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## **Health and Environmental Justice**

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### **Abstract**

In the last ten years, research needs and scientific gaps have been discussed around the efforts of linking environmental exposure disparities to health disparities. This paper provides an argument that links environmental exposure disparities to health disparities. The public health approach is viewed as important in addressing environmental health disparities. Despite progress in addressing research needs and scientific gaps on linking environmental exposure disparities to health disparities, the problems remain very complex, the solutions to these problems are uncertain, and there is discussion around the costs versus the benefits of environmental exposure and its impact on the health of individuals.

### **Introduction**

Since the initial focus of national attention on environmental justice, the related scientific, social, and economic landscape has been dynamic and volatile. The magnitude, scope, and sophistication of environmental health sciences research are unprecedented and continue to increase dramatically. For instance, a growing community of researchers is studying the multiple dimensions of air pollution and respiratory health. The catalyst for these studies has been the identification of research priorities for airborne particulate matter by the National Academy of Sciences and the intensifying need to better understand the association between air quality and respiratory tract disease. Additionally, an increasing sophistication among the environmental justice advocacy community of academics, clergy, and grass roots participants has raised new concerns about adverse human health effects associated with the environment.

The increased attention to risk assessment has resulted in the process becoming more relevant to environmental justice issues. Increasingly, social and economic factors are included when describing risk. In addition, risk assessment methods have evolved to evaluate non-cancer risks to human health as well as risks to ecologic systems. The concern for reproductive, hepatic, neurological and behavioral risks and outcomes is mounting.

Clinically, advances in the treatment of lung disease have been significant. Such advances are undoubtedly due to successful research on the biology, biochemistry, and molecular biology of the lungs in health and disease. Despite concerns and advances such as these, the inclusion of environmental medicine into medical education and medical practice has been limited.

As our understanding of environmental determinants of disease and disability have increased, the complexity and utility of using morbidity and mortality data as metrics to evaluate differences in the distribution of environmental risk factors among population groups becomes more evident. Nonetheless, the establishment of a causal relationship between a health condition and the siting of an environmental hazard in proximity to low income or minority communities remains a complicated and debatable exercise.

In this paper, we discuss asthma as an example of the difficulty in relating a health outcome to disparities in exposure to environmental health hazards. The objective of this paper is not to present a rationale for abandoning current or future efforts to achieve environmental justice; rather, it serves to underscore the critical need for even better scientific knowledge to guide environmental justice efforts, and to ensure that related policies and programs effectively address environmental health disparities. As we strive to enhance environmental science capacity, the precautionary principle should be emphasized, "when an activity raises threats of harm to human health or the environment, precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically".<sup>1</sup>

The identification of key knowledge gaps should help shape the future environmental justice research agenda. As key

knowledge gaps are filled, our understanding of environmental components of disease (e.g. asthma, cancer, neurodegenerative disorders, etc.) will give decision and policy makers, and health professionals' additional useful insight. In consideration of the association between human health and the environment, one fundamental assumption is tacitly accepted even though this assumption has no universal assent. That assumption, which is central to this review, is that nothing in the history of the behavior of the public or private sector suggests that anthropogenic environmental "tampering" will discontinue.

Intellectual reinforcement for this assertion comes from numerous sources including a recent (2002) United Nation report: *North America's Environment: A Thirty-year State of the Environment and Policy Perspective*.<sup>2</sup> The study warns that the wasteful penchant for driving automobiles, owning big houses and living in sprawling suburbs appears to be gaining steam. According to the report, this lifestyle is eroding 30 years of environmental progress. Clearly, the health sciences community must hasten its inquiries into, and measurement of, the effects of such activities upon biological functions, in general, and disease, in particular, among populations at highest risk. Air pollution and asthma are used as one example of the interaction of the environment and human health.

### **Problem Identification**

Before discussing the issues of the study of air pollution and asthma, one must consider the current system for defining the scope and depth of the asthma problem. After all, the collection, analysis, and dissemination of data concerning asthma must drive the prevention policies and programs, including the assessment function, of public health. Unfortunately, there are few data on the prevalence of asthma at the state or local level, or in specific populations. Indeed, there is wide variability in the capacity of state and local health agencies to maintain and utilize surveillance information. For example, more than two-thirds of the U.S. utilizes surveillance for tracking lead poisoning, however, fewer than 25% maintain surveillance systems for other environmentally-related disorders such as asthma. The few data sets that exist are neither complete nor completely accurate. Each has problems and issues related to completeness, accuracy, and timeliness.<sup>3</sup>

To improve the data available on asthma thus providing a more comprehensive picture of the problem, and to allow for a more rational strategy for prevention and control of the disease, the Centers for Disease Control and Prevention (CDC) has taken some action. The CDC has added optimal asthma questions to the Behavioral Risk Factor Surveillance System (BRFSS). BRFSS is a state-based telephone survey of non-institutionalized U.S. adults, 18 years of age and older, that measures modifiable risk factors for chronic disease and other leading causes of death. The continued use of BRFSS asthma questions will enable state health departments to monitor trends in asthma prevalence and to better direct management efforts by providing prevalence rates for detailed demographic subgroups at the state level.<sup>4</sup> While this system has its merits, even more comprehensive tracking system is desirable for a better understanding of asthma and other environmentally related disease problems.

The best available data indicates that asthma is a serious and growing health concern in the U.S., which affects more than 5 million children under age 18. The U.S. Centers for Disease Preventive and Control found the asthma rate for 5 to 14 year olds increased 74 percent between 1980 and 1994, making it the third ranking cause of hospitalization among children under age 15. People of color and children living in inner cities are more likely to die of, or be incapacitated by the illness.<sup>5</sup> Urban asthma is frequently cited as an example of an outcome of higher exposure to environmental risk factors among people of color than non-Hispanic whites. People of color and inner city residents of all race/ethnic status may experience higher than average exposure to air pollutants, such as allergens, or irritants, which may initiate or exacerbate asthma.<sup>6,7</sup> This notion has been fueled by evidence that poorer communities are often located closer to sources of air pollution such as waste incinerators, superfund sites, diesel bus depots, and excess vehicular traffic.<sup>8</sup> For instance, in New York City, 6 of 19 bus depots are located in neighborhoods with predominantly African-American and Hispanic populations. In the United States, in the 1990s, 62.2% of blacks and 71.2% of Hispanics lived in non attainment (i.e., not meeting national air quality standards) areas, compared to 52.2% non-Hispanic whites.<sup>8</sup> These data suggest that nonwhites may experience a higher exposure to airborne environmental pollutants which may increase the risk of respiratory disease. However, the rise in asthma prevalence has also occurred in all ethnic groups and in both sexes.<sup>9</sup>

## Definition

The definition of asthma has challenged epidemiologists and other health scientists for many years. While a number of definitions have been proposed, the absence of a universally accepted definition of asthma makes it difficult to arrive at a consistent operational definition for asthma epidemiologic studies, and to design public health interventions. The definition riddle was examined by the Institute of Medicine's (IOM) Committee on the Assessment of Asthma and Indoor Air.<sup>10</sup> The Committee highlighted two important concerns with one commonly used definition of asthma: 1) *the definition implies that asthma is a single disease entity, although much of the contemporary evidence suggests that asthma is a syndrome caused by several different mechanisms;* and 2) *many interpret this definition to mean that asthma results from variations of the immune system leading to chronic inflammation.* Discovering the origin or origins of the inflammatory response, however, remains a critical unanswered question for researchers.

Another definition used in asthma epidemiology within the past year is "physician-diagnosed" asthma with wheeze. This term is imprecise since there is little information about the reasoning and consistency used by physicians when making this diagnosis. The IOM Committee defined asthma as a chronic disease of the airway characterized by inflammatory response involving many cells. According to the committee, *both genetic and environmental factors appear to play important roles in the initiation and continuation of the inflammation.*<sup>10</sup>

## Asthma Development

Within the last 10-15 years, it has become apparent that asthma is a chronic inflammatory disorder of the airways, with the extent of inflammation determined by environmental factors, endogenous factors, or both. Although several models have been proposed, there appears to be no consensus regarding the process by which asthma develops or exactly where in the process environmental factors may influence asthma development.

Understanding the mechanism by which an environmental agent induces asthma is important for several reasons.

Mechanistic insights will lead to a more rational assessment of human risk based on epidemiologic studies. Further, this information may suggest whether or not the associations between environmental factors and asthma are, in fact, biologically plausible. Geneticists recognize that there is no single gene for asthma. Not surprisingly, the study of genetics of asthma is complex and involves investigation of a multitude of factors. Although a difficult task, understanding gene-environment interactions is critical to understand diseases suspected of having an environmental association because each gene and environmental factor can contribute significantly to the overall risk. Depending on the genetic make-up of an individual, environmental factors are likely to be more or less influential, such as in the development of asthma. However, the study of gene-environmental interaction may be difficult, and will require large populations along with appropriate measures of environmental exposures. Predictions of future advances in understanding asthma include the identification and characterization of genes involved in the susceptibility to asthma. Also, it is predicted that asthma will be a prototype disease in which a DNA fingerprint will define susceptibility, thereby alerting individuals to avoid interaction with environmental stimuli that are specific for their asthma.<sup>11</sup>

In addition to considering environmental exposure as risk factors for the development of asthma, it is also important to consider environmental exposures that may be protective factors. Identical environmental exposures can be risk factors, protective factors, or not influential, depending on the host and his/her stage of development of asthma. Thus, investigations must consider all environmental exposures, not simply those considered adverse.

Many environmental factors have been proposed as having potential influence on the development of asthma. However, research is needed to evaluate the relative importance of these factors. This research should enhance the design of effective prevention/interventions. In the absence of this knowledge, a control policy might fail to reduce the incidence of asthma simply because the classes of environmental factors reduced are not the most important in influencing the development or the exacerbation of the disease. Unfortunately, this dilemma characterizes many of the public health challenges to prevent, diagnose, and treat environmentally related diseases and dysfunctions. Further, knowledge of the

process can also increase understanding of the person-to-person variation in risk for asthma.

A review by Gold<sup>12</sup> of important considerations of the process by which asthma develops underscores that the development of an allergy is not equivalent to the development of asthma. Allergic asthma likely requires localization of the allergic inflammatory response to the lung and also the development of bronchial hyperresponsiveness. Bronchial hyperresponsiveness has been described as excessive sensitivity of the airway to a variety of environmental agents<sup>12</sup>.

Distinct genetic and environmental factors may interact to influence the development of an allergy. As early as the 1920s, studies demonstrated that a familial predisposition for asthma existed, suggesting that genetics may play a role in the development of the syndromes.<sup>13</sup> This genetic influence seems to have remained constant in subsequent studies. For instance, recent research in the field of pharmacogenomics has shown that variations in the beta 2AR gene determine how asthmatic patients respond to certain medications.<sup>14</sup>

### **Linking Air Pollution and Asthma**

Information linking the exacerbation of asthma to air pollution exposure may come from a number of scientific approaches, including experimental animal studies, clinical exposure studies, and epidemiologic investigations. The most convincing evidence of the effects of air pollution on asthmatics is a well-conducted epidemiologic study in which a positive association is found between exposure to the relevant pollutants and disorder or disease. That obvious and substantial advantage is offset by inherent problems that leave room for uncertainty about study results. In environmental epidemiologic studies, an important issue is assessing exposure to sources of air pollution. The process is complicated by the numerous factors which may influence exposures. This may explain why little is known about variations in personal exposures across communities in relation to local sources of pollution such as power plant and incinerators. Factors that can influence personal air pollution exposure include proximity to local ambient sources, indoor penetration of outdoor air pollution, time-activity patterns and individual behaviors.

Assessing exposure to outdoor air in an effort to link environmental pollution to asthma requires information on concentrations to which people are exposed, as well as the frequency and duration of that exposure. Factors such as local meteorological conditions, pollutant volatility, and the time a resident spends proximate to sources (i.e., on the street) are significant determinants of personal exposure. Furthermore, the degree of air exchange from outdoors to indoors (i.e., ventilation) has a significant impact on the contribution of outdoor sources to exposure.<sup>15</sup> Sorting out or quantifying these various factors is a daunting task. But such assessment is critical because direct biological effect of a chemical (gaseous, or particulate) can only occur when the chemical causing the effect is present in the tissue of the organism where the toxic effects occur (i.e., the site of toxic action). Stated differently, this underlying toxicological concept implies that regardless of how hazardous a pollutant is – without exposure – there is not risk. In addition, the amount of chemical at the site of action in cells and tissues is proportional to the amount absorbed or inhaled. The amount inhaled depends on the concentration of the pollutant in the air to which an individual is exposed.

This principle illuminates the central role of a valid exposure assessment in epidemiologic studies which seeks to characterize populations at risk for air pollution-induced asthma exacerbation. Additionally, valid exposure assessments may indicate the distribution of exposure and enhance the identification of risks to susceptible populations - a long standing concern of environmental justice advocates.

Enmeshed within this exposure assessment quandary are questions regarding environmental justice. Are socioeconomic characteristics and ethnicity associated with an increased potential environmental exposure? If so, do they systematically result in higher actual exposure? The answers to these questions must consider some key concepts. Measurements in air represent “*potential*” exposure rather than “*actual*” exposure. Individuals residing in the community are likely to have significantly different exposures to air pollutants, or asthma triggers, depending on a number of factors (i.e., indoor air pollution sources, occupation and other activity patterns) which will ultimately characterize that individuals’ encounters with various sources of exposure. Therefore, although the potential for exposure may be the same for all persons residing within a given community, not all potentially exposed persons will have the same actual exposure.

Adding to this construct is the more recent aspect of air pollution research which suggests that the concepts of a regional air supply may be outdated, at least as it has been traditionally defined. Rather, a series of microclimates, differing neighborhood by neighborhood and street by street, are being increasingly recognized.<sup>16</sup> According to the microclimate concept, each individual carries around a personalized “cloud” of pollutants, depending on his/her environment, lifestyle and consumer product choices. In capsule, the pollutant burden - the inhaled dose - may be different for each individual, resulting in varying shapes of the air pollution exposure-response relationship.

In epidemiologic studies, the failure to accurately assign individual exposure leads to measurement errors. Measurement errors are one of the major sources of bias in epidemiological studies. Such errors can lead to spurious conclusions about the relationship between environmental exposure and asthma. Adding to this complexity are two classes of exposure measures that have been identified: 1) the theoretically ideal (typically unknown), risk-relevant exposure metric ( $E_{RR}$ ) that represents the individual breathing zone concentration of a pollutant relevant to asthma, and 2) the practical and available exposure surrogate that correlates to a greater or lesser extent with the  $E_{RR}$ . The  $E_{RR}$  will differ depending on the health outcome under consideration, for example, asthma development versus asthma exacerbation. An exposure surrogate now commonly used is any available measure of exposure that is positively correlated with the  $E_{RR}$ .<sup>6</sup> The  $E_{RR}$  is the theoretical measure of exposure that best represents the risk of asthma. Unfortunately, enough is not known about asthma process or mechanism to confidently identify the appropriate  $E_{RR}$ .

Over the years, a wide range of exposure surrogates have been developed. Direct exposure surrogates include personal monitoring, involving measurement of pollutant concentrations using monitors carried by individuals, and biological markers, involving the measurement of pollutants in biological samples such as urine or blood. Indirect measures include environmental area monitoring models (i.e., fixed-station samples) or microenvironmental models.

Direct exposure surrogates such as personal monitors offer promise for minimizing uncertainty about the degree of exposure, however, there are difficulties in using this monitoring technique. First, it is almost implausible to measure the personal exposure of a significant number of people at risk for asthma. There is also the problem of determining the appropriate duration of monitoring and obtaining a valid measurement without altering the individual’s behavior. Moreover, the availability and accuracy of personal monitors varies widely among pollutants. For some pollutants, the personal monitoring technology is available, but the device still requires improvement.

Various indices of air quality have been used, as indirect exposure surrogates. Consider the following examples:

1) An investigation of air pollution, specifically, motor vehicle emissions, and respiratory health used an exposure assessment model that represented a refinement over previous models. The model in this study accounts for emissions from all major streets. A limitation of the model is that data were not readily available to account for individual activity pattern in time and space.<sup>17</sup> Another limitation of this study was the lack of validation of the exposure model through spot checks of personal monitoring which would provide a close approximation of exposure. Without robust validation of the model, the exposure may be overestimated or underestimated. This study did not find an association between area exposure to motor vehicle emission and socioeconomic status, which might have been expected.

2) A study of the burden of air pollution on racial minorities in New York City used potential exposure data from several sources including the state’s daily monitoring system and measurements made by the researchers themselves. A major limitation of this study was the lack of pollution exposure estimates for each group (African American, non-Hispanic white, Hispanic) in the study.<sup>18</sup>

3) A study of asthma, hospital admissions and air quality (i.e. gaseous and particulate matter) conducted in Seattle, Washington used daily averages from four monitoring stations.<sup>19</sup> Results from this study revealed a significant association between air pollution and increased emergency room visits for asthma.

4) A study in Anchorage, Alaska used the daily average 8-hour maximum concentration of pollution during winter months.<sup>20</sup> A similar study in Reno, Nevada used the highest hourly maximum level of local air pollution recorded in a network of fixed monitoring systems.<sup>21</sup> Neither of these studies found a significant association between asthma visits to emergency departments and air pollution.

A major criticism of exposure assessments based on fixed-site ambient measurement is the poor correlation between personal exposure and ambient concentrations.<sup>22,23</sup> This criticism is valid, as the result could be a source of error in epidemiologic studies. Thus, if there is a causal relationship between air pollutants and asthma, there is no reason to believe that the exposure indicators or surrogates capture that causal relationship with precision. Moreover, the variability in the various indices of air quality has led to different conclusions drawn from the same data set. Also different investigators have not always used the same parameters or the same approach in analyzing exposure and related data. Therefore, differences in conclusions about air pollution and asthma, and other respiratory disorders, are to be expected.

## **Mixtures**

Another relatively complex issue is that of mixtures. For some observed respiratory effects, pollutants have been studied separately (i.e., particles, ozone). However, the most common types of exposures of these individual pollutants occur in the form of mixtures.<sup>24</sup> In fact, particulate matter (PM) in ambient air is a complex mixture containing particles of different sizes and chemical composition. There are also differences in the chemical composition of fractions of different sizes for eastern versus western U.S. For instance, in Los Angeles nitrates are a major contribution to PM. For some fractions of PM, minerals are a major contributor, especially in the western U.S.

While advances have been made in studies of the respiratory effects of single pollutants (i.e., PM), few advances in methods have resolved the problems of estimating exposure to complex mixtures. The methodological challenges have been cited in a number of papers.<sup>25,26</sup> The components of mixtures relevant to asthma may not be known. Therefore, the measurement of all component mixtures in the context of an epidemiologic investigation is not possible for most mixtures of concern. Marker components, referred to as tracers or proxies, have been used to represent exposures to mixtures. However, exposure measures of a single marker for a complex mixture may not reflect synergistic interactions among components, therefore the use of tracers or proxies is problematic. Volatile organic compounds (VOCs) illustrates this complexity. VOCs are complex mixtures of contaminants, resulting from many sources (e.g., industrial processes, consumer products, home and office furnishings). VOCs may trigger asthma as well as cause a wide range of adverse respiratory effects, as well as other health effects.<sup>27</sup>

Another example of a complex mixture to which many urban residents are exposed, is photochemical smog. This includes the primary pollutants of sulfur dioxide, nitrogen oxide and hydrocarbons, in addition to diverse reactive species produced by atmospheric reactions.<sup>15</sup> Identification and measurement of all the reaction by-products of the components of smog is difficult, if not impossible. The challenges in assessing exposures to link asthma to complex mixtures clearly underscores the difficulty in using asthma as evidence of the disproportionately higher prevalence of harmful environmental exposures.

## **Health Indices**

In addition to the problems of air quality indices, there are issues related to health indices. Epidemiologic studies have shown statistical associations of ambient air pollution with a variety of endpoints including mortality, hospital admissions for asthma, respiratory symptoms and lung mechanical functions. Of these end points, hospital admissions, or emergency room visits for asthma are most frequently used in epidemiologic investigations. As has been pointed out by the IOM Committee on Environmental Justice,<sup>28</sup> the use of these data (i.e., number of hospitalizations due to asthma) as a measure of the relative impact of environmental risk, such as air pollution, on those with low socioeconomic status is problematic. These indicators are strongly influenced by other factors such as lower rate of health insurance or lower levels of access to high quality primary care.<sup>28</sup> Moreover, the clinical diagnosis of asthma is sometimes complex and diagnostic methods and accuracy may vary among different population groups. In addition, the level of patient education and counseling regarding personal preventive measures varies among physicians. Some patients are exposed to more extensive physician counseling and education regarding asthma than others. For children with asthma, parental knowledge of asthma risk factors may influence the rate of asthma hospitalization or emergency room visits.<sup>28</sup>

## Comments

The establishment of a link between disparities in exposure to environmental agents, and disease and disability is a complicated and debatable exercise. The issues of asthma cited herein, including defining the problem, the process by which the syndrome develops, and an accurate assessment of exposure, are selected examples of the challenges in determining the link between environmental exposure and health status. Sorting out these types of issues in the broader environmental justice context are especially important in evaluating the validity and strength of inferences or conclusions that disparities in exposure are responsible for disparities in health.

Over the past decade, much has been written about the research needs, challenges, and the potential contribution of the public health approach to environment justice. These writings have recognized that environmentally-induced diseases are complex.<sup>29</sup> The many variables that determine the development and severity of adverse health effects of environmental exposure defy simple summary. However, they do suggest that in the 21<sup>st</sup> century, the environmental justice movement must view health disparities through a different prism. Currently, the most common diseases are multifactorial, hence they cannot be ascribed to a single gene or to a single environmental factor. Instead, these conditions develop from the combined action of many genes, environmental factors, and risk-conferring behavior, which may be influenced by socioeconomic constructs. This matrix should not suggest that the solutions will come only from large and expensive interventions. Mechanistic or biological insights may lead to simple interventions or prevention strategies, as has been the case with a number of complex diseases.

Clearly, a better understanding of exposure would provide much needed insight into the relationship between exposure as a consequence of race/ethnic and/or socioeconomic status and disease. Nonetheless, we must acknowledge that maximum exposures to environmental pollution in the U.S. have been regulated and, consequently, reduced. For instance, the Clean Air Act has resulted in significant progress in control over emissions of the major combustion-related air pollutants.<sup>30</sup> A recent CDC analysis found that enforcement of automobile emission standards set by the 1970 Clean Air Act may explain the decline in the unintentional carbon monoxide (CO) related death.<sup>31</sup> Indeed, the introduction of the catalytic converter for automobile use in the United States in 1975 improved the completeness of engine combustion and reduced CO emission.<sup>32</sup> Furthermore, the Internal Revenue Service has now approved "*clean fuel automobiles*" for which individual owners can receive a \$2,000 tax deduction. This is in compliance with a 1993 law offering tax deductions for "*clean-fuel vehicles*." The law was largely unused until August 2002 when the government certified the first models for low emission tax deduction. Further reduction in potential exposure to auto emission can be anticipated.

Stationary sources of pollution have also been the focus of regulations. In 2000, the EPA issued final regulations that will subject several hundred facilities to a new regime of air pollution standards. Estimates are that new rules will cut emissions of mercury by 55%, particulate by 42%, cadmium and lead by 88%, and chromium by 75%.<sup>33</sup>

The transition to unleaded gasoline in automobiles is responsible for the 98 percent decline in lead emissions since the 1970s.<sup>34</sup> But much remains to be done. One of the challenges facing the public health community and its allies is to sort out how multiple factors of disease interact in ways that translate into effective policy, programs, and services for prevention. Thus, the compelling need for the Nationwide Health Tracking Network, first proposed as a federal project by the Pew Charitable Trust's Environmental Health Commission.<sup>35</sup> This proposition should be vigorously supported with appropriate resources. The network will help to expand knowledge regarding suspected links between non-infectious diseases and environmental pollutants. Its principal component is a national and state tracking of chronic disease and environmental exposure. This system would be complemented by appropriate laboratories and related resources. An important element in this network will be the seamless collaboration among clinicians, epidemiologists, geneticists, toxicologists and numerous other specialists in the environmental health sciences, as well as in the social and behavioral sciences.

A substantial challenge for this group will be to correctly interpret the outcome of genetic testing, human clinical exposure studies, field exposure assessment studies, and epidemiologic investigations. Also, environmental health researchers will need to have a focus that is different from that of scientists who study genes that lead to disease. Instead, the focus of environmental health scientists should be on variations in genes that, in and of themselves may be harmless,

but, when coupled with a given environmental exposure can amplify one's risk of disease and disability.

These activities may be aided by efforts of EPA's newly (1999) established Office of Environmental Information. The office is in the process of integrating the Agency's different information systems on air, water, land, waste, and pesticides, so that "*people can make the right decision for the best environmental results*".<sup>36</sup> Also of potential importance is the EPA's 2002 policy on use of genomic data in risk assessments, including the use of genomics data to rank chemicals for further testing. Genomics data could have a significant impact on the assessment of risk from exposure to environmental pollutants.<sup>37</sup> This is another example of an area in which more work must be done.

Another challenge which pervades most environmental justice debates is to overcome the tensions that often exists due to differing, or unclear goals of community advocates and research scientists.

Community groups are usually in the position of advocating policy changes. Scientists are often uncomfortable working in such settings because of concerns about the impact on the scientific process. For example, media attention might affect the ability of a scientist to publish data in peer-reviewed journals where it can benefit a larger segment of the population and achieve recognition by the scientific community. Unfortunately, current training programs, including those in the health sciences and medical schools, do not adequately teach students how to deal with these challenges and opportunities.

### **Demographic Shifts**

Finally, the "*demographic angle*" of the new prism through which environmental health disparities should be viewed cannot be overlooked. Prevention of disease and the identification of risk factors requires something more than knowledge of specific etiologic factors of disease. Of equal importance is a knowledge of the people in the community, their concerns and demands, and their organization and movements.

Defined in the initial 2000 census data and elaborated on by demographic analysts, new population patterns of dispersion are emerging. More than half of Latinos, part of the initial "*environmental justice cohort*", now live in the suburbs, and many migrants who have low socioeconomic status are skipping city centers ("*brownfields*") and heading for jobs and housing in outlying areas ("*greenfields*"). In general, cities with the largest most well-established Latino population experienced the lowest rate of growth in the late 1990s and early 2000. At the same time, newer hubs grew by more 200 percent or more. It is predicted that this trend will continue.<sup>38,39</sup>

Another demographic shift is the wave of newer, mostly non-Hispanic white, residents of high socioeconomic status who are settling into the pricier apartment buildings, the renovated row houses, and stately homes which are replacing previously substandard neighborhoods in urban centers.<sup>40</sup> These "*new-comers*" are giving increased attention to the enforcement of zoning regulations and the application of other resources for environmental improvement – to the benefit of both affluent and remaining less affluent residents in urban centers.

Further scrambling the "environmental justice landscape" is the producing groups of middle-class African Americans who are returning to the poorest, and most beleaguered areas of the inner city (i.e., Hill District of Pittsburgh, PA, Harlem, New York, parts of Washington, DC and Chicago). These neighborhoods are undergoing residential shifts that some sociologists refer to as "*black gentrification*".<sup>41</sup>

Of concern to public health are data from the 2000 census and the CDC which show an increase in the rate of babies born with low birth weight in suburban America, a risk factor for infant mortality typically observed in inner city neighborhoods. For instance, in New York City, the rate of low birth weight dropped from 9.3 percent in 1990 to 8.5 percent within the ten year period. Conversely, the rate of babies born with low birth rate rose from 5.9 percent in 1990 to 7.1 percent.<sup>43</sup> It has been suggested that population shifts, along with increasing multiple births, may both contribute to the higher rate of low birth weight babies. Clearly, these issues require further study.

## Summary

Over the past decade, the concepts and goals of environmental justice have been the focus of much attention. As a result of this focused attention, a list of research needs and scientific data gaps have been articulated, particularly related to efforts to link environmental exposure disparities to health disparities.

Research needs and scientific data gaps continue to fuel research activities and improvements in research methods. The complexity of translating research results into policy and programs designed to reduce the risk of environmentally-related disease is also illuminated. Increasingly, the public health approach is viewed as key in dealing with environmental health disparities. Progress is being made to reduce human exposure to environmental pollution - a development that will benefit individuals and communities at highest risk of environmentally mediated disease.

Despite this progress, important environmental problems continue to exist. Moreover, the remaining problems are highly complex. The solutions are uncertain, involve a complicated mix of traditional sciences, and the weighing of costs versus benefits. Questions about what constitutes an acceptable level of risk and how to balance health- and technology-based environmental standards remain particularly vexing.

Finally, equally relevant are demographic shifts along with the effects of global economic integration on income, society, the environment and health. These developments call for a broad discussion of a 21<sup>st</sup> century agenda and priorities for the environmental justice movement including an assessment of policy and programmatic ramifications, as well as the appropriate response and support from government, academia, the private sector, and the general public at large.

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