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AMBIENT AIR POLLUTION EXPOSURE AND THE INCIDENCE OF RELATED  
HEALTH EFFECTS AMONG RACIAL/ETHNIC MINORITIES

Running head: Air Pollution Exposure and Related Health Effects

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Differences among racial and ethnic groups in morbidity and mortality rates for diseases, including diseases with environmental causes, have been extensively documented.<sup>1,2</sup> In general, U.S. minority population groups have substantially elevated rates of certain respiratory diseases and cancers, which have been causally linked to air pollutants. Minority groups also have elevated rates of lead poisoning, which is linked both to household environmental contamination and to air pollution. Differences in rates of environmentally-induced diseases among population subgroups may be due to differences in either susceptibility, or pollutant exposures, or both. Analyses have shown that the elevated age-adjusted mortality rates among racial/ethnic minorities are primarily due to living conditions and social factors, such as income, rather than genetic susceptibility.<sup>1,3</sup> However, documenting the linkages between environmental contaminants, individual exposures, and disease incidence has been hindered by difficulties in measuring exposure for the population in general<sup>4</sup> and for minority populations in particular.

After briefly discussing research findings on associations of common air pollutants with disease incidence, we summarize recent studies of racial/ethnic subgroup differences in incidence of these diseases in the United States. We then present evidence of both historic and current patterns of disproportionate minority group exposure to air pollution as measured by residence in areas where ambient air quality standards are violated. The current indications of disproportionate potential exposures of minority and low-income populations to air pollutants represent the continuation of an historical trend. The evidence of linkage between disproportionate exposure to air pollution of racial/ethnic minorities and low-income groups and their higher rates of some air pollution-related diseases is largely circumstantial,

but extensive and consistent. To the degree that pollution exposures contribute to excess disease rates, a portion of the disease burden of minority groups is preventable. Reducing these disease rates may have substantial social benefits since it is likely that this disease burden decreases productivity and income for the affected groups, and increases health care costs.

## **DIFFERENTIAL RATES OF AIR POLLUTION-RELATED HEALTH EFFECTS**

Air pollutants can cause or contribute to both acute and chronic health effects that result in early mortality, or in impaired physical or mental development or function. Many studies have sought to identify relationships between exposures to specific air pollutants and particular health effects, such as respiratory illnesses (especially asthma), cancer mortality, and lead poisoning. We include lead poisoning in this group because airborne lead is still a source of exposure (in spite of its reduced concentration in gasoline) and it affects adults as well as children. The causes of these diseases are complex and their development is apparently influenced by characteristics of exposed individuals such as nutritional status and age. Figure 1 shows major factors, including environmental pollutants, that have been found to influence morbidity and mortality rates. The complexity and interrelatedness of some of these factors makes specification of health effect "causes" extremely difficult.

Subgroups of the population differ in their susceptibility to effects of pollutant exposure. For instance, young children are especially susceptible to some effects of pollutant exposure. Children have a higher asthma incidence rate and are generally more severely affected by intake of toxic substances in the environment, such as lead and mercury. The

effects of poverty can also exacerbate the effects of some air pollutants, through both undernutrition and inadequate medical care. The health effect susceptibility of individuals also increases with the presence of chronic diseases like emphysema and with the normal effects of aging. In addition, behaviors such as smoking can increase the likelihood of environmental pollution-related health effects due to apparent synergistic action of disease inducing agents in tobacco smoke and the environment. Thus, the fact that minority populations are younger, poorer, less healthy, and more likely to smoke cigarettes than the majority increases their relative risk of health effects.

Identifying disease causes is also complicated by the complexities of air pollutant chemistry. For instance, particulate matter, one of the criteria pollutants<sup>1</sup> considered in measures of air quality, has toxic constituents. Particulates are a category that is defined by particle size rather than chemical composition and can include metals or organic compounds. Evans, et al.<sup>6</sup> critically reviewed the extensive literature on air pollution and mortality, and, while cautious about the problems in determining causality presented by confounding factors, concluded that the studies "reflect a causal relationship between exposure to airborne particles and premature mortality."

In spite of the complexity of the research issues, there is a substantial body of evidence tying ambient air pollution exposures to increased rates of several types of health effects in the general population. (For broad reviews of the literature related to respiratory and other air pollution effects see Evans et al.<sup>6</sup> and Thurston et al.;<sup>7</sup> for lead poisoning, see

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<sup>1</sup> The criteria pollutants regulated by the Clean Air Act<sup>5</sup> and its 1990 Amendments are lead, carbon monoxide, nitrogen oxides, sulfur dioxide, particulate matter and ozone.

Muchak et al.<sup>8</sup> and Muchak<sup>9</sup>.) Differences in disease incidence and mortality rates among racial/ethnic groups in the United States are discussed below for respiratory diseases, cancers, and lead poisoning. Table 1 lists the pollution-related diseases for which there are substantial differences in incidence or mortality rates, the direction of the difference, the measure used, and the study or information source. These studies provide the basis for our discussion of disease rate differentials.

### Respiratory Disease

Chronic exposures to sulfates, particulates, ozone, and nitrogen oxides have been causally linked to various forms of respiratory system impairment. Both ozone and nitrogen oxides have been implicated in the development of emphysema<sup>17</sup> and nitrogen oxides have also been found to impair lung defenses against influenza viruses.<sup>18</sup> Exposure to sulfur dioxide has been found to stimulate asthmatic reactions and the admixture of ozone or particulates may exacerbate the effect.<sup>19</sup> Acidic aerosols derived from sulfur dioxide are also implicated in the development of both asthma and chronic bronchitis.<sup>18</sup> In addition, carbon monoxide impairs oxygen transport<sup>20</sup> such that acute exposures cause early mortality in the portion of the population with pre-existing respiratory system disease.

Chronic obstructive pulmonary disease is the major category under which bronchitis, emphysema, and asthma are recorded in the mortality statistics. Age-adjusted mortality rates for Hispanics, African-, Asian-, and Native-Americans from chronic obstructive pulmonary disease are substantially lower than those of Whites.<sup>3</sup> However, within this broad disease category, mortality rates for asthma are substantially higher for African-Americans than for

Whites.<sup>2</sup> Elevated rates of asthma incidence among African-Americans have received the most research attention, but there are also elevated rates of other respiratory diseases among minority populations. For instance, age-adjusted death rates for pneumonia and influenza are substantially higher for African- and Native-Americans.<sup>3</sup>

For asthma, the highest incidence rates are found among Puerto Ricans and African-Americans.<sup>3</sup> During the 1980s, the rates of hospitalization and of mortality from asthma increased for the population as a whole and increased at an even faster rate for African-Americans. The mortality rate for Blacks rose from twice that of Whites in 1979<sup>21</sup> to more than triple it by 1988.<sup>22</sup>

Weiss and Wagener<sup>10</sup> examined the trend to identify the underlying factors and the population subgroups that are most affected. Their analysis indicated that the increase in mortality was not merely a reflection of changes in diagnosis. During the 1980s, asthma mortality overall increased by 6.2% per year, with the fastest increase being among children five to 14 years old. The rate of increase was slightly higher for Whites than for nonwhites, but the asthma mortality rate for nonwhite children and young adults remains approximately four times that for Whites. African-American males are the group at highest risk. About 80% of the asthma-related deaths occurred in urban areas, and there are four areas where the mortality rate has consistently remained high: New York City; Cook County, IL; Maricopa County, AZ; and Fresno County, CA. Weiss and Wagener conclude that the mortality rates in New York City and Cook County are essentially driving the national trend.

In a similar study, Gergen and Weiss<sup>11</sup> analyzed the National Hospital Discharge Survey for 1979 to 1987 and found that the hospitalization rate for 0 to 17 year olds

increased from 1.73 per 1000 population to 2.57. In a comparison of rates for population subgroups, only the rates of increase for 0 to 4 year olds and for African-Americans of all ages were statistically significant. Treatment protocols indicate that these hospitalizations also represent an increase in severity of symptoms. The authors suggest that fine particulate pollution and poverty are reasons for the increase in hospitalization rates for young African-American children.

In an attempt to identify asthma risk factors, Weitzman et al.<sup>23</sup> examined the effect of maternal smoking, race, sex, residential location, etc. Significantly higher risks were associated with being African-American and male, and with maternal smoking. A constellation of factors that are commonly associated with poverty were also implicated in increased incidence and were found to account for the racial difference in asthma incidence rates. Thus, differential poverty rates, rather than genetic factors, appear to play a major role in the elevated asthma incidence rate of African-American children.

Overall, evidence regarding differential rates of air pollution-related, respiratory disease among racial/ethnic groups is mixed. Mortality rates for chronic obstructive pulmonary disease are higher for Whites than for any of the major minority populations. However, within this broad disease category, higher age-adjusted mortality rates are found for African-Americans and Puerto Ricans from asthma, and for African- and Native-Americans from pneumonia and influenza. Air pollution exposure has been implicated in the asthma rate differential, but differences in the other disease rates are largely unexplained.

## Cancers

The U.S Environmental Protection Agency (EPA) has estimated that exposure to toxic substances in ambient air causes 1700 to 2700 cancer deaths per year.<sup>24</sup> (This is out of a total of about a half-million cancer deaths per year.) The EPA reviewed studies covering 90 types of toxic air pollutants to ascertain the magnitude of cancer risks associated with each pollutant and the uncertainties in the risk estimation. The major contributors to the cancer mortality rate are: products of incomplete combustion; 1,3-butadiene; hexavalent chromium; benzene; formaldehyde; and chloroform. Motor vehicles accounted for almost 60% of the emissions linked to cancer fatalities, while point sources, such as refineries and dry cleaners accounted for 25%. Because of the nature of the sources of these emissions, the study noted that risks may be relatively higher for individuals in urban areas or living adjacent to point sources of some pollutants. National data from which to estimate population exposures to ambient concentrations of specific airborne toxic chemicals are unavailable. However, these chemicals are released, in varying proportions, by the same processes that result in ambient concentrations of the criteria pollutants.

Cancer mortality rates for Whites and minorities differ substantially for specific cancers that may be directly related to air pollution exposures, as well as for all types of cancers combined. There is considerable geographic variation in mortality rates, with higher rates occurring in the Northeast and the Louisiana Delta area.<sup>25</sup> The age-adjusted rates shown in Table 2 for Whites, all minorities, and African-Americans indicate substantial excess mortality for minorities, especially for African-Americans. In fact, age-adjusted statistics for a number of cancer types indicate that African-Americans have the highest

overall rates for both cancer incidence and mortality of all race/ethnic groups.<sup>12</sup> Whether these differences are due to higher cancer incidence rates in these populations or represent premature mortality due to less effective treatment is unclear. However, it has been shown that African-Americans have a lower survival rate for 22 out of 25 cancer sites (e.g. lung, bone, etc.).<sup>12</sup> Detailed demographic studies have found that socioeconomic factors account for much of the racial differential in rates.<sup>1,3</sup>

A detailed study of cancer incidence in children found that African-American children had lower incidence rates of several types of cancer (almost half the rate of leukemia for Whites), but had a much lower survival rate.<sup>13</sup> Several studies have shown that children's cancer survival rates, for all races, decline with income. While these studies provide information on the role of genetic and treatment factors in morbidity and mortality, they do not address the issue of environmental exposures. The long latency periods of many cancers and difficulty in documenting exposures make identifying the cause of any specific case of cancer nearly impossible.

Population studies, however, have shown strong associations between some types of air pollution and certain cancers. An early study by Hoover and Fraumeni<sup>14</sup> analyzed cancer mortality rates geographically and correlated those rates with demographic characteristics. Using data covering the 1960s and focusing on the counties in which chemical industries were most highly concentrated, they found elevated bladder, lung, liver, and other cancer rates for males. They attributed the elevated cancer rates more to smoking behavior than to socioeconomic class, urbanization, or industrial factors. However, minorities exhibited a ten

percent higher mortality rate than the White population in those counties. This relationship held for total cancers and for cancers of the lung and bladder.

In summary, the proportion of cancer mortality attributed by the EPA to air pollution exposures is relatively small, less than 1% of the total. For cancers, however, the differences in mortality rates among population subgroups are quite pronounced. The rates for minorities in general, and African-Americans in particular, are substantially higher for all types of cancers combined, and for cancers that are often associated with toxic exposures, such as lung and gastrointestinal cancers. Whether these differences in mortality are due to primarily to differences in incidence or to differences in treatment is unclear.

### **Lead poisoning**

Of the air and environmental pollutants, the effects of lead are probably the best documented (for a comprehensive review, see Mushak<sup>25</sup>). Lead is absorbed through inhalation and, especially in children, by ingestion. Lead intake in excess of the amount that the body can eliminate on a daily basis is primarily deposited in the bones and teeth, from which it is released on a continuing basis. Once absorbed, it can damage the nervous system, gastrointestinal tract, kidneys, formation of blood components, and reproduction.<sup>26</sup> Aside from exposure due to residence near a point source of lead emissions, most exposures are due to the former use of lead in paints and as a gasoline additive. Though lead has been reduced to trace levels in these products as currently sold, lead dust from both sources has accumulated in both houses and soils, particularly in older and heavily traveled urban areas, where it is subject to resuspension. There has been a public health payoff from the

regulation of lead content in products, however. Recent analysis has shown dramatic decreases since the mid-1970s in blood-lead levels among all segments of the population.<sup>16</sup>

Clinical studies have shown that even very low levels of lead in blood are associated with adverse effects, such as subtle "disturbances in cognition, behavior, and attention".<sup>15</sup> Prenatal exposures are related to delays in cognitive development, and, in older children, lead exposure has been related to diminished IQ test scores. Elevated blood lead levels (up to 35  $\mu\text{g}/\text{dL}$ ) are associated with declines as large as 15 points in IQ scores in some individuals. The mean IQ decrease in children with high blood-lead levels is about five points.<sup>27</sup> An analysis by Needleman and Gatsonis<sup>28</sup> of 12 studies linking IQ to lead exposure found consistently lower scores as lead concentrations in blood or teeth increased. This effect was statistically robust across studies in spite of relatively low levels of exposure. There is considerable evidence that the neurological effects are long-lasting,<sup>8,29</sup> though the degree of impairment may decline as blood lead levels decrease over time.<sup>30</sup>

In adults, high levels of lead exposure have been associated with the development of cerebrovascular and kidney disease and with hypertension. Recently, low levels of lead exposure (<3  $\mu\text{g}/\text{dl}$  blood) have been found to correlate significantly with blood pressure.<sup>31</sup> Groups with high diastolic blood pressure (>90 mm Hg) had significantly higher blood-lead levels. Higher blood-lead levels were found in older age groups and among African-Americans. When factors such as age and race were statistically controlled, Harlan found that the relationship of lead levels to blood pressure was significant for males but not females.<sup>31</sup>

Relative to White children (and Mexican-American children in the Southwest) Black children have a considerably higher incidence of blood-lead levels in the range of clinical concern. Table 3 shows percentages of children by racial group with lead levels exceeding 10  $\mu\text{g}/\text{dL}$  of blood and the changes over the period from 1976 through 1991. Though the percentages of persons with elevated lead levels have decreased substantially over the time period, they are higher for lower income groups, and the percentage of African-American children affected is the highest for any category at 20%. Recent epidemiological studies have shown that permanent damage occurs even at relatively low lead levels, so 10  $\mu\text{g}/\text{dL}$  of blood is now defined as a toxic level of lead. Needleman<sup>15</sup> estimates that 17% of all children and over 50% of poor black children enter school with blood-lead levels over 10  $\mu\text{g}/\text{dL}$ . Poor and minority children are at greater risk for exposure because they are more likely to live in deteriorating housing that contains lead paint and in urban areas with high levels of lead in soils. Concentrations of lead in soils are highest in the inner-city areas of large cities and the pattern of lead concentration in soils along older thoroughfares is an indication of substantial lead deposition from gasoline combustion products.<sup>32</sup>

Differential rates of lead poisoning have been clearly documented, with higher rates among African-American children in all income groups, and among low-income children in general. African-American adults have also been found to have elevated blood-lead levels and, in males, this has been related to hypertension.

## **DIFFERENTIAL EXPOSURE OF MINORITIES TO AIR POLLUTION**

Given the research linking diseases to air pollution exposure and the indications that

minority populations experience some of these diseases at higher rates, we examine indications of potential exposure to pollutants that may explain the disease rate differentials. We use National Ambient Air Quality Standard compliance (or lack of it) in the county of residence in 1990 as an indicator of air pollution exposure. Areas with substandard air quality are, generally, either urban or adjacent to point sources of pollution. The main exception to this is for particulates, which may be high in desert or farming areas as well. It is the location of people relative to these substandard air quality areas that primarily determines potential exposure. To the degree that racial/ethnic minorities are concentrated in urban areas, their potential exposure to air pollution may be increased. Likewise, the potential exposure of minorities will be higher if they are concentrated in regions with more severely affected air quality. We first present an overview of studies related to air pollution exposure and then provide a summary of current differences in minority and majority population exposures based on the degree to which minority population concentrations coincide geographically with substandard air quality.

### **Historical Evidence**

Evidence on the distribution of air pollution exposure among population subgroups has been accumulating since the early 1970s. Many empirical studies have investigated the relationships between air pollutant concentrations and property values, and some of these studies have examined differential impacts on disadvantaged or minority populations. A few representative examples are summarized here.

The distributions of sulfur dioxide, particulates, and carbon monoxide within the 31-county New York City region were analyzed by Zupan,<sup>33</sup> who found that low-income groups were substantially less likely to reside in areas with low concentrations of each pollutant than were high income groups. However, both high- and low-income groups were exposed to high pollutant concentrations in some areas of the city. The differences in exposure of high- and low-income groups were more pronounced over the whole suburban area than they were in the city itself.

In a study designed to address equity issues, Freeman<sup>34</sup> investigated the distribution of sulfate and particulate exposures among various income and racial groups in Kansas City, St. Louis, and Washington, D.C. He found that air pollution levels within areas of each city decreased as the income of residents increased, but there were substantial differences in pollution levels between cities. In each of the cities, the average Black family was exposed to higher pollution levels than White families in the lowest income category.

In a 1975 study in Washington, DC, Kruvant<sup>35</sup> found that individuals who are poor, of low occupational status, residents of low-rent areas, and Black are mainly clustered in urbanized areas and are more likely to be heavily exposed to "common air pollution." Relating family income with ambient carbon monoxide levels, Kruvant found that 85% of families with median incomes greater than \$20,000 in 1970 lived in areas where levels averaged  $< 2 \text{ mg/m}^3$  (below the federal air quality standard). In contrast, only 17% of families with median incomes less than \$7000 lived in  $< 2 \text{ mg/m}^3$  areas. For the  $< \$7000$  income category, 23% lived in areas averaging 5-9.99  $\text{mg/m}^3$ , and 13% lived in areas that exceeded the federal standard of 10  $\text{mg/m}^3$  for carbon monoxide. Even more dramatic

exposures to sulfur dioxide and particulate concentrations above the federal standards were found for the <\$7000 group; 63% and 80%, respectively, resided in areas exceeding the standards.

Asch and Seneca<sup>36</sup> employed pollutant data for 284 cities in 23 states to investigate both inter- and intra-city variation in population exposures. They examined the relationships at the state level between the average particulate levels and income, poverty status, education, home value, rent, population density, race, and age. At this extremely aggregate level of analysis, they found a strong, negative relationship between income level and the average concentration of particulates, i.e. the poorer the state, the higher the pollution level. The nonwhite population percentage was positively associated with particulate levels, but not as strongly. To determine if similar relationships hold within cities as well, the authors matched air monitor data for three pollutants with demographic data for the census tracts containing the monitors in Chicago, Cleveland, and Nashville. Pollution levels had a strong, inverse relationship with income levels within each city, but the relationship with race varied across cities and pollutants.

In a national study, Gelobter<sup>37</sup> analyzed data for particulates as well as for a combination of pollutants, with results similar to those of Freeman<sup>32</sup> and Zupan.<sup>31</sup> Gelobter found that, based on 1970 to 1984 data, there were considerable differences in exposures to certain air pollutants among subgroups. Over that period, minorities had greater exposures than Whites within urban areas, as well as for the nation as a whole.

A detailed exposure model incorporating age and activities was used by Brajer and Hall<sup>38</sup> to examine the distribution of ozone and particulate exposure among population groups

in the Los Angeles metropolitan area. Detailed modelling of daily activities showed that population movements among areas of the city for work and recreational activity lessen differences in estimated exposures that are based solely on residential patterns. For instance, a substantial number of suburban residents may have pollutant exposures due to commuting to and working in the central city. Correlations between median income levels and concentrations of ozone and particulates were negative but weak. Exposures of children were significantly higher than those of other age groups for both pollutants. Particulate exposure rates were significantly higher for Hispanics and African-Americans while those for ozone were elevated but nonsignificant.

### Current Trends

Demographic data are combined with data on areas in violation of National Ambient Air Quality Standards to examine minority-majority population differences in potential exposure to air pollution. The Clean Air Act<sup>5</sup> sets Ambient Air Quality Standards for six pollutants: carbon monoxide, sulfur dioxide, ozone, fine particulates, lead, and nitrogen oxides. (Nitrogen oxides are omitted from this analysis because virtually all counties met the standard in 1990.) EPA data for areas in violation of the Standards were used to identify counties that were wholly or partly in non-attainment status in 1990. We recognize that counties in most regions of the country are too large to represent homogeneous geographic distributions of most types of air pollution. This data limitation will likely result in underestimating differences between the percentages of minority and non-minority populations exposed to air pollution. This is likely because previous analyses on the sub-

county level<sup>34,36</sup> have shown minorities to be disproportionately concentrated in sub-county areas with greater air pollution. In addition, it is important to note that living in an EPA-designated air quality non-attainment area does not necessarily mean that one is exposed to harmful levels of air pollution. As Sexton and Ryan<sup>4</sup> point out, a variety of other factors, such as workplace location, commuting patterns, and leisure time activities, affect exposure levels. Nevertheless, one can reasonably expect that, generally, individuals residing in non-attainment areas have a higher probability of exposure to air pollutants than those residing outside of such areas.

Demographic data for White, African-American, Hispanic, Asian-American, Native-American, and "Other Race" populations are from the 1990 Census.<sup>39</sup> These population categories are not all mutually exclusive, in that members of the Hispanic ethnic group may be of any race. Table 4 presents the percentages of each group living in non-attainment counties (excluding Alaska and Hawaii), by pollutant. Of the racial groups, relatively high percentages of Asian-American and Other Race populations live in carbon monoxide and ozone non-attainment areas; this is also true for Hispanics. In contrast, the percentages of Native-Americans living in the various types of non-attainment areas are generally quite low. The African-American population percentages in non-attainment counties generally fall between the lower percentages of Whites and the higher ones of Asian, Other Race, and Hispanic-Americans. The exception to this pattern is for sulfur dioxide, where the African-American percentage is the highest of all racial or ethnic groups.

Comparing the minority group percentages with those for Whites shows that, in general, higher percentages of the total African-, Hispanic-, and Asian-American populations

reside in non-attainment areas, relative to Whites. The exception is sulfur dioxide, for which the White percentage slightly exceeds those for each of the other population groups except African-Americans. The percentages of Whites exposed are also greater than the percentages of Native-Americans living in carbon monoxide, ozone, and sulfur dioxide non-attainment areas. This is largely due to the location of major portions of the Native-American population outside of urban areas. In all other cases, the minority population percentages are greater than or equal to those for Whites. The proportion of the minority groups exposed is ten percentage points higher than for Whites in over half of the cases where minority exposure is higher.

The percentages in Table 4 cannot be summed by racial/ethnic group, since some counties are designated non-attainment for more than one pollutant. Such multiple-pollutant non-attainment is of interest and potential health significance. Exposure to multiple pollutants may be an important factor in health effect incidence if the pollutants have interactive or cumulative effects. This could occur, for instance, when two pollutants affect the same organ(s), or when exposure to one pollutant makes a person more vulnerable to health effects when exposed to another pollutant.

Table 5 shows the extent to which each population subgroup resides in counties with multiple air quality standard violations. Unlike Table 4, the percentages in Table 5 can be summed by racial/ethnic category. Whites are more concentrated in the "none" and "1 pollutant" levels than are any of the minority groups, except Native-Americans. In contrast, most minority groups are relatively more concentrated in the areas where more (2 through 5) air quality standards are unmet. About 50 % of the African-American population, nearly

three-quarters of Asian-Americans, and 60 % of Hispanics lived in areas violating two or more air quality standards, compared to less than 35 % of Whites. These groups are less concentrated in areas with better air quality than Whites, as is indicated by the percentages in the top row of Table 5. In contrast, Native-Americans are less concentrated than Whites in non-attainment areas. For the other minority groups, however, the multiple pollutant patterns in Table 5 show the same degree of minority "excess exposure," compared to majority exposure, as do the patterns for single pollutants presented in Table 4. In general, the pattern in Table 5 shows that substantially higher percentages of minorities live in non-attainment areas for multiple pollutants than do Whites.

## CONCLUSIONS

The evidence is substantial that African-, Asian-, and Hispanic-American populations are disproportionately exposed to levels of air pollution that are considered injurious to health. Prior research indicates that this exposure differential has existed in some areas of the country at least since the 1970s and it certainly existed in 1990. The evidence regarding differential rates of diseases associated with air pollution exposure is not clear. Some minority groups have substantially higher morbidity or mortality rates for some types of health effects, especially asthma, cancer, and lead poisoning. However, Whites have higher rates of some respiratory diseases, such as emphysema and bronchitis, in which air pollution exposure is a precipitating or exacerbating factor. Though a clear, causal relationship between disproportionate minority exposures to air pollutants and elevated rates of air-

pollution-related health effects has not been documented, there is considerable circumstantial evidence of a relationship.

The difficulties in measuring exposures and controlling external variables in epidemiological research on air pollutants are so great that racial differences are generally not a major research focus. Accounting for multiple pollutant exposures and cumulative exposure over time adds additional complexity to the effort to understand disease causality. However, there appears to be a basis and need for action regardless of the research gaps since the evidence points to greater rates of current exposure of minority groups to multiple pollutants, and there is a history of disproportionate exposure of these populations, and minorities have elevated morbidity and mortality rates for some air-pollution-related diseases.

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**Figure 1. Factors Influencing Mortality Rates**

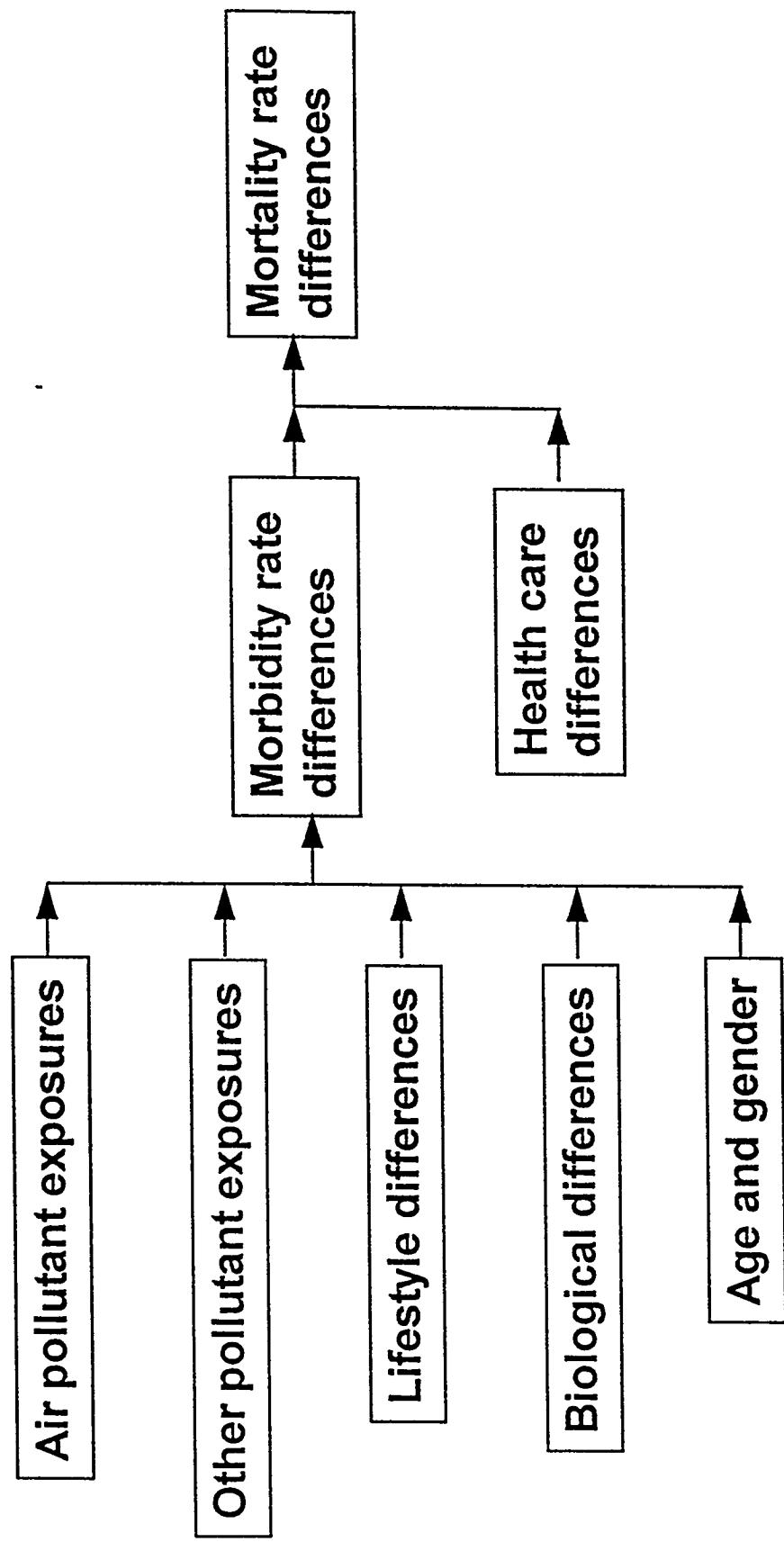


Table 1. Summary of Finding Regarding Differential Rates of Air Pollution-Related Health Effects

Disease	Comparative Rates	Measure Employed	Study
Chronic obstructive pulmonary disease	African-American, Hispanic, and Native-American lower than White	Age-adjusted mortality	3
Chronic bronchitis	African-American Lower than White Higher for low income groups	Self-reported incidence	3
Pneumonia influenza	African-American and Native-American Higher than White	Age adjusted mortality	3
Asthma	African-American higher	Mortality by age group	10
Asthma	African-American higher	Hospitalization	11
Cancers	African-American highest overall	Incidence and mortality	12
Cancers (leukemia)	African-American children lower <u>but</u> survival rate lower	Incidence	13
Cancers (lung and bladder)	Non-white males higher than White	Mortality	14
Lead poisoning	African-American children higher than White in all income groups	Blood lead levels $> 15 \mu\text{g/dL}$	15
Lead poisoning	African-American children and poor children higher	Blood lead levels $> 10 \mu\text{g/dL}$	16

Table 2. Age-Adjusted Cancer Mortality Rates for Population Subgroups, 1988<sup>22</sup>

Mortality rate per 100,000 population

Cause of Death	White	All Minority	African-American
All Cancers	130.0	151.9	171.3
GI Tract Cancer	28.6	40.2	43.7
Lung Cancer	39.4	43.3	49.8

Table 3. Percentage of Children Under Five Years Old With Blood Lead Levels  
 $> 10 \text{ } \mu\text{g/dL}$ , Change Over Time<sup>16</sup>

Population Group	1976-1980	1982-1984	1988-1991
Total, age 1-5 y	88.2	--	8.9
Non-Hispanic Blacks	97.7	--	20.6
Non-Hispanic Whites	85.0	--	5.5
Mexican-American (4-5 y)	--	61.5	4.9

Table 4. U.S. Racial/Ethnic Population Percentages in Substandard Air Quality Areas, By Pollutant, 1990

Pollutants	Whites	Racial/Ethnic Groups				
		African-Americans	Native-Americans	Asian-Americans	Other Races	Hispan Ameri
Carbon Monoxide	32.6	45.9	26.8	71.6	63.4	58.0
Lead	5.9	8.9	3.8	16.1	21.6	18.8
Ozone	51.9	63.1	30.7	82.1	80.0	74.1
Particulate Matter	14.8	15.7	17.4	32.0	41.5	36.1
Sulfur Dioxide	6.5	11.1	5.3	4.4	6.2	5.6

Table 5. Percentages of U.S. Racial/Ethnic Populations in Counties with Multiple Air Quality Standard Violations, 1990

Number of Air Quality Standards Violated	Racial/Ethnic Groups					
	Whites	African-Americans	Native-Americans	Asian-Americans	Other Race	Hispanic-Americans
0	41.4	34.0	59.1	12.2	18.4	16.3
1	24.1	16.4	13.4	14.2	19.6	15.7
2	21.6	30.6	15.3	45.0	28.9	29.8
3	7.9	10.3	8.5	12.8	17.2	18.6
4	4.5	7.6	3.4	15.5	15.8	19.4
5	0.5	1.2	0.1	0.3	0.1	0.2
Total	100.0	100.1	99.8	100.0	100.0	100.0