the role of scientific research in evaluating and resolving issues of environmental justice.

Assessment and Management of Environmental Health Risks

Actions taken by society to protect its members from the harmful health consequences of pollution are predicated on established or postulated links among pollution sources, human exposures, and adverse health effects. If environmental health risks, which result from exposures to environmental toxicants at levels sufficient to cause illness or injury, are deemed unacceptable, then public or private intervention is justified to bring them within acceptable limits.

The premise underlying much of the ongoing debate about environmental justice is that disadvantaged communities, including low-income and minority groups, encounter, on average, higher exposures to environmental agents than do white, middle-class Americans. It

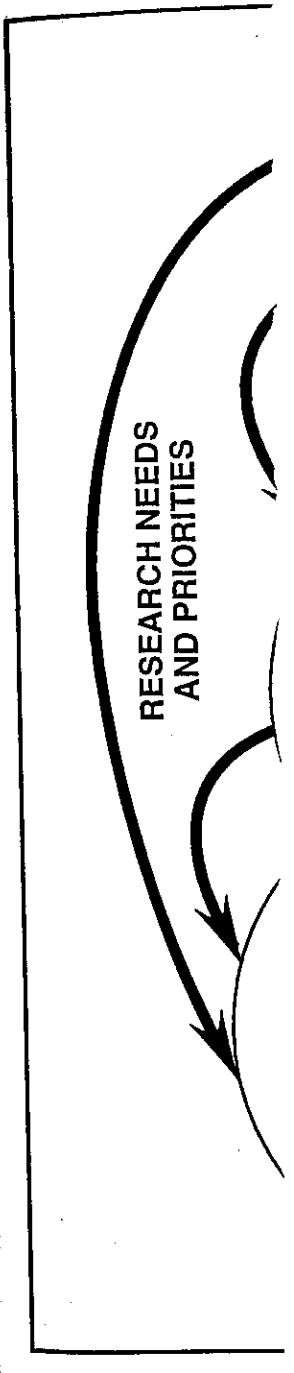
is further postulated that these elevated exposures increase the probability of environmentally induced illness and injury, thereby contributing to observed disparities in health status. Thus, identification and evaluation of environmental health risks are fundamental to determining whether society has achieved environmental justice for everyone, and, if not, how best to rectify the situation.

Credible risk assessment and risk management decisions must be based on credible science (EPA, 1992b). Scientific research is the foundation upon which to base improvements in the quality of decisions about what actions to take to prevent or reduce unacceptable health risks. The interrelationships among environmental health research, risk assessment, and risk management should form a feedback loop that fosters more informed judgments (see Figure 4). The feedback loop requires that information flow in two directions. First, the information needs identified as part of risk assessment and management must drive the direction and nature of supporting research. And second, the information and understanding generated by the research program must directly improve the scientific basis for decisions about ameliorating risks (Sexton, 1992).

The simplified representation (Figure 5) of the key steps intervening between the emissions of toxic agents into the environment and potential disease or dysfunction in human populations is an "environmental health paradigm." The chain of events represented by the environmental health paradigm can serve as a useful conceptual basis from which to understand and evaluate environmental health risks (Sexton, 1992).

Risk assessment is a formalized, structured process used to estimate the magnitude, likelihood, and uncertainty of environmentally induced health effects (NRC, 1983). Professional risk assessors, using the best available science, attempt to answer four crucial questions related to different aspects of the environmental health paradigm (Figure 6) (Sexton, 1992):

- Hazard Identification (qualitative)—Is the environmental agent capable of causing an adverse effect in humans? (Is it intrinsically hazardous?)
- Dose-Response Assessment (quantitative)—What is the relationship between dose to the target tissue and adverse effects in humans?
- Exposure Assessment (quantitative)—What environmental exposures occur or are expected to occur in human populations, and what is the resulting dose to the target tissue?
- Risk Characterization (quantitative)—Based on a synthesis of exposure and dose-response assessments, what is the estimated human health risk from the anticipated environmental exposures?



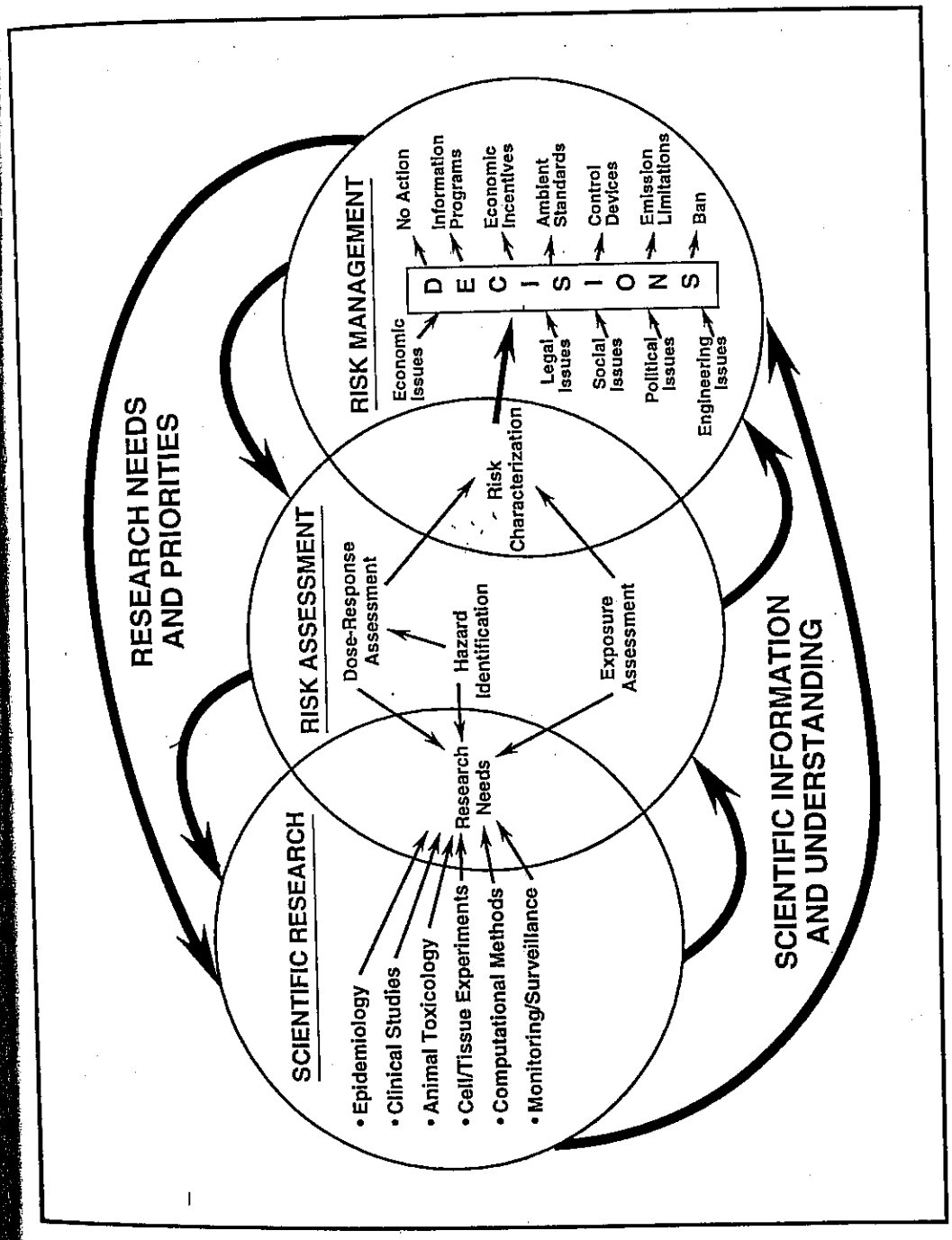


FIGURE 4. The feedback loop connecting scientific research, risk assessment, and risk management (Sexton, 1992).

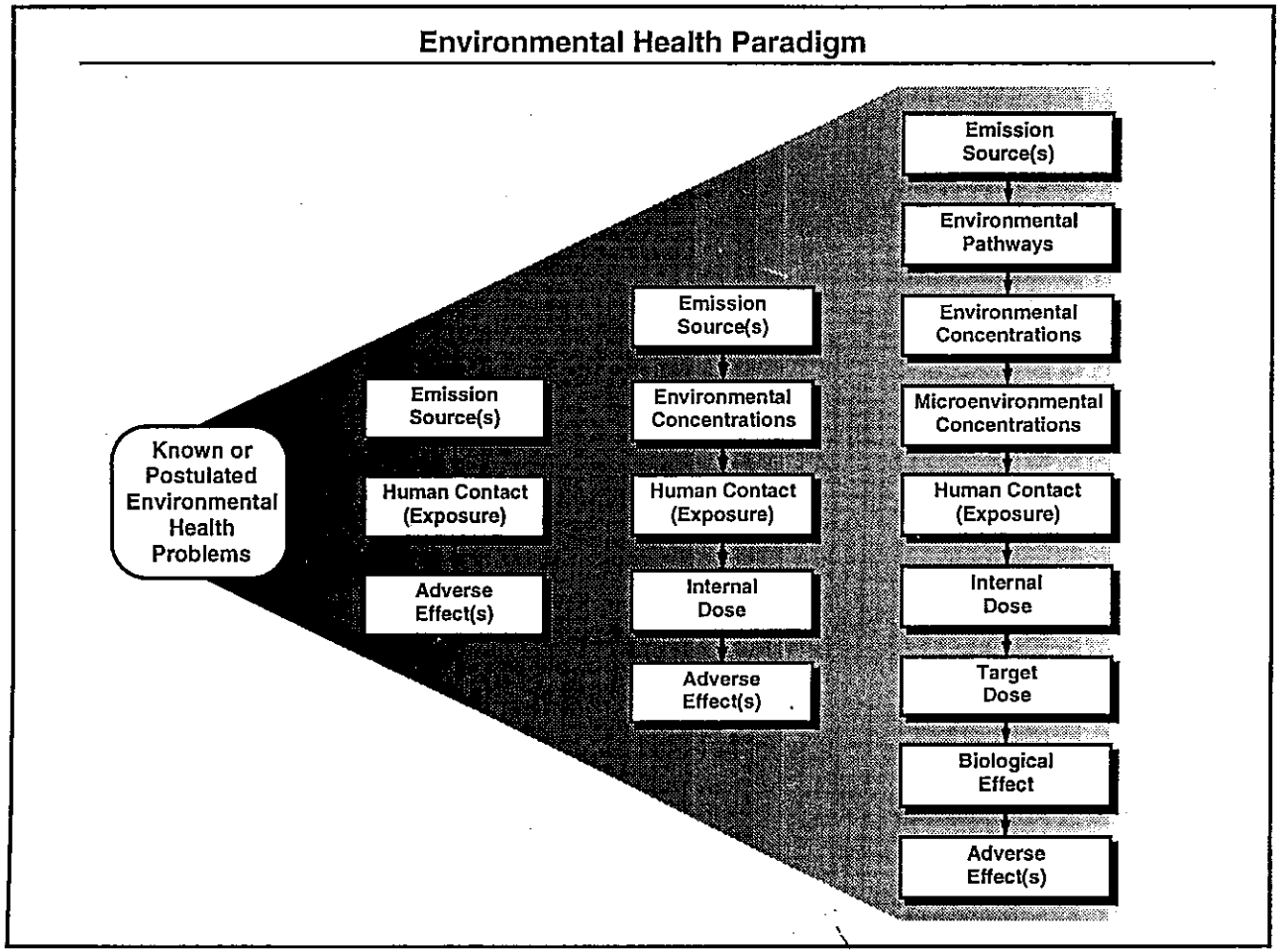
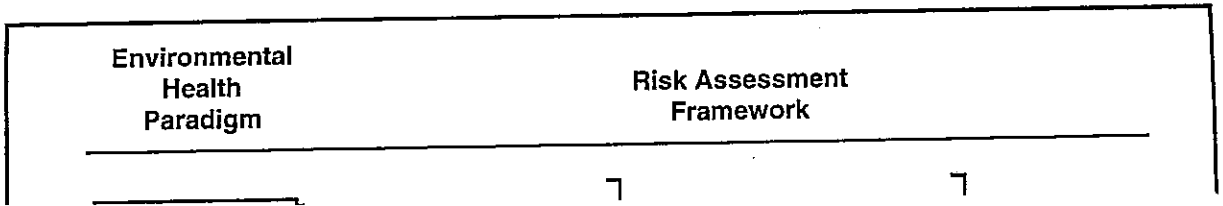
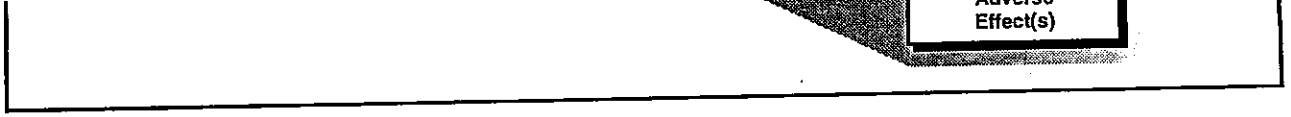


FIGURE 5. An environmental health paradigm identifying some of the important events leading from release of an agent into the environment to adverse health effects in people.





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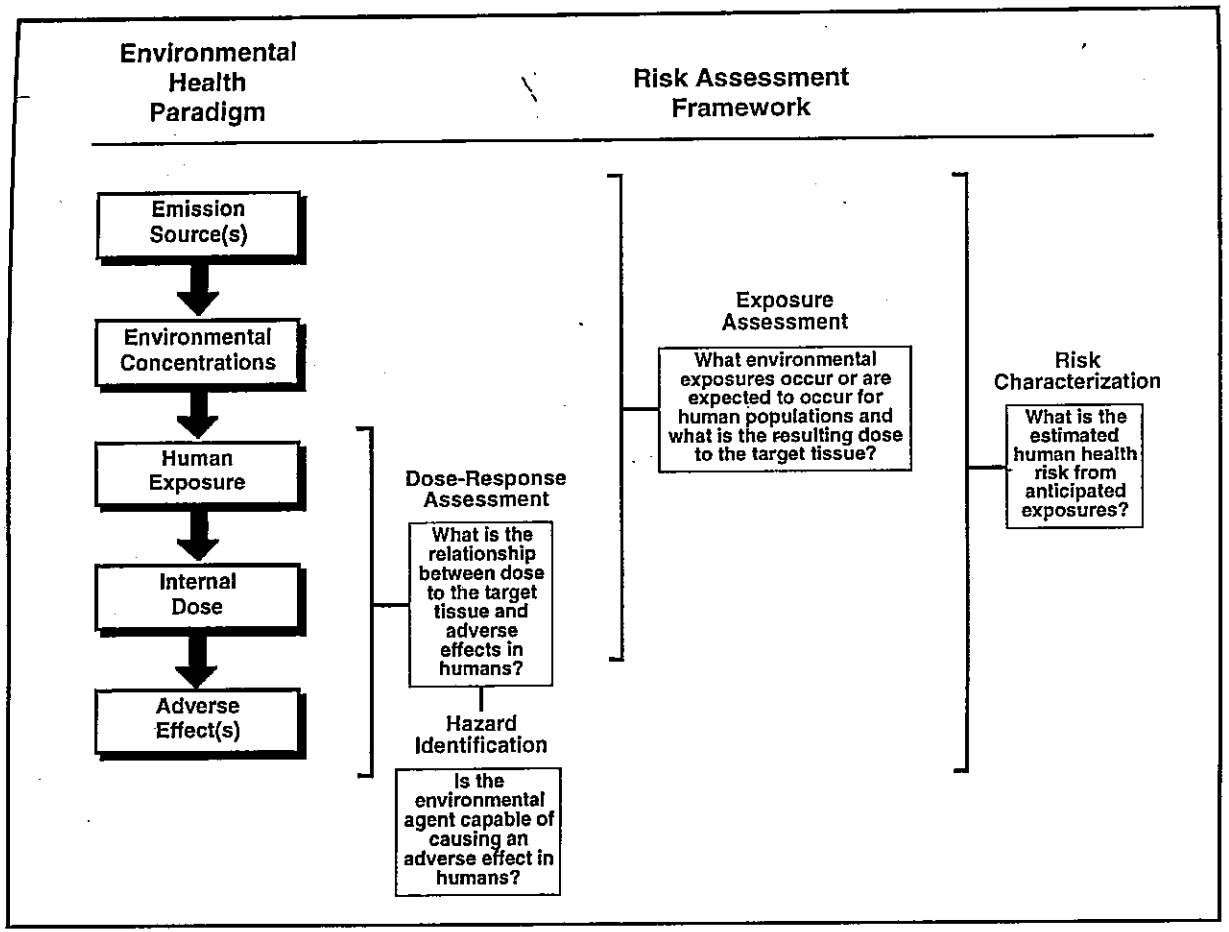


FIGURE 6. The risk assessment process and its relationship to important events in the environmental health paradigm.

In the process called risk management, results of the risk assessment are used to aid policy makers in deciding whether risks are unacceptable, and, if so, what to do about them. Although quantitative risk estimates are an important aspect of most policy and regulatory decisions, they are by no means the only consideration. Risk managers must also take into account other factors, such as economic, social, legal, and political issues, as well as issues about whether control is feasible, before they decide what, if any, actions are needed to safeguard public health (NRC, 1983; Sexton, 1992).

The Importance of Identifying and Evaluating Groups Potentially at Greater Risk

A primary goal of risk-based approaches is to identify and evaluate those populations, subpopulations, and individuals at highest comparative risk so that, if warranted, appropriate mitigation actions can be implemented. Conceptually, individuals and groups are deemed to be at potentially greater risk when they are 1) exposed above some health-related benchmark, and/or 2) more susceptible to the effects of exposures (see Figure 7).

Measures of central tendency, such as the median and average, along with expressions of variability, like the geometric standard deviation, are commonly used to describe the distribution of exposures for a population (See Figure 8A). Often, the relative position of an individual or group in the exposure distribution is of primary interest for risk assessment. Among the most frequently used exposure descriptors are 1) values near the middle of the distribution, 2) values above the 90th percentile, which is arbitrarily defined by EPA as the "high end" of the distribution, and 3) values at the extreme upper end, such as for the most exposed person in the population (EPA, 1992c).

But exposures of concern are not necessarily just those above the 90th percentile (see Figure 8B). For many systemic (noncancer) toxicants, benchmarks, such as reference concentrations, ambient standards, and workplace exposure limits, have been established to protect public health whether they are above the 90th percentile or not. Exposures that exceed these values raise concerns about potentially elevated health risks. Currently, however, quantitative assessment of noncancer risks is problematic in most cases because the shape of the dose-response curve (i.e., the relation between exposure/dose and adverse health outcomes) above the benchmark is not well defined. Nevertheless, those whose exposures are above such benchmarks are also generally considered to be at increased risk.

Quantitative risk assessment for carcinogenicity is a well-established, albeit controversial procedure. It is possible to estimate cancer risk for individuals directly from the exposure (dose) distribution by assuming a linear, nonthreshold model for carcinogenicity (see Figure 8C). Under this assumption, it is also possible to estimate the number of excess cancer cases in the entire population by multiplying the average dose by the total number of people exposed by the dose-response slope factor. Typically, the highly exposed category is defined to include all those whose estimated individual risk for developing cancer (based on their putative exposure) is above some arbitrarily defined minimal risk level (e.g., 1 in 1,000,000).

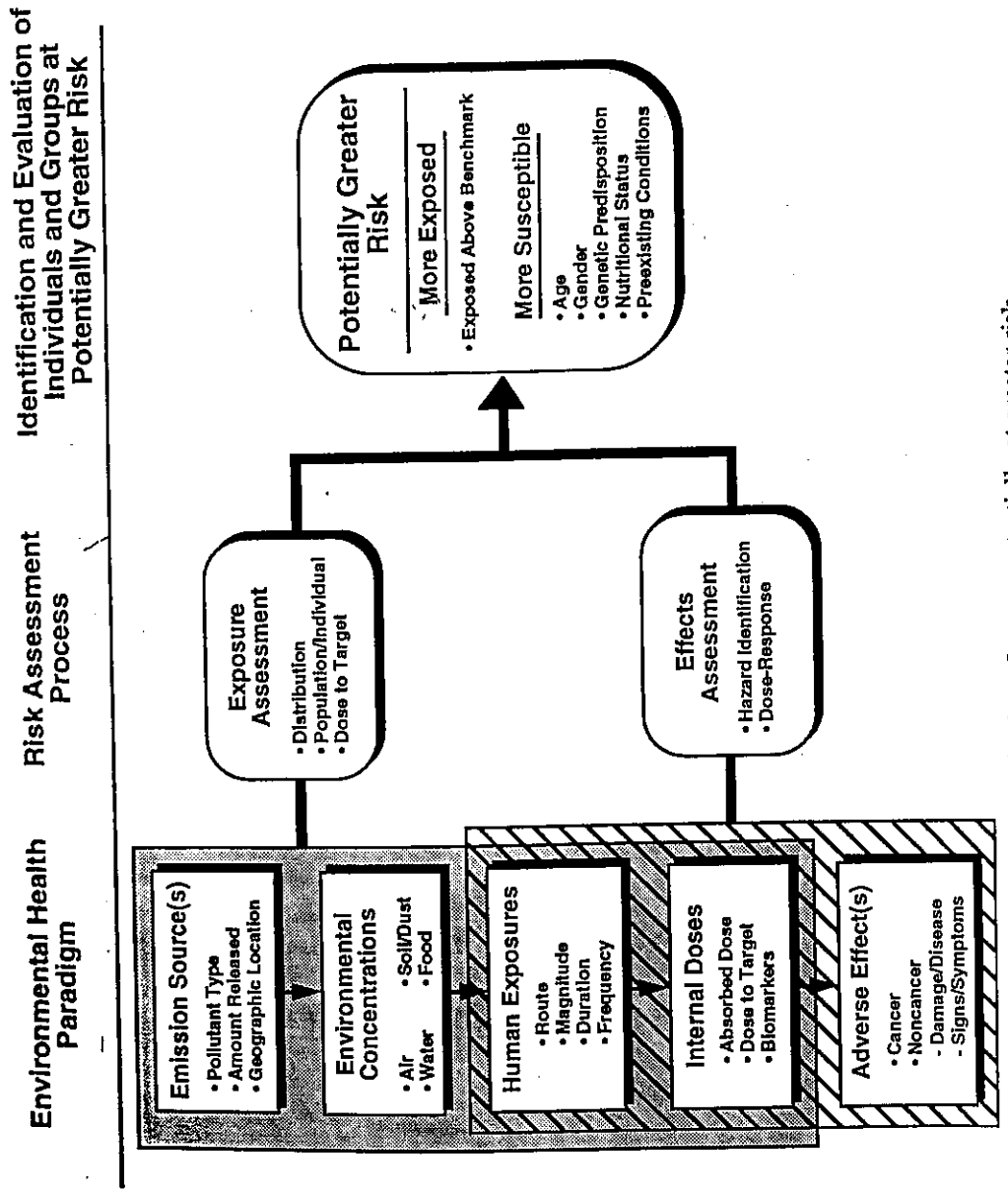


FIGURE 7. Identification and evaluation of groups potentially at greater risk.

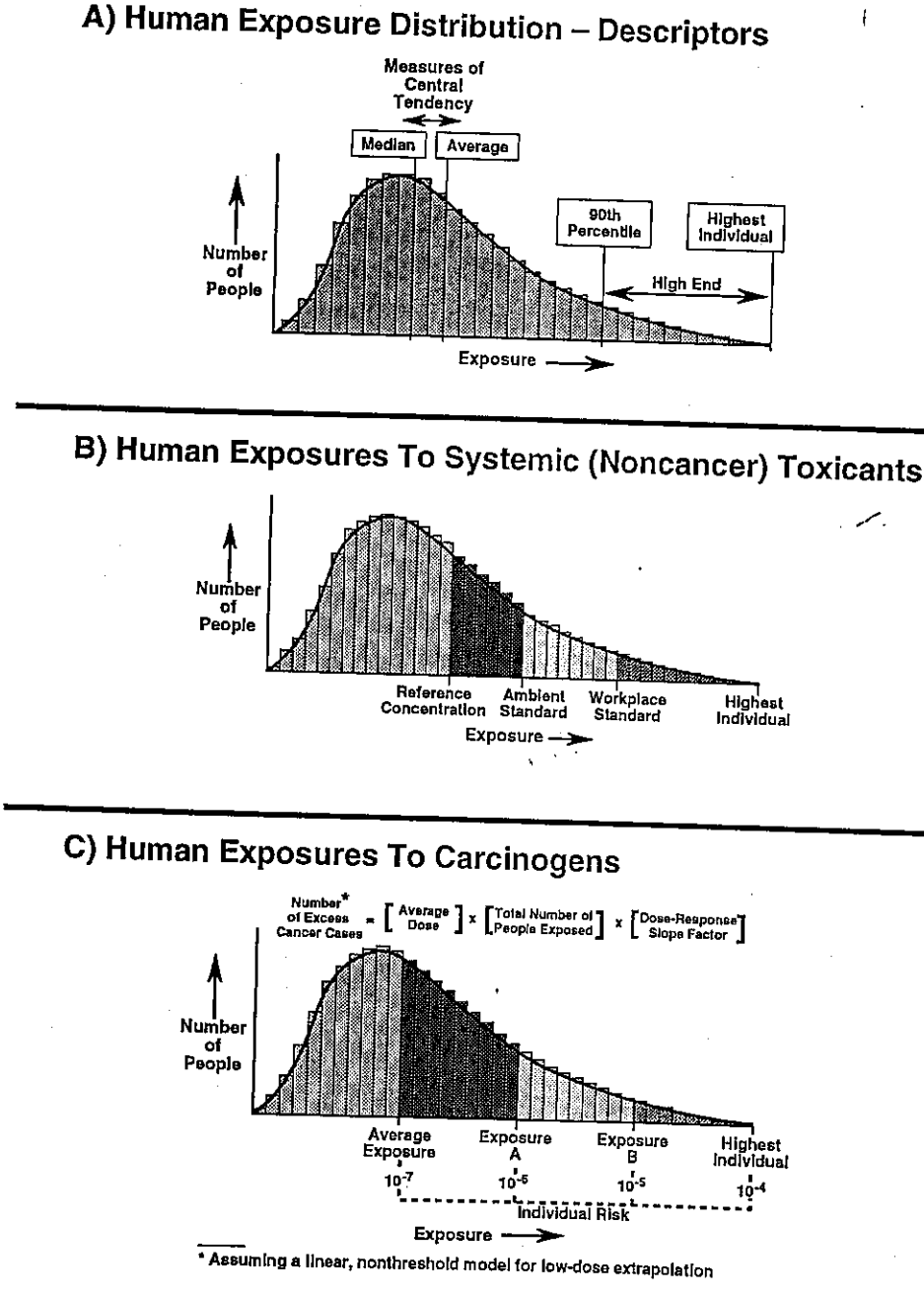


FIGURE 8. The distribution of human exposures to an environmental agent in the general population: a) generic descriptors; b) for a systemic toxicant; and c) for a carcinogen (Sexton, 1993).

Individuals and groups can also be at increased risk because they are more susceptible to the adverse effects of a given exposure. Among the factors that can increase susceptibility are inherent genetic variability, age and gender differences, preexisting conditions (e.g., disease), inadequate diet, lifestyle factors (e.g., smoking), stress, and inadequate access to health care (Rios et al., 1993). Systematic differences in these factors occur both within and among racial and ethnic groups in the United States (Calabrese, 1984; Polednak, 1989; Rios et al., 1993), but, with few exceptions, their relation to enhanced susceptibility has not been firmly established.

Inherent genetic variability, for example, is often cited as a potential cause of differential susceptibility to environmental exposures (Calabrese, 1984; Polednak, 1989; Rios et al., 1993). There is a substantial body of evidence showing differences in genetically determined traits among certain racial and ethnic groups, including differences in anthropometric, physiologic, and biochemical characteristics between blacks and whites (Table 2) (Polednak, 1989), and differences in response to cardiovascular agents (Table 3) and central nervous system agents (Table 4) (Levy, 1993) among a variety of groups. There are also data suggesting that genetic factors affect susceptibility to certain environmental carcinogens (Table 5) (Vine and McFarland, 1990). In general, however, the extent to which genetic factors, and other potential contributors as listed above, contribute to increased susceptibility and to related increases in environmental risks, are not well elucidated.

TABLE 2. Examples of Anthropometric, Physiologic, and Biochemical Characteristics of Black and White Populations (Adapted from Polednak, 1989)

Higher in Blacks	Lower in Blacks
Density of bone	LDL cholesterol in blood
HDL cholesterol in blood	Leukocyte count in blood
Fibrinolytic activity in blood	Hemoglobin concentration in blood
Testosterone in blood (males)	Lactase deficiency
Red cell sodium concentration	Width of female pelvis
Blood pressure (U.S., parts of Africa)	Birth weight and gestation length
Intraocular pressure	
Body fat (adult females)	
Lean body mass and density	
Leg length	
Shoulder/hip breadth	

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

TABLE 3. Examples of Ethnic and Racial Differences in Response to Cardiovascular Agents (Adapted from Levy, 1993)

Comparison Groups	Drug Classes/Examples	Clinical Response
Blacks/Whites	Beta-blockers, especially propranolol (also nadolol, pindolol, atenolol)	Blacks less responsive
Blacks/Whites	Labetalol, combined alpha- and beta-blocker	Equally effective in blacks and whites
Blacks/Whites	Diuretics e.g., hydrochlorothiazide	Blacks respond better to monotherapy
Blacks/Whites	ACE inhibitors e.g., captopril	Monotherapy more effective in whites; no difference in diuretic added
Chinese/Whites	Propanolol	Chinese twice as sensitive to effects on blood pressure and heart rate
Chinese/Whites	Atropine	Chinese show greater increases in heart rate

TABLE 4. Examples of Ethnic and Racial Differences in Response to Central Nervous System Agents (Adapted from Levy, 1993)

Comparison Groups	Drug Classes/Examples	Clinical Response
Chinese/Whites	Benzodiazepines (diazenpam, alprazolam)	Chinese require lower doses; more sensitive to sedative effects
Chinese/Whites Hispanics/Anglos	Antidepressants (imipramine, desipramine amitriptyline, clomipramine)	Chinese and Hispanics require lower doses; side effects greater in Hispanics
Asians/Whites	Neuroleptics (e.g., haloperidol)	Asians require lower doses
Asian Indians/Whites	Analgesics (e.g., acetaminophen, codeine)	Asian Indians have greater clearance rates
Chinese/Whites	Analgesics (e.g., morphine)	Chinese less sensitive to cardiovascular and respiratory effects, but more sensitive to GI side effects
Asians/Whites	Alcohol	Asians more sensitive to adverse effects
Native Americans/Whites	Alcohol	Native Americans have faster metabolism and less tolerance

TABLE 5. Selected Genetic Factors Suspected of Being Associated with Increased Susceptibility to Cancer (Adapted from Vine and McFarland, 1990)

Genetic Susceptibility Factor	Environmental Agent	Disease
Debrisoquine hydroxylation phenotype	Cigarette smoke Aflatoxin	Lung cancer Liver cancer
Acetylation phenotype	N-substituted aryl compounds	Bladder cancer
Heterozygous for ataxia-telangiectasia gene	Ionizing radiation	Cancer of the breast, ovary and gastrointestional tract, as well as lymphoma, and leukemia
Skin color	Ultraviolet light	Skin cancer
Xeroderma pigmentosum	Ultraviolet light	Skin cancer
Aryl hydrocarbon hydroxylase (AHH) inducibility	Polycyclic aromatic hydrocarbons	Lung cancer

Despite the uncertainties involved with estimating both exposure and susceptibility, the basic tenet remains the same. For disparities in environmental health risks to occur by socioeconomic status or ethnicity/race, these demographic variables must be associated with systematic differences in (1) exposure to environmental agents, (2) susceptibility to the effects of environmental agents, or (3) both exposures and susceptibilities. Thus, research to investigate whether environmental justice is being achieved should focus specifically on determining whether certain segments of the population are disproportionately more exposed and/or more susceptible to environmental agents.

A Conceptual Model Relating Class and Race to Environmental Health Risks

The conceptual models of Freeman (1989, 1991), Wagener and Williams (1993), and Polednak (1989) provide qualitative theories about the major determinants of health status (e.g., health care, diet, environmental agents). They do not provide much help, however, in developing testable hypotheses about how class or race might cause disparities in environmental health risks. This section outlines a conceptual model that lets us postulate how class and/or race may be causally related to higher exposures or to increased susceptibility, and therefore to higher than average health risks.

Hypothesized, though as yet largely unexplored interrelationships among class and race, exposure- and susceptibility-related attributes, and environmental health risks are shown in Figure 9. By postulating causal relationships between class and race (explanatory variables) and environmental health risks (outcome variable), the model provides a conceptual basis that enables us to generate testable hypotheses about environmental justice. The model highlights three critical questions.

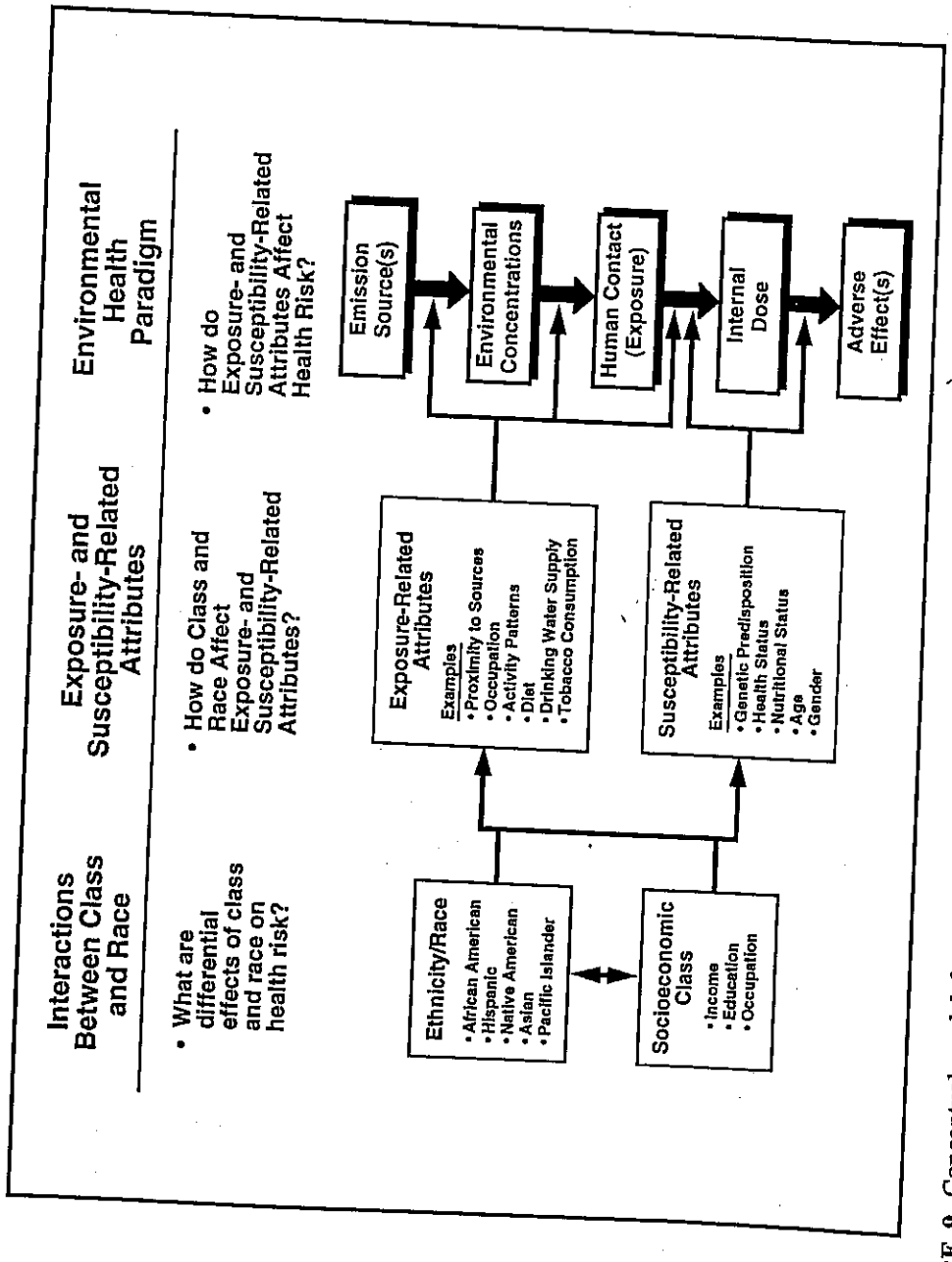


FIGURE 9. Conceptual model for generating testable hypotheses about causal relationships between demographic variables and environmental health risks.

(1) How do important exposure- and susceptibility-related attributes affect environmental health risks?

(2) How do class and race affect important exposure- and susceptibility-related attributes?

(3) How do class and race differentially affect environmental health risks?

Resolution of the first question allows for realistic and quantitative assessment of environmental health risks encountered by those at potentially greater risk. Answers to the second question provide an indication of whether certain demographic groups are disproportionately represented in at-risk categories. Answers to the third question illuminate some of the interrelationships among class and race and afford an opportunity to separate the relative influence of each on environmental health risks.

These three questions are not independent of one another. Answers to the first question allow us to raise the second, and, taken together, answers to the first two questions let us draw some tentative conclusions about the third. These tentative conclusions can be formulated into testable hypotheses by proposing quantitative answers to the question: how do class and race differentially affect environmental health risks?

Scientifically credible answers to this question will tell us which socioeconomic and ethnic/racial factors contribute most significantly to environmentally related risks. It will then be possible to begin identifying viable mitigation/remediation strategies, and to compare them on the basis of effectiveness, efficiency, and equality. Although other factors (e.g., social, political, legal) will enter into societal decisions about how best to safeguard public health, answers to the three questions posed above (see Figure 9) will provide a sound scientific basis for decisions about whether injustices exist, and, if so, what to do about them.

RESEARCH TO EVALUATE AND RESOLVE "ENVIRONMENTAL JUSTICE" ISSUES

Duality of Research Goals Related to Environmental Justice

An apparent dichotomomy of opinion about appropriate goals for research related to environmental justice seems to exist between affected communities and many in the general scientific community (Bryant and Mohai, 1992a; Bullard and Wright, 1993; Comparative Risk Bulletin, 1993; Finkel and Golding, 1993; RFF, 1993). The "community perspective," represented by, among others, civil rights leaders, community activists, religious leaders, involved citizens, and some social scientists, holds that the available evidence is sufficient to justify intervention to mitigate risks in disadvantaged communities (Bullard and Wright, 1993). Many of those who take this position believe that "environmental racism" is an important factor contributing to existing problems. From their point of view, the goals of

Effect(s)

FIGURE 9. Conceptual model for generating testable hypotheses about causal relationships between demographic variables and environmental health risks.

research on environmental justice issues should be 1) to remedy inequities and injustices, 2) to prevent such occurrences in the future, and 3) to ensure full and meaningful participation by all affected parties, especially poor and minority communities, in the decision making related to issues of environmental justice.

Many scientists, on the other hand, tend to see the goals of environmental justice research primarily in terms of 1) identifying, 2) assessing, 3) comparing, 4) managing, and 5) communicating health risks associated with exposures to environmental agents. From their perspective, it is important that relevant and timely research be conducted to elucidate the magnitude and extent of any problems, and to understand cause-and-effect relationships. In this way, research can be used to put problems into context and to strengthen the scientific basis for decisions about environmental justice.

It is not surprising that misunderstandings have occurred between adherents of these two perspectives. From the community perspective, there are concerns about past failures of risk-based approaches to ensure equity and justice. In addition, there are also ongoing concerns about the possibility that research might be used as an excuse to do nothing. From the risk-based perspective, there is uneasiness because the community approach seems to substitute advocacy for science.

As interactions between proponents of both perspectives have increased over the past few years (EPA, 1992a; Johnson et al., 1992), an awareness of common purpose and shared objectives has gradually emerged. Those coming from a community perspective have increasingly recognized and acknowledged the importance of building a strong scientific basis for action. Those coming from a risk-based perspective have increasingly recognized and acknowledged the need to sometimes take corrective action based only on preliminary scientific evidence.

Both factions are beginning to see that their respective research goals are complementary rather than conflicting. It is not a question, then, of choosing one approach over the other. Both approaches are consistent with the public health emphasis on prevention as the primary goal and mitigation as a secondary goal.

Future research to improve our understanding of environmental justice is likely to be conducted within a risk-based framework. That framework must, however, encourage community involvement throughout the process, and make specific provisions for addressing stakeholder concerns. Because assessment of environmental health risks is central to addressing issues of environmental justice, we briefly examine the types of research needed to reduce the most critical scientific uncertainties.

Research to Reduce Uncertainties in Health Risk Assessment

Quantitative risk assessment in general, and evaluation and resolution of environmental justice

issues in particular, are impeded by two generic problems: 1) lack of data, which causes errors of estimation and omission; and 2) lack of scientific understanding, which causes errors of specification and extrapolation (Table 6). Filling key gaps in the data by conducting toxicologic, epidemiologic, or clinical studies will reduce estimation and omission errors, while applying mechanistically based methods and models will reduce specification and extrapolation errors. In the first instance, research strengthens our ability to estimate risks by enhancing the quality and quantity of data available, and in the second, it accomplishes the same objective by improving our ability to interpret these same data.

In trying to remedy the first problem, lack of data, existing methods and models are used to collect suitable information. Testing for carcinogenicity in an appropriate animal model, for example, could potentially decrease the uncertainty about whether the agent is intrinsically hazardous. Similarly, analytic epidemiologic studies in relevant occupational settings might reduce uncertainties about whether the agent is a human carcinogen, and/or provide data to improve estimates of dose-response relationships in people.

The second problem, lack of understanding, is commonly referred to under the rubric of "extrapolation issues." Extrapolation means 1) to infer values of a variable in an unobserved interval from values within an already observed interval; or 2) to project, extend, or expand known data or experience into an area not known or experienced so as to arrive at conjectural knowledge of the unknown area.

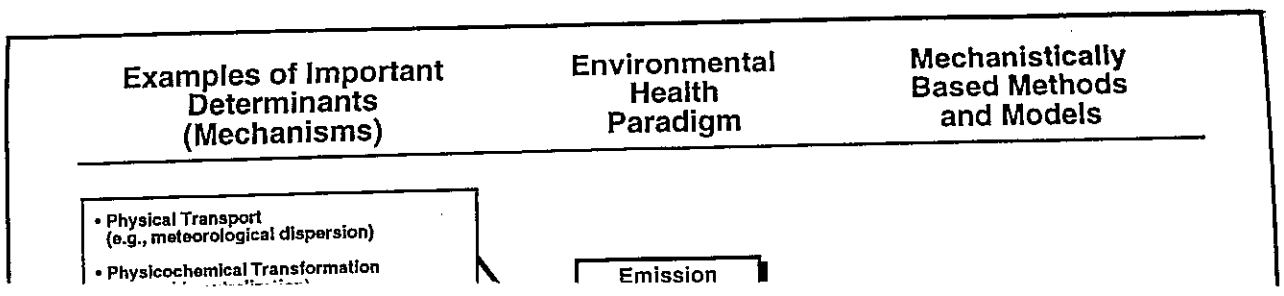
Risk assessors essentially take what is known about the environmental health paradigm, make assumptions as necessary, and estimate risks. During this process, they are often confronted with the need to extrapolate data from experimental conditions to real-world situations, or from one real-world situation to another. This typically necessitates extrapolating information from species to species, from one individual or subgroup to another individual or subgroup within the same species, from one set of exposure conditions to another, or from acute to chronic effects. Informed, realistic extrapolation is frequently impaired by a lack of knowledge and understanding about two general issues: 1) the basic mechanisms that determine exposure, dose, and effects in people; and 2) the extent to which pharmacokinetic and pharmacodynamic mechanisms are comparable between laboratory animals and humans.

"Mechanisms" are fundamental processes or factors that determine exposure, dose, or effects in either animals or people. They may be biological (e.g., movement of lead across the blood-brain barrier), chemical (e.g., interactions to form lead acetate), physical (e.g., lead in plumbing leaching into drinking water), or sociological (e.g., lifestyle attributes that affect consumption of tap water). Although mechanisms are diverse, they share common functional properties: they control, determine, and/or regulate key processes delineated in the environmental health paradigm (see Figure 10).

TABLE 6. Comparison of Two Generic Types of Problems that Create Uncertainties in Health Risk Assessments

Category	Generic Causes of Uncertainties in Risk Assessment	
	Lack of Data	Lack of Understanding
Sources of Uncertainties	Inadequate or Inappropriate Data	Inability to Interpret Data
Associated Errors in Risk Assessment[a]	1) Estimation Errors - Statistical Error in Measurements, Model Parameters, etc.	1) Specification Errors - Mistakes in Functional Form of Models
	2) Omission Errors - Misidentification of Relevant Hazards or Causal Pathways	2) Extrapolation Errors - Misuse of Proxy Data from Analogous Contexts
Goal of Research	Improve Quantity and Quality of Available Data	Improve Scientific Understanding and Related Ability to Make Extrapolations and Predictions
Research Approach	Generate Relevant Data with Existing Methods and Models through Tests, Experiments, and Studies	Develop Mechanistically Based Methods and Models through an Iterative Process of Generating, Testing, and Refining Hypotheses
Examples	• Animal Testing for Carcinogenic Potential	• Biologically Relevant Methods to Characterize Toxicity
	• Analytical Epidemiologic Studies to Estimate Relative Risks	• Integrated (Total) Human Exposure Models
	• Monitoring Networks to Measure Environmental Concentrations	• Physiologically Based Pharmacokinetic Models
	• Clinical Studies to Estimate Dose-Response for Acute (Reversible) Effects	• Biologically Based Dose-Response Models

[a]Bogen, 1990



Concentrations

- Clinical Studies to Estimate Dose-Response for Acute (Reversible) Effects
- Biologically Based Dose-Response Models

[a]Bogen, 1990

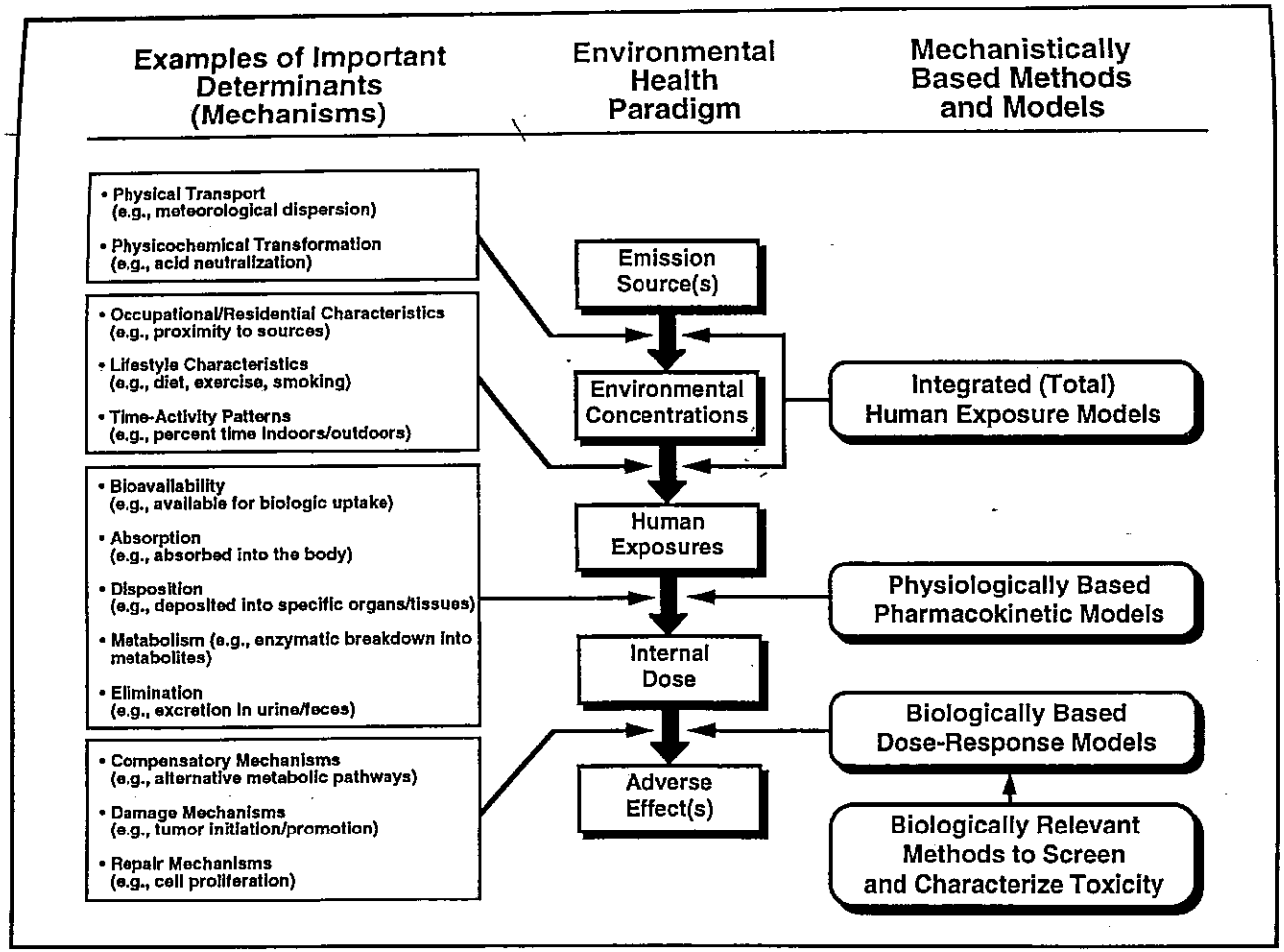


FIGURE 10. The relationship of mechanistically based methods and models to the environmental health paradigm and associated mechanisms.

Research to reduce extrapolation uncertainties must focus on developing and refining "mechanistically based methods and models," which attempt to describe the interrelationships among selected mechanisms, and to characterize and/or quantify their relation to the event of interest (e.g., exposure, dose, effect). Four complementary types of mechanistically based approaches are needed:

- Biologically relevant methods to screen and characterize the toxicity of environmental agents (directly relevant to improved hazard identification);
- Biologically based dose-response models to estimate quantitatively the adverse health effects from a given exposure or dose (directly relevant to improved dose-response assessment);
- Physiologically based pharmacokinetic models to estimate quantitatively the dose delivered to the target tissues (directly relevant to improved exposure and dose-response assessments);
- Integrated human exposure models to estimate the number of people exposed to specific levels of pollution (directly relevant to improved exposure assessment).

Overall, scientific uncertainties seriously hinder attempts to characterize risks for the general population and for population subgroups defined by socioeconomics, ethnicity, and/or race. It is essential to reduce these uncertainties by improving 1) the quantity and quality of data available, and 2) the capability to interpret these data. Environmental health research (e.g., epidemiology, exposure analysis, clinical studies, toxicology, disease surveillance, *in vitro* experiments) is necessary to build a strong scientific foundation for credible decisions about environmental justice (Figure 11). To be most effective, research should be coordinated to focus complementary approaches on answering the critical questions confronting risk assessors and risk managers.

SUMMARY

This paper emphasizes the following points:

- Attainment of environmental equity and justice is a top priority on the nation's agenda for safeguarding public health.
- Both socioeconomic class and ethnicity/race influence health status, although mechanisms of action are not well defined.
- Conceptual models provide a framework for generating testable hypotheses about the interrelationships among class, ethnicity/race, environmental factors, and health status.

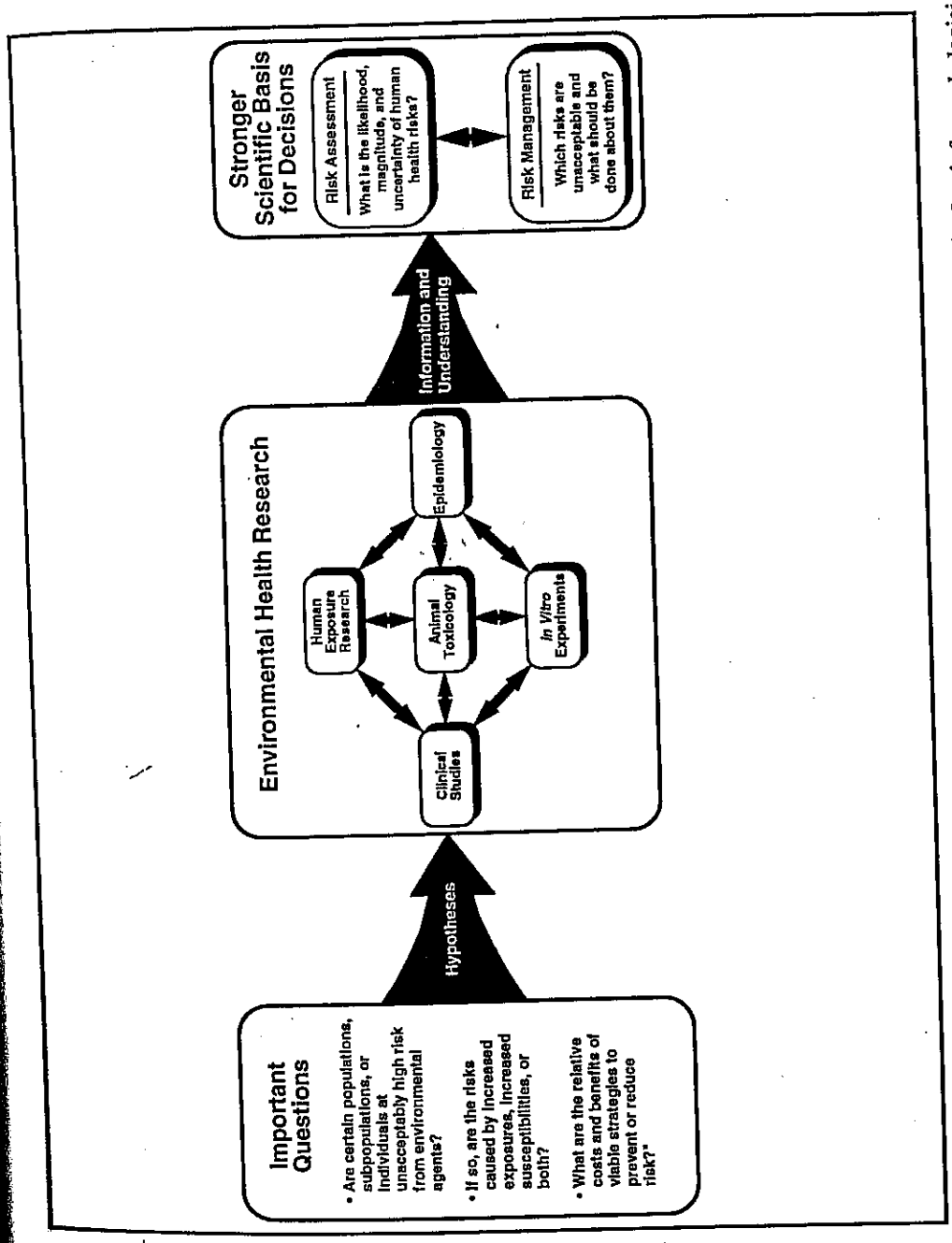


FIGURE 11. The central role of environmental health research in improving the scientific basis for informed decisions about environmental justice.

- Environmental health research is related to risk assessment and risk management through a feedback loop, which is at the core of improving the scientific basis for decisions about adequate and equitable protection for all citizens.

- Environmental justice issues are increasingly discussed in terms of "comparative risks," although the risk-based approach does not explicitly capture the inherent social, ethical, and moral dilemmas.

- If certain segments of the population, such as low-income and minority populations, are disproportionately represented in one or both of two categories (more exposed and/or more susceptible), then they are potentially at greater risk of environmentally related health effects.

- Research to investigate equity-related issues should be structured to generate, test, and modify hypotheses about causal relationships between class and race and environmental health risks.

- Research to reduce critical uncertainties in health risk assessment (i.e., lack of data and lack of understanding) is at the heart of efforts to evaluate and resolve questions of environmental justice.

CONCLUSIONS

Scientific data are currently insufficient to establish unequivocally the link between environmental health risks and variables such as socioeconomic status and ethnicity/race. Despite the absence of systematically collected information, there is strong presumptive evidence that economically disadvantaged communities, on average, experience higher exposures to many environmental agents than the general population. Exposure differences might cause disparities in environmentally induced health risks, even though the disparities are of unknown magnitude. This is cause for legitimate concern, and it justifies prudent actions aimed at improving environmental health for those potentially at higher risk.

The field of environmental health sciences historically has made substantial contributions to the protection of public health. In the current debate about environmental justice, research must play a central role if decisions are to be based on sound science. In the future, environmental health research will be successful in fostering attainment of environmental justice to the degree that it advances our ability to identify, evaluate, prevent, and/or reduce risks for all members of society.

Helpful comments
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