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Segregation and Black/White Differences in Exposure to Air Toxics in 1990

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I examined non-Hispanic Black and non-Hispanic White differences in exposure to noncriteria air pollutants in 44 U.S. Census Bureau–defined metropolitan areas with populations greater than one million, using data on air toxics concentrations prepared for the U.S. Environmental Protection Agency as part of its Cumulative Exposure Project combined with U.S. census data. I measured differences in exposure to air toxics through the calculation of a net difference score, which is a statistical measure used in income inequality analysis to measure inequality over the whole range of exposures. The scores ranged from 11.52 to 83.60. In every metropolitan area, non-Hispanic Blacks are more likely than non-Hispanic Whites to be living in tracts with higher total modeled air toxics concentrations. To assess potential reasons for such a wide variation in exposure differences, I performed a multiple regression analysis with the net difference score as the dependent variable. Independent variables initially included were as follows: the dissimilarity index (to measure segregation), Black poverty/White poverty (to control for Black/White economic differences), population density and percentage of persons traveling to work who drive to work (alone and in car pools), and percentage of workforce employed in manufacturing (factors affecting air quality). After an initial analysis I eliminated from the model the measures of density and the persons driving to work because they were statistically insignificant, they did not add to the predictive power of the model, and their deletion did not affect the other variables. The final model had an R^2 of 0.56. Increased segregation is associated with increased disparity in potential exposure to air pollution. *Key words*: air toxics, environmental justice, race, segregation. *Environ Health Perspect* 110(suppl 2):289–295 (2002).
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For over 15 years, there have been efforts to study the relationship between race, income, and exposure to environmental hazards. This study introduces statistical methods developed in the social sciences and analyzes new environmental data. In this study I examine whether non-Hispanic Blacks and non-Hispanic Whites have differing levels of potential exposure to a wide range of toxics in ambient air, a possibly important component of racial environmental disparities. (Throughout this article, Black means non-Hispanic Black and White means non-Hispanic White, unless specifically mentioned otherwise.) Then I analyze the relationship between disparate exposure levels and segregation, a potential mechanism for the adverse impact of racism and environmental injustice on people of color.

Using a measure of inequality called the net difference score, this study combines U.S. census data and U.S. Environmental Protection Agency (U.S. EPA) estimates of 1990 air toxic levels to assess the differences in potential exposure of Blacks and Whites. I found that in every large U.S. metropolitan area of over one million people, Blacks are more likely than Whites to be living in census tracts with higher estimated total air toxic levels. The study uses a model that explains the variation of the net difference score using independent variables including the ratio of Black poverty rates to White poverty rates, the percentage of the total metropolitan workforce that is employed in manufacturing, and the level of racial residential segregation as measured by the dissimilarity index. The hypothesis is that the relationship between disparities in potential exposures and disparities in poverty and manufacturing would be consistent with other studies. The nature and strength of the relationship between segregation and potential exposure disparities suggest that one consequence of racial residential segregation is disproportionate risk of exposure to environmental burdens. Although the relationship between this difference in total potential exposure and individual health risk is unknown, this suggests one way segregation may adversely affect the health of U.S. Blacks.

Background

Despite continued controversy, a consistency of results is developing from studies assessing the relationship between race, ethnicity, income, and potential exposure to environmental hazards or problems (*1*). In general, studies that use a geographically restrictive methodology—microarea studies comparing tracts or block groups that have one or more large users of toxic chemicals or hazardous waste facilities with tracts or block groups that have none—tend to find that race is not a significant factor in the siting of these facilities. However, these studies find that the higher the percentage of low-income households (or lower mean or median household income), the more likely the tract is to have an undesirable facility (*2–5*). These studies show that microareas with undesirable facilities tend to be disproportionately the home of lower income, White and manufacturing-employed people. If the exposure area definition is meso-area based (*6*), in other words, expanded to include census tracts adjacent to the facility in question, the ZIP code of the facility, or tracts within a certain geographical distance, then race and ethnicity appear more important and low income (however defined and measured) recedes as a risk factor (*7–9*).

Macroarea studies comparing region, state, or county levels are more difficult to characterize. For example, they tend to find that counties with polluting facilities have higher incomes and more persons of color than counties without such environmental issues (*10–12*). This may be a result of a clustering of hazardous facilities and users in urban areas that collectively are wealthier than those in rural counties, have greater amounts of economic activity, and, for reasons not yet understood, are more likely to have large non-White populations. These macroarea analyses may be problematic because they are ecologic (*13*) or because they are too large in scale to capture local effects.

There have been few comprehensive examinations of nationwide racial disparities in exposure to broad levels of pollution (*14*). Many studies have focused on only a single metropolitan area, state, or region and may not adequately reflect national trends (*15,16*). Most have included single pollution sources or a single category of sources, usually toxic waste facilities or facilities that report under the Toxic Release Inventory (TRI) (*17–20*). All large facilities that release

more than threshold amounts of specified potentially hazardous chemicals report their releases to the federal government and are included in the TRI. Thousands of these facilities exist across the United States, and many may have significant adverse effects on local environments. But TRI facilities contribute only an estimated 10% of the total amount of toxics in our air. The remainder are also produced by cars, buses, and small users of chemicals. Focusing only on TRI produces only a limited picture of ambient pollution exposures (21). In addition, as manufacturing shifts from cities to suburbs, from north to south, and from the United States to other countries, the importance of TRI may decline in older center cities. Furthermore, to the extent that economic restructuring has disproportionately affected Black communities and inner cities (22,23), it may lessen TRI-based disparities. Other studies have only examined potential disparate impacts in single states or regions defined by the U.S. EPA. There has been no comprehensive national study of metropolitan conditions.

Because most air monitoring data are not easily adapted to the task, comparing air pollution exposures across racial groups has been very difficult, and results have been unclear (24). Most metropolitan areas have a limited number of monitoring stations, usually fewer than a dozen in a multicounty area, making local area extrapolations difficult. Most of these monitoring stations only track a subset of the criteria pollutants (ozone, oxides of nitrogen, sulfur dioxide, particulates, carbon monoxide, and lead). Furthermore, some pollutants do not vary across a metropolitan area; for example, ozone in the Northeastern United States results in part from long-range transport from outside the region, with only minor variation in local levels (25).

Since the 1950s, there has been a broad effort to measure and track the degree of racial segregation in U.S. cities (26). A number of indexes have been developed and used to compare cities in social science research (27,28), but they have not been used in environmental research. Although it has declined from peaks right after World War II, Black–White segregation persists at levels markedly higher than that for other groups (29,30). Compared with historical and contemporary levels of segregation of other groups, Black–White segregation ranges from high (31) to what has been characterized as hypersegregation, extremely high levels of segregation as measured on several different dimensions (32). As yet, environmental justice researchers have not incorporated these indexes into their study designs.

A number of studies have examined the impact of segregation on the health of Blacks (33–36). Not only have these studies found that racial segregation is associated with increased mortality; they have also pointed the way for further research that may lead to better understanding of why Black–White health outcomes remain unequal (37). Most of these studies postulate that increased segregation leads to increased stress, which in turn leads to ill health (38,39), or that segregation results in a lack of services that eventually affects the health of Blacks (40). Although stating the possibility of a segregation–environment link, they have not examined the relationship between segregation and exposure to pollution that may lead to further understanding of how segregation leads to poor health outcomes.

Methods

This study is designed to test the hypothesis that there are inequities in potential exposure to air pollution between Blacks and Whites in large U.S. metropolitan areas. The study has two parts: an examination of potential disparities in exposure to air toxics between Blacks and Whites, and a multiple regression analysis of what factors may contribute to these disparities.

The proposed model is that the difference in overall exposure between Black and White populations in a metropolitan area is a function of the degree of Black–White residential segregation, differences in poverty rates between Blacks and Whites, the amount of manufacturing, overall population density, and the amount of automobile use in the metropolitan area. I used the overall population exposure to avoid problems in predicting individual exposures. Not only are the air toxics modeling predictions less reliable at the individual level, but controlling for individual characteristics would also be necessary. Segregation is a community-level attribute that reflects the degree to which one population has a different spatial distribution than another across an area. Any individual may or may not live in an area with a high percentage of their own racial group, but the group's overall exposure can only vary to the degree to which its distribution differs. Segregation establishes the outer boundary of populationwide exposure differences. The poverty variable, chosen here to represent the differences between Black and White income, controls for the fact that many studies have found that low income is a risk factor for living near an environmental hazard. Manufacturing, density, and driving variables control for the three main types of inputs into the air toxics model. They are responsible for a large percentage of air toxics production and may provide an alternative nonracial explanation for differences in Black–White exposure.

Exposure is defined as the total modeled concentrations of air toxics in the census tract of residence of each person in a metropolitan area, expressed in micrograms per cubic meter. Total modeled concentrations for 1990 vary from approximately 8 to more than 200 $\mu g/m^3$ in urban census tracts. This is not a measure of health impacts, and the relationship between ambient air concentrations and individual exposures is not known. Nor is there an understanding of how these concentrations may be related to health. Although some studies have weighted these concentrations by cancer death risk and deviation from noncancer threshold impacts (41), I use this nonweighted approach here to avoid methodologic questions on how to evaluate individual health impacts of these concentrations and how these impacts may be affected by differences between Black and White populations. The summed total of air toxics in a census tract may not be indicative of anything more than overall air quality and may not have any health implications. Racial variations in health impacts of these concentrations are a subject for future study.

I limited the scale of the study to metropolitan areas for several reasons. States are too large and heterogeneous, and their use as a frame of reference could result in potential confounding by urban/rural differences. Cities vary greatly in their proportion of metropolitan area and population, and the fact that city boundaries are set by historical and local political reasons makes them less uniform. Also, using them as the scale for a study would exclude important inner-city/suburban variation in exposure. Metropolitan areas are more uniform, having been defined by a standard methodology on a national basis. They best reflect residential housing markets, they function as coherent economic and social units, and comparing racial differences in exposure within a metropolitan area avoids potential confounding issues posed by trying to contrast exposures in radically different regions of the country.

I limited the study to metropolitan areas of over 1,000,000 people. The 1990 metropolitan areas have been defined by the U.S. Census Bureau as specified constituent counties (and a few individual independent cities). For Boston and Providence, I used the definition of a New England County Metropolitan Area as designated by the U.S. Census Bureau because the Bureau of Labor Statistics and other agencies use these county-based definitions (New England data are also available in census-defined metropolitan areas formed as aggregates of cities and towns, but this format is less compatible with other data sources). I obtained data on the White population and Black population

from the U.S. Census Bureau's Summary Tape File 3 (STF-3) data set (*42*).

I compare each metropolitan area's Black population with its White population. Although I could do this analysis for other racial groups, I restricted it to Black–White differences because large numbers of Black people live in all but one large metropolitan area (Salt Lake City, Utah) and Black–White disparities in segregation and health status are greater than those between other groups (*32*). The way in which the U.S. Census Bureau asks information about race and Hispanic ethnicity affects how they present the data. Persons of Hispanic origin may be of any race, so it is necessary to distinguish between Hispanic and non-Hispanic individuals. No biologic or genetic basis supports these racial and ethnic definitions. Numbers of persons of each race are based on self-report to the Census Bureau. Other studies have found that Hispanics have large differences from non-Hispanic Whites in potential exposure to environmental problems (*43,44*).

Net Difference Scores

The net difference score is a statistical measure based upon cumulative frequency distributions. Historically, it has been used in income inequality analysis, and I adapted it here to measure inequality in exposure to air toxics (*45,46*). The advantage of using a net difference score is that it measures inequality over the whole range of exposures. As used here, the cumulative distribution function ranks a population by exposure levels and the percentage of the total population at or below a given exposure level. If graphed, the *y*-axis ranges from 0 to 100% of the population and the *x*-axis is the exposure level, ranging from lowest to highest.

Comparing means or medians of exposure would overly focus on differences near the center of the exposure distribution and would be insensitive to values at the high and low ends. Exposures at these upper and lower ends may be of critical importance in assessing potential health impacts. Alternate measures are also based on cumulative frequency distributions. But most of these alternatives weight values at the high or low ends or both, making changes in the distribution at these points more influential on results than changes made elsewhere. Although this may have utility in other research areas, it is problematic in environmental research. For exposure to a negative such as air toxics, any decrease in potential exposure is desirable. To the extent that most toxicologic models assume a straight-line dose–response relationship, any change in potential exposure should be considered of equal value, regardless of where on the distribution the change occurs. The net difference score best meets these attributes.

The net difference score is calculated using the following formula:

$$ND_{wb} = \sum_{i=1}^{I} \sum_{j=1}^{I} \left(X \cdot W_i \cdot B_j \right) \qquad [1]$$

where, if $i > j$, $X = +1$; if $i = j$, $X = 0$; and if $i < j$, $X = -1$. W = Whites, B = Blacks, i is the exposure level of Blacks, and j is the exposure level of Whites. W_i is the number of Whites living in a census tract at exposure level i. B_j is the number of Blacks living in a census tract at exposure level j.

The net difference score takes the cumulative distribution function for each group and determines the probability that an individual in group W is more exposed than a person in group B minus the probability that an individual in group B is more exposed than a person in group W. That is, if I randomly pick a Black person and a White person in a given metropolitan area, the net difference score is the probability that the Black person is living in a census tract with a higher level of total estimated air toxics than the White person, minus the probability that the White person is living in a census tract with a higher level of total estimated air toxics than the Black person. To put these results on the same scale as other measures used in this analysis, I multiply the results by 100. A score of 100 would mean that all Blacks are breathing air with a higher total modeled air toxics concentration than all Whites. A score of –100 means the reverse (*47*).

Dissimilarity Index

The dissimilarity index was developed in the 1950s to quantify racial residential segregation. It is commonly described as the percentage of Blacks (or any other group under consideration) that would have to move in order for them to be evenly distributed across a given metropolitan area. For example, a Black–White dissimilarity index score of 60 would mean that 60% of all Blacks in a metropolitan area would have to move from their current census tract of residence in order for the Black population to have a similar distribution as the White population in a metropolitan area. The formula for the dissimilarity index is

$$D_{bw} = \frac{1}{2} \sum \left| \frac{b_i}{B} - \frac{w_i}{W} \right| \qquad [2]$$

where w is the number of White persons living in tract i, b is the total number of Black persons living in tract i, W is the total number of White persons in the metropolitan area, and B is the total number of Black persons. As in the other measures used in this study, I multiply it by 100.

For this study, the dissimilarity index reflects the degree to which Blacks are over- or underrepresented relative to Whites across all the census tracts in a given metropolitan area. Segregation is a characteristic of the entire population of an area and does not simply reflect where an individual may live or population levels in a single census tract. Many other indexes measure segregation, but the dissimilarity index is the most commonly used. The others are based on measures of clustering, isolation, and the degree to which the group of interest is concentrated in center cities. The advantage of the dissimilarity index is that it is geographically based and can be calculated on any geographic level. It measures the degree of segregation in residential location. Polednak (*48*) used 1990 census data to calculate the dissimilarity index scores used in this study.

Air Toxics Data

The U.S. EPA Air Toxics Data, a national set of estimates of ambient air concentrations for all 60,000+ census tracts in the continental United States, are relatively new (*49,50*). Air toxics are noncriteria air pollutants that have been prioritized because of their potential health consequences (*51–53*). They include 148 chemicals such as benzene and formaldehyde; metallic compounds including chromium, lead, and mercury; and complex compounds including polycyclic organic compounds. The air toxics data are comprehensive, including estimates on each of these toxics in every census tract. The data have great variability; the census tracts with the highest total modeled concentrations have levels over 20 times those of the tracts with the lowest modeled concentrations.

Data on 1990 air toxics, published by the U.S. EPA (*54*) on CD-ROM, are the first reliable national estimate of local pollution exposures. The U.S. EPA developed the model to assist in assessing the exposure of the United States population to air toxics in the ambient environment. The model relies on the inventory of toxics users and other permitted facilities, other large stationary sources, small stationary sources such as dry cleaners, and mobile sources including cars, trucks, and trains. Approximately 20% of the total volume of air toxics in 1990 came from large stationary sources, 40% came from small stationary sources, and 40% from mobile sources. The estimates are derived using a dispersion model that predicted the transport of air toxics up to 50 km from their sources and includes estimates of decay, secondary formation, and deposition appropriate for each substance. For each census tract, I used a geographic center point called a centroid to determine distance from various pollution

sources. I did not incorporate any long-range transport into the model.

The model has been peer reviewed for accuracy and reliability and compared with actual monitoring data (55). The database has been used to estimate potential health risks (56), and a study examining the links between race and exposure to air toxics in the Los Angeles, California, area is underway (57). Evaluations of the quality of the data have been published, and the data have been used in several published studies by U.S. EPA–funded researchers. Comparisons to actual measured air toxics are close, with the modeled predictions tending to be lower than actual air samples, perhaps in part because the model does not include long-range transport of air toxics. The primary concern with these estimates is that they are outdated and do not reflect efforts to improve air quality after 1990 (58). I extracted estimated concentrations and matched them, census tract by census tract,

to each tract in the metropolitan areas included in the study. The correspondence between the census data and the air toxics data was 100%. I summed air toxic concentration estimates for each of the 148 chemicals and compounds to give an overall score for each census tract expressed in micrograms per cubic meter.

For each metropolitan area, I regrouped tracts by their total air toxic concentrations into eleven categories: <10 $\mu g/m^3$, 10 to <20 $\mu g/m^3$, . . . , 90 $\mu g/m^3$ to <100 $\mu g/m^3$, 100 $\mu g/m^3$ and above. The net difference score depends on the number of data categories, and it is necessary to use a standardized number of groupings in order to compare different metropolitan areas. To date, there has not been a net difference methodology applied to these data, and I developed an 11-group characterization to provide a useful distribution of exposures. I summed the numbers of Whites and Blacks for each category, which served as the basis for calculating the net difference score.

Other Data

I obtained 1990 census data for population density, Black poverty rates, and White poverty rates for each metropolitan area from the U.S. Census Bureau website. I combined them to form a ratio of Black poverty rate to White poverty rate for each metropolitan area. I obtained the percentage of people employed in manufacturing in 1990 from the Bureau of Labor Statistics website (59) and divided the number of people employed in manufacturing in January 1990 by the total number of people in the civilian labor force for that metropolitan area at that time. I also took the percentage of people driving to work, including those driving alone and those in car pools, in each metropolitan area from the 1990 U.S. census.

I used SAS (60) to calculate the correlation of each dependent variable with the net difference score. I then performed an initial regression analysis with the net difference score as the dependent variable and the Black/White poverty ratio, percent employed in manufacturing, dissimilarity index, population density, and percent driving to work as the independent variables. In the initial analysis, population density and the percent driving to work variables proved to be poorly correlated with the net difference score, did not add to the predictive value of the regression model, and had very high *p*-values. In

the final regression, I discarded them, leaving the Black/White poverty ratio, percentage employed in manufacturing, and dissimilarity index as the input variables.

Results

This analysis includes 44 metropolitan areas (Table 1). Collectively they represent 41% of the total U.S. White (both Hispanic and non-Hispanic) and 56% of the total U.S. Black population (both Hispanic and non-Hispanic) (Table 2). Total estimated air toxic concentrations varied substantially from census tract to census tract both within metropolitan areas and between metropolitan areas. But in every metropolitan area, Blacks are more likely than Whites to be living in tracts with higher estimated total air toxics. The net difference scores ranged widely, with some metropolitan areas having small net differences (Salt Lake City, Utah = 12, Dallas, Texas = 16) and others approaching an extreme level of inequality (Cincinnati, Ohio = 84, Detroit, Michigan = 71).

In general, metropolitan areas in the Midwest and East had the largest net difference scores. Newer metropolitan areas in the West and South and those metropolitan areas with relatively small Black populations had smaller net difference scores. The New York City metropolitan area, consisting of the five city boroughs and two suburban counties, appears to be atypical. Because the vast majority of both Whites and Blacks live in census tracts with high total estimated air toxic levels in the city, and the lower level suburban tracts contained but a fraction of the total population, the distribution of exposures was skewed to the higher categories and the net difference score was small, only 21. It is anomalous because a large percentage of Whites live in center city tracts with high modeled exposure levels.

The Los Angeles, California, metropolitan area, consisting of Los Angles County, had a more typical distribution of Black and White exposures. Its net difference score of 45 is close to the mean for all the study's metropolitan areas (Figure 1). Graphing the distribution of Whites and Blacks across all 11 exposure-level categories shows that Whites had a higher percentage in each of the six lowest exposure categories. Blacks had higher percentages in the five highest exposure categories. These are percentages of each group, not actual numbers. For example, because several times as many Whites as

Table 1. Black/White net difference scores, 1990.

Metropolitan area	Net difference score
Atlanta, GA	45
Baltimore, MD	48
Boston, MA	32
Buffalo, NY	52
Charlotte, NC	36
Chicago, IL	43
Cincinnati, OH	84
Cleveland, OH	64
Columbus, OH	45
Dallas, TX	16
Denver, CO	40
Detroit, MI	71
Ft. Lauderdale, FL	30
Houston, TX	27
Indianapolis, IN	60
Kansas City, MO	56
Los Angeles, CA	45
Memphis, TN	51
Miami, FL	28
Milwaukee, MI	63
Minneapolis, MN	56
Nassau–Suffolk, NY	26
New Orleans, LA	41
Newark, NJ	62
New York, NY	21
Norfolk–Virginia Beach, VA	31
Oakland, CA	44
Orange County, CA	21
Philadelphia, PA	56
Phoenix, AZ	27
Pittsburgh, PA	51
Portland, OR	68
Providence, RI	61
Riverside–San Bernardino, CA	19
Sacramento, CA	21
Saint Louis, MO	66
Salt Lake City, UT	12
San Antonio, TX	25
San Diego, CA	31
San Francisco, CA	43
San Jose, CA	27
Seattle, WA	50
Tampa, FL	40
Washington, DC	41

Table 2. Summary statistics for 44 large U.S. metropolitan areas, 1990.

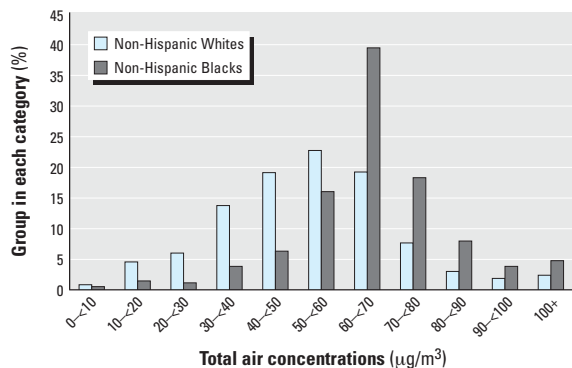| Variable	Mean	Median	Standard	Minimum	Maximum
Net difference score of exposure to air toxics	42.58	43.1	16.88	11.52	83.60
Black/White poverty rate	3.67	3.59	1.13	1.45	7.12
Dissimilarity index of residential segregation	69.16	70.5	12.32	41	89
Manufacturing employment	14.11	12.97	5.5	4	31

Figure 1. Los Angeles metropolitan area: estimated 1990 air toxics concentration totals by race.
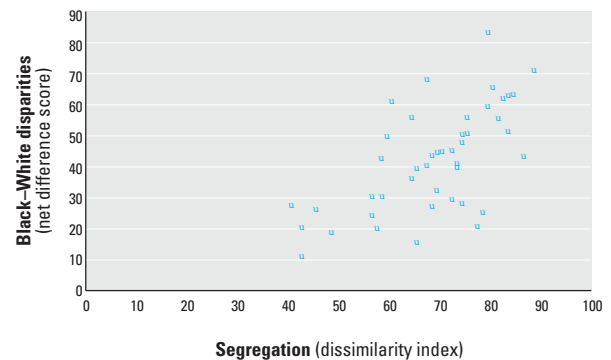

Figure 2. Black–White differences in exposure to air toxics in 1990 for 44 large U.S. metropolitan areas.

Table 3. Correlation of variables: 1990 Black/White difference in exposure to air toxics.

Variable	Manufacturing employment	Dissimilarity index of residential segregation	Black/White poverty	Net difference score of exposure to air toxics
Manufacturing employment	1.00	0.10	0.30*	0.42**
Dissimilarity index of residential segregation		1.00	0.58**	0.63**
Black/White poverty			1.00	0.61**
Net difference score of exposure to air toxics				1.00

Pearson's correlation coefficients. *Significant at the 0.05 level; **significant at the 0.01 level.

Table 4. Ordinary least-squares estimate of net difference scores for Black/White exposure to air toxics, 1990.

Independent variable	Parameter estimate (95% confidence limits)
Intercept	–27.16 (48.7, 5.6)
Dissimilarity index of residential segregation	0.62 (0.18, 0.97)**
Manufacturing employment	0.92 (0.24, 1.60)**
Black/White poverty	3.84 (–0.18, 7.87)

R^2 = 0.56. **Significant at the 0.01 level.

Blacks live in the Los Angeles metropolitan area, more Whites than Blacks live in census tracts with total modeled air toxic concentrations above 100 μg/m³, even though the percentage of all Blacks living in these tracts is higher. Some Whites live in census tracts with higher total estimated air toxic concentrations than Blacks, even though Blacks are overall more likely than Whites to live in census tracts with higher total estimated air toxics.

Individually, the percentage of people employed in manufacturing, the dissimilarity index, and the Black/White poverty ratio were all well correlated with the net difference score, with the last two variables particularly strong (Table 3). In addition, I observed a moderate degree of correlation between the dissimilarity index and the Black/White poverty ratio. This may be because poverty is concentrated by segregation levels, as hypothesized by Massey and others (61,62) and/or, conversely, poverty increases segregation.

Each of the independent variables performs as predicted with positive parameter estimates (Table 4). The dissimilarity index has the widest range of values, but its coefficient is still large. The ratio of Black poverty to White poverty has the smallest initial range and has a very large coefficient that just missed being significant at the 0.05 level. Perhaps a larger sample would result in meeting this threshold. The percentage of people employed in manufacturing also has a

large parameter estimate that reflects in part the small range of input values.

The final regression model was a good predictor of the net difference score, responsible for over half the variation in the scores. The dissimilarity index was highly correlated with the net difference score, with each point rise in the dissimilarity index resulting in a 0.62 increase in the net difference score, holding the other factors constant. The percentage of total work force employed in manufacturing was also a significant factor, although it is difficult to interpret which variable is more important given the differences in scale.

Discussion

In every large metropolitan area, Blacks are more likely than Whites to be living in census tracts with higher total modeled air toxic concentrations (Figure 2). However, the data have several limitations. Most important, living in a census tract is not the same as actual exposure or individual health risk. It is unclear how toxics modeled for a census tract centroid are related to levels throughout the tract and how modeled outdoor levels relate to indoor concentrations. Individual attributes, including the amount of time a person spends in the outdoors or outside his or her tract of residence, could greatly affect individual exposure. Air toxics vary considerably in their degree of toxicity. Weighting the individual air toxics for carcinogenicity, for

example, may produce substantially different results than weighting the toxics by reproductive toxicity. Further research should include a variety of weighted analyses as well as analysis of individual air toxics. Finally, the full health consequences of long-term exposure to air toxics at these modeled concentrations are unknown. Given the large disparity of incidence and outcomes for a number of diseases between Blacks and Whites, however, we should explore the possibility of an environmental influence on health disparities. Certainly the inequity of potential exposure is a matter of concern.

Although the air toxics levels are estimated, the model they are based on appears to be a good predictor when compared with actual monitoring data. The potential factor that may affect the results of this study is whether the underlying model has a systematic flaw that would result in errors related to the tract's racial composition. If the model has less predictive value in inner-city rather than in suburban tracts, for example, net difference scores may result because Blacks are more likely to live in those tracts (63). I have no evidence, however, that these data have such problems.

Three factors, Black/White poverty levels, percent employed in manufacturing, and degree of segregation as measured by the dissimilarity index, collectively explain over half the variation in the net difference score for exposure to air toxics in large U.S.

metropolitan areas. Other potential factors, including overall income inequality, relative political power, and local variation in environmental regulation (64), may also affect net difference scores and should be included in future research.

Income disparities have proven to be less predictive of potential exposure disparities in almost every environmental justice study that uses meso-area geographic exposure definitions. This study is consistent with those results because the model estimated the air toxic concentrations across multiple census tracts using a dispersion mode, making this is a meso-area study. The results here show that Blacks are more likely than Whites to live in census tracts with higher total modeled air toxics concentrations, partly because they are more likely than Whites to live in poverty, and poverty itself may be a risk factor for living in a poor-quality environment. Perhaps if I had used another measure of Black–White income differences, ratio of Black median income to White median income, or a net difference score calculated on Black–White incomes, the relationship may have been different. An interesting avenue of research would be to compare low-income and high-income Blacks and Whites. This may be an alternative way of understanding the interaction of race, income, and exposure to environmental problems.

Percent manufacturing is positively associated with a higher net difference score, consistent with other studies finding a relationship between race and living nearby (but not necessarily in the same census tract as) toxic chemical–using manufacturing facilities. Interestingly, a variable associated with the large stationary sources that represent only 20% of the model's total volume of concentrations proved to have predictive value, whereas the variable associated with mobile sources, which represent 40% of the total modeled air concentrations, did not have much utility. It could be that large facilities are locally significant even though they do not have a large national impact, or that the dispersion model does not adequately account for long-range transport of toxics. Whatever the relationship, this study puts toxic-emitting facilities in perspective, representing one set of factors among others (small-area sources, mobile sources) that collectively result in potential disparate exposures.

The relationship between percentage of manufacturing employment and disparities in potential exposure is interesting in light of historical studies documenting the problems Blacks had in accessing manufacturing employment. For example, the core of Detroit's Black neighborhoods is not a traditional heavy industrial area (65). In Philadelphia, Blacks initially settled in newly undesirable former streetcar suburbs (that had become part of the city) rather in the more industrial south side (66,67). On the other hand, maybe the metropolitan areas with high percentages of manufacturing employment represent a subset of older, more polluted cities with highly ghettoized populations. Despite these residential patterns in individual cities, manufacturing continues to be highly correlated with higher racial exposure disparities nationally.

One reason the percent driving to work variable was so poorly related to the net difference score may be that, except for the New York City, San Francisco, and a few other transit-dependent metropolitan areas, I observed little variation among metropolitan areas, with almost all within a few percentage points of 90%. Perhaps the density variable does not affect Black/White exposure differences because, despite its contribution to overall air toxics concentrations, it does not produce variability across a metropolitan area.

The strong relationship between segregation and net differences in exposure is surprising and disturbing. To the extent that Black/White segregation persists, so may disparities in exposure to air toxics. There are several potential ways that the level of segregation may affect the size of potential exposure disparities. Segregation may ultimately result in lower relative earnings and lower levels of wealth for Blacks, lessening their ability to move away from polluted areas. Segregation by definition represents limits on residents' location choices, and this also would decrease Blacks' ability to move away from pollution. Segregation may be symptomatic of an underlying level of racism in an area that may also be related to disparate siting decisions or other factors that result in a higher net difference score. Regardless of the mechanism, the relationship between segregation and disparate exposure is strong. Environmental factors should be considered in assessing the impact of segregation on health, and given the large percentage of Blacks living in segregated neighborhoods, should be included in research on Black/White disparities in health. Segregation should be considered as a risk factor for unequal exposure to environmental air toxics.

This study shares the drawback of other environmental justice studies in that it is cross-sectional, a snapshot in time of a relationship that may well be changing over time (68). It does not address the issue of whether the census tracts Blacks live in were polluted before they moved or whether pollution levels rose after Blacks were already residing there. Unfortunately, the data to make this assessment do not exist. In any case, the strong relationship between segregation levels and disparate exposure suggests that interpretations of historical precedence of negative facilities should be tempered by the possibility that segregation, discrimination, and constraints on residential location may be more determining than choice of neighborhood in the movement of Blacks into polluted areas.

In conclusion, Blacks are more likely than Whites to be living in census tracts with higher total modeled air toxics in every large metropolitan area in the United States. In addition to income inequality between Blacks and Whites as measured by the ratio of Black poverty to White poverty and the percentage of the metropolitan civilian work force employed in manufacturing, the degree of residential segregation as measured by the dissimilarity index predicts the level of estimated exposure differences to air toxics. Although this disparity may or may not be related to health, disparate exposure to environmental hazards may be one result of a segregated society.

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