The riskscape and the color line: examining the role of segregation in environmental health disparities

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Abstract

Environmental health researchers, sociologists, policy-makers, and activists concerned about environmental justice argue that communities of color who are segregated in neighborhoods with high levels of poverty and material deprivation are also disproportionately exposed to physical environments that adversely affect their health and well-being. Examining these issues through the lens of racial residential segregation can offer new insights into the junctures of the political economy of social inequality with discrimination, environmental degradation, and health. More importantly, this line of inquiry may highlight whether observed pollution – health outcome relationships are modified by segregation and whether segregation patterns impact diverse communities differently.

This paper examines theoretical and methodological questions related to racial residential segregation and environmental health disparities. We begin with an overview of race-based segregation in the United States and propose a framework for understanding its implications for environmental health disparities. We then discuss applications of segregation measures for assessing disparities in ambient air pollution burdens across racial groups and go on to discuss the applicability of these methods for other environmental exposures and health outcomes. We conclude with a discussion of the research and policy implications of understanding how racial residential segregation impacts environmental health disparities.

Keywords: environmental justice, segregation, health disparities, race/ethnicity

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1. Introduction

"The color line is not static; it bends and buckles and sometimes breaks."

(Drake and Cayton, 1945)

Race, as a social construct and mechanism of classification, has historically defined and continues to shape the distribution of power, privilege, and economic resources in American society (Crenshaw, 1988; Jones, 2001; Lawrence, 1987; Wellman, 1993). Myriad forms of past and present discrimination in the U.S. are imprinted onto our urban landscape, as evidenced by the persistent spatial separation of diverse communities along racial/ethnic and, to a lesser extent, class lines (Farley, 1995; Jargowsky, 1997; Logan and Molotch, 1987; Massey and Denton, 1993; Massey and Gross, 1994; Walker, 1981). Wide-ranging and complex political, socioeconomic, and discriminatory forces coupled with patterns of industrialization, disinvestment, and development have segregated people of color, particularly African Americans, into neighborhoods with some of the highest indices of urban poverty and deprivation (Peet, 1984; Schultz et al., 2002; Walker, 1985). Indeed, uneven industrial development, the movement of economic opportunities away from inner cities, real estate speculation, discrimination in government and private financing, and exclusionary zoning have led to systemic racial segregation among diverse communities with important implications for community health and individual well-being (Bobo, 2001; Harvey, 1989; Logan and Molotch, 1987; Massey, 2004; Morello-Frosch, 2002; Sinton, 1997; Wilson, 1996). The socioeconomic effects of urban segregation are further amplified by racialized boundaries in schools, the workplace and in some regions through policies such as immigration law and welfare reform (Hersh, 1995; Morello-Frosch, 2002; Pulido et al., 1996).

Although elements for understanding the relationship between residential segregation and community environmental health can be found separately in the sociology literature and the environmental justice literature, only two previous investigations have combined these lines of inquiry to analyze the relationship between outdoor air pollution exposure and segregation (Lopez, 2002; Morello-Frosch and Jesdale, 2006). Some researchers have recently argued that residential segregation is a crucial starting point for understanding the origins and persistence of environmental health disparities (Gee and Payne-Sturges, 2004; Lopez, 2002; Morello-Frosch, 2002b; Morello-Frosch et al., 2001, 2002b). Here we examine theoretical and methodological questions related to racial residential segregation and environmental health. We seek to address the following questions: 1) What are the various ways that segregation is conceptualized and how are these concepts measured? 2) Given that most measures of segregation consider only dyads, to what extent are existing measures of segregation valid for multi-ethnic regions? 3) How have segregation measures been applied to examine environmental health disparities such as air pollution? 4) Can these methods be used for other exposures and health issues? The paper begins with an overview of race-based segregation in the United States and proposes a framework for understanding its implications for environmental health disparities. We then discuss applications of segregation for assessing disparities in ambient air pollution burdens across racial groups and go on to discuss the applicability of these methods for other environmental exposures and health outcomes. Finally, we conclude by outlining some of the policy and regulatory implications of using residential segregation measures to research and track structural drivers of environmental health disparities.

2. Environmental health disparities in the context of neighborhoods and regions

The burgeoning literature on health disparities has compelled researchers to move beyond proximate causes of poor health toward identifying socioeconomic factors that shape distributions of health and disease in populations (House and Williams, 2000; Kaplan and Lynch, 1999; Link and Phelan, 1995; Navarro, 2002). This requires examining how the socioeconomic conditions of residential environments affect health and well-being and how the historical and locationally based antecedents of contemporary health issues continue to impact communities. Indeed, research strongly suggests that place affects health (Macintyre et al., 2002; Yen and Syme, 1999). Yet, despite the proliferation of work on the issue of segregation, there is a lack of scientific consensus about what it is about neighborhoods, and segregated neighborhoods in particular, that affects health (Evans and Kranowitz, 2002). Neighborhood-level factors associated with racial residential segregation may affect individual health by influencing food security (access to affordable markets with fresh fruits and vegetables); proximity to crucial services such as health care, parks, and open space (Center for Third World Organizing, 2002; Diez-Roux, 1997; Morland et al., 2002); the social environment (social capital, cohesion, and crime rates) (Conley, 1999; Kawachi and Berkman, 2003; Keister, 2000; Sampson, 1987); and the physical environment (traffic density, abandoned properties, and housing quality) (Reynolds et al., 2002; Shenassa et al., 2004; Wallace, 1990).

Researchers, policy-makers, and advocates concerned about environmental justice argue that communities of color who are segregated in neighborhoods with high levels of poverty and material deprivation are also disproportionately exposed to physical environments that adversely affect their health and well-being. Examining these issues through the lens of racial residential segregation offers insights into the junctures of the political economy of social inequality with

discrimination, environmental degradation, and health. This perspective also highlights how diverse legacies of discrimination shape current spatial distributions of pollution sources among diverse communities. More importantly, this line of inquiry may reveal whether observed pollution – health outcome relationships are modified by segregation and whether segregation disproportionately impacts certain populations. These issues are all important for understanding how place-based measures of social inequality shape environmental health disparities among diverse communities.

Segregation also promotes a regional perspective for understanding the dynamics of environmental health disparities. For example, conventional theories regarding regional development suggest that the formation of large cities in the United States was consonant with a history of industrial agglomeration in the urban core followed by a more recent countervailing trend of selective suburban economic development that drove desirable land uses to the periphery while remaining undesirable land uses continued to cluster in center cities and older ring suburbs. The morphology of the urban landscape is also shaped by shifting patterns of capital and state investment; governments at the local, state, and federal levels often promote industrial expansion by facilitating investment flows to outlying regional areas through highway construction and other infrastructure projects, tax breaks, and mortgage subsidies (Hise, 1997; Logan and Molotch, 1987). Historically, working-class and poor communities of color have been spatially bound in this process, remaining close to aging, large production facilities, because of limits imposed by job search, work hours, income, and exclusionary and discriminatory housing development policies (Guhathakurta and Wichert, 1998; Massey and Denton, 1993). Preliminary research using longitudinal data has sought to disentangle the causal sequence of facility siting in poor communities of color over time. Results have found little evidence of so-called “minority

move-in" into areas where potentially hazardous facilities had been previously located suggesting that the facilities are sited in previously established poor minority communities (Been and Gupta, 1997; Pastor et al., 2001; Saha and Mohai, 2005).

Imposed limitations on the spatial mobility of certain populations also undercuts their economic mobility because of the close connection between these two phenomena (Massey and Fong, 1990; Massey et al., 1991). Indeed, the historical and contemporary racial segmentation of the housing market erodes the property values of Black housing and limits the capacity of Black families to accumulate wealth through home equity (Conley, 1999; Oliver and Shapiro, 1995). Segregation can also cause so-called "spatial mismatch" between the location of lucrative jobs and the residential location of the communities that need them (Kain, 1968; Preston and McLafferty, 1999), leading to longer commute times, and possibly higher pollution burdens overall. Conversely, wealthier, mostly White, classes enjoy the mobility and privilege to pursue emerging economic opportunities and to escape the toxic zones of industrial activity (Pulido, 2000). Therefore, segregation can play out so that certain groups become concentrated, centralized, and isolated in abandoned inner city cores where employment opportunities are few and where communities are clustered around industrial sites, undesirable land uses, and/or transportation corridors that pose significant health hazards (Pulido et al., 1996).

Segregation, whose effects are experienced by individuals, is a phenomenon that occurs at a group level. By definition, segregation refers to the distribution of a specific demographic group across a geographic region, such as a metropolitan area. Therefore, the community health effects of segregation must be examined and remedied through policy decisions and interventions at the regional, metropolitan, state, or national levels. In general, the structural forces that create segregation tend to operate regionally, as evidenced by many current political

and economic regions that are not producing optimal outcomes for communities of color, the working class, and the poor, in terms of economic growth, and environmental quality (Pastor, 2001; Pastor et al., 2000). Metro areas and cities that are integrated along economic, political, and environmental lines have a more equitable distribution of resources and tend to collectively fare better on a number of important outcomes. Examples of such benefits include a stronger, more stable tax base, healthy communities, and planned land use development (Pastor, 2001; Pastor et al., 2000). The importance of regional equity can be extended to address regional disparities in health and the potential for improving outcomes by linking together the future of suburbs and cities. From a public health perspective, the rationale for taking a regional approach to examining links between segregation, environments, and health disparities is twofold: First, research strongly suggests that it is more fruitful to assess drivers of environmental health disparities at the regional level because economic trends, transportation planning, and industrial clusters tend to be regional in nature, even as zoning, facility siting, and urban planning decisions tend to be local (Morello-Frosch et al., 2002a). Second, research that examines how health inequities play out regionally could have implications for the development of interventions and policy initiatives that ameliorate fundamental drivers of environmental health and disease among diverse communities.

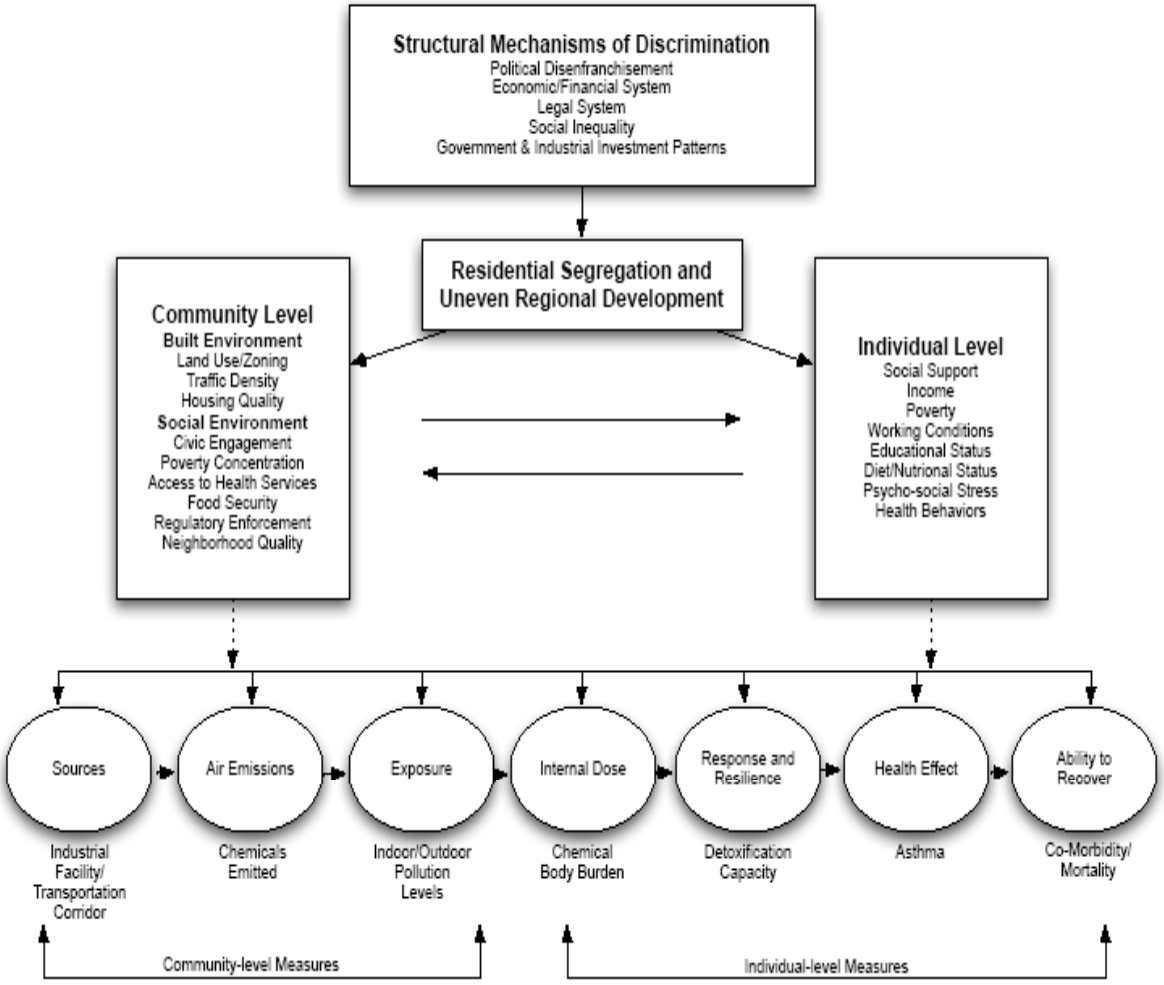

Fig. 1. Framework for understanding segregation and environmental health.

3. A conceptual framework for linking segregation to environmental health disparities

We propose a framework for understanding the relationship between racial residential segregation and various indicators of environmental health inequalities. Building on concepts proposed by other health inequality researchers (Gee and Payne-Sturges, 2004; Schultz et al., 2002), Figure 1 demonstrates an ecosocial or biosocial framework (Krieger, 1994, 1999; Massey, 2004) that connects a spatial form of social inequality (i.e., racial segregation) to community-level conditions that disproportionately expose communities of color to environmental hazards and stressors. These stressors potentially amplify individual-level vulnerability to the toxic

effects of pollution. We posit that this dynamic may partially explain persistent racial and class-based health disparities that are environmentally mediated.

The top of the figure shows the structural mechanisms that lead to residential segregation and result in community- and individual-level factors that influence disease burdens among diverse populations. Segregation solidifies racial disparities in socioeconomic status (SES), and it shapes the distribution of resources and wealth at the individual and community levels with important implications for community health. The bottom of the figure shows how these community and individual-level factors influence the exposure-health outcome continuum by increasing exposures to environmental hazards, amplifying the probability of adverse health effects, and affecting the ability to recover from hazardous exposures. This exposure-health outcome continuum connects the emission of a contaminant from a source (e.g., an industrial facility or transportation corridor in a neighborhood) to human exposure via various media (e.g., air), body burden and internal dose of contaminants, individual resilience (e.g., through detoxification mechanisms) and the occurrence of a health effect (e.g., asthma), and the ability to recover. The framework assumes that environmental contaminants lead to human exposures that can overcome the body's defense systems and have adverse health effects. This dose, if not effectively metabolized, neutralized, or excreted by the body's detoxifying and/or immune systems, can lead to biological effects that may alter system functioning and damage target organ systems. Individual and community-level stressors shape the effects of these differential exposures, including increasing or decreasing absorption, ability to detoxify or recover from toxic exposures, and the ultimate short- and long-term health effects from environmental contaminants. Community- and individual-level stressors and buffers can protect against or amplify vulnerability to the toxic effects of contaminants (Brunner, 2000; Gordon, 2003; Perera

et al., 2003; Rauh et al., 2004; Rios et al., 1993). Therefore, it is important to examine both community and individual levels of stressors (Diez-Roux, 1997, 1998, 2000) to assess their impact on health outcomes that are environmentally and socially mediated. These factors can include both social and biological elements, including pre-existing health conditions, socioeconomic circumstances, and psycho-social stress (Brunner, 2000; McEwen and Lasley, 2002).

The case of childhood asthma highlights connections between the elements of our conceptual framework, as researchers have begun to examine how stressors at the individual and community levels may influence the development and severity of disease among diverse populations (Busse et al., 1995; Gilliland et al., 1999; House and Williams, 2000; Institute of Medicine, 2000; Wright et al., 1998). Discriminatory forces leading to segregation drive community-level disparities in the quality of the built environment (e.g. traffic density and housing quality) and the social environment (e.g. poverty concentration, access to health services, food security, and regulation). Each of these community-level dimensions can act as stressors or buffers that impact individual-level vulnerability to air pollutant exposures that may be associated with childhood asthma. Gold and Wright hypothesize that community- and individual-level factors can act as potential modifiers of the relationships between pollutant exposures and asthma, through: 1) differential environmental exposures, 2) psychosocial stress and 3) the impact of 1 and 2 on individual health behaviors (Gold and Wright, 2005). Most important, these community-level stressors (e.g. poor housing conditions, food insecurity and poor neighborhood quality) can influence individual living conditions and health behaviors (e.g. household crowding, diet/nutritional status, and smoking). The interplay of these individual and community-level stressors results in a feedback loop: individual factors influence community

exposures that compound individual vulnerability, which ultimately influences the biological pathways linking pollutant exposures to asthma exacerbation and possibly the development of disease.

For example, tobacco smoke exposure is an important factor associated with the occurrence of childhood asthma (Li et al., 2005; Strachan and Cook, 1998), and smoking prevalence is often associated the target marketing of tobacco products in poor communities of color (Pollay et al., 1992) and with chronic stress (Kleinschmidt et al., 1997). Similarly, community food security includes access to affordable supermarkets, which can affect an individual's dietary intake of fresh fruits and vegetables. Recent research indicates that diet and nutritional status impacts respiratory health in children (Gilliland et al., 2003). Certain vitamins found in fruits and vegetables may protect the lungs against oxidative stress and promote healthy lung function and development (Gilliland et al., 2003). In addition to environmental factors, chronic life stress experiences may also affect childhood asthma morbidity. Recent studies indicate that higher levels of caregiver stress, due to lack of community social support, access to preventive health care services (Wright et al., 1998, 2002) and exposure to community violence (Wright et al., 2004) are associated with more severe asthma morbidity.

4. The dimensions and measurement of racial residential segregation

The fields of sociology and demography have given substantial attention to theorizing and measuring dimensions of segregation (Duncan and Duncan, 1955a, 1955b). Although a report by the U.S. Census lists over a dozen measures (Iceland et al., 2002), five basic dimensions of racial and ethnic segregation dominate the literature: *evenness, isolation, concentration, centralization,* and *clustering* (James and Taeuber, 1985; Massey and Denton, 1988; Stearns and Logan, 1986; White, 1986). *Evenness* measures the degree to which the

proportion of a particular racial or ethnic group living in residential areas (e.g. census tracts) approximates that group's relative percentage of an entire metropolitan area (Massey et al., 1996). This measure is the most extensively used indicator of segregation, both in the sociological and public health literature (Acevedo-Garcia et al., 2003). *Isolation/Exposure* assesses the extent to which a member of a particular racial/ethnic group is likely to have contact with members of the same group (isolation) or, conversely, the degree to which different groups would be exposed to each other by sharing common residential areas (exposure) (Massey et al., 1996). The point of this measure is to assess the diversity of neighborhoods and to capture some assessment of the daily experience of segregation felt by certain racial groups. *Concentration* measures the population density of a certain racial/ethnic group within a metro area (Massey and Denton, 1988). *Centralization* refers to a group's proximity to the center of a metropolitan area, which in some of the nation's older cities is characterized by extremely high levels of poverty, poor housing quality, and economic abandonment (Massey and Denton, 1988). *Clustering* assesses whether minority census tracts are contiguous and form a sort of "ethnic enclave" or are fairly spread out throughout a metro area (Massey and Denton, 1988). A table summarizing the formulae to derive these segregation measures appears in Appendix A. Census tracts and metropolitan statistical areas tend to be the primary macro and micro units of analysis to calculate these measures, but segregation measures can be derived using other units as well (e.g., zip codes or block groups to characterize the segregation of counties) (Iceland and Steinmetz, 2003).

Nearly all of the segregation measures focus on dyadic racial/ethnic comparisons: Black/White, Asian/White, Hispanic/White, and so on. Usually Non-Hispanic Whites serve as the reference group in these comparisons. Although these measures are informative, generalized

measures can illustrate patterns of segregation in a context of diversity where multiple racial/ethnic groups are simultaneously segregated from one another (Iceland, 2004). The generalized dissimilarity index, which is a variation of the dyadic evenness measure described above, measures segregation among many racial/ethnic groups simultaneously and provides a method for examining segregation in a way that accounts for the rise of multiracial metropolitan areas (Grannis, 2002; Reardon and Firebaugh, 2002; Sakoda, 1981).

Racial composition, or the existence of census tracts with a high proportion of specific minority groups, has been interpreted as a measure of the magnitude of segregation in a metro area. For example, the percentage of Blacks in a census tract has been used to study the health effects of segregated neighborhoods (Acevedo-Garcia et al., 2003; Fang et al., 1998; Jackson et al., 2000; Yankauer, 1950). Using racial composition as a way to operationalize segregation, these studies assume that racial composition directly reflects a dimension of racial/ethnic unevenness in a particular metro area. However, racial composition may not always be a true reflection of segregation *per se.* This is because segregation is a contextual measure that depends on the relationship between racial groups in neighborhoods (e.g., census tracts) across a larger geographic area (e.g., a metro area). Thus, while percent minority measures reflect the composition of a particular neighborhood, it does not assess whether a metro area's spatial organization reflects larger dynamics of racial inequality. For example, if a particular neighborhood in City X were composed of over 75% Latinos, this may give the impression that Latinos are highly segregated in that particular city. However, if the entire population of City X is 80% Latino, then the racial composition of that neighborhood merely reflects the larger racial composition of the metro area.

Table 1: Metropolitan Segregation with Whites Dissimilarity Index* for 1980-2000

	1980	1990	2000
African Americans	73.8	68.8	65.0
American Indians	37.3	36.8	33.3
Asian & Pacific Islanders	41.2	42.0	42.1
Hispanic	50.7	50.6	51.5

Source: U.S. Census 2000
**See text for explanation of the dissimilarity index.*

Table 1 shows patterns of racial segregation in the United States between 1980 and 2000. The segregation measure is a dyadic dissimilarity index, which calculates the level of inequality in the distribution (or unevenness) of each racial/ethnic group compared to Whites. Given the history of discrimination in the U.S., it is not surprising that African Americans experience the highest levels of residential segregation, although these levels have declined slightly over the last twenty years. It should be noted that the major portion of this decline has occurred in smaller metropolitan areas with smaller populations of African Americans. For other racial/ethnic groups, there has been surprisingly little change in their levels of segregation over the last twenty years.

In sum, the choice of which segregation measure to use depends on what dimension is being investigated. In general, segregation measures tend to be correlated; metropolitan areas with high levels of segregation along one dimension tend to have high scores on the other dimensions as well. All of the measures have different conceptual implications for environmental health research and assessing disparities in pollution exposures and outcomes that

may be environmentally mediated. Evenness is best adapted to study how segregation potentially modifies exposure-health outcome relationships. This measure can be used to compare environmental health indicators between metro areas and it is not affected by the relative proportion of the demographic groups being examined. The isolation or the exposure metrics reflect how members of minority groups actually experience residential segregation within metro areas and within their neighborhoods (Farley, 1984), through for example, access to supermarkets or the location of dismenities such as chemical plants or smelters that are fairly rare across the landscape.

The other three dimensions of residential segregation, (concentration, centralization, and clustering) are used less frequently, but tend to characterize the spatial patterns of segregation within metro areas. These measures may be particularly useful when examining environmental health questions involving a small number of metropolitan statistical areas (MSAs) that are similar in demographic make-up and overall size. These last three measures can help researchers better grasp how different spatial forms of segregation may disproportionately expose certain population groups to specific environmental stressors that ultimately degrade community health.

5. Analytical applications of segregation measures in environmental health

It remains unclear how socioeconomic inequality and segregation degrade the health of populations living in hazardous physical and social environments and ultimately lead to environmental health disparities. Place-based inequality measures, such as segregation, may modify and compound the adverse effects of hazardous environmental exposures, although this issue has not been thoroughly researched (Evans and Kranowitz, 2002). Few environmental health issues have been studied in the context of segregation, but air pollution has received some attention. These studies illustrate potential pathways between segregation and environmental

health outcomes. They also provide a framework for discussing other environmental health problems that have yet to be fully studied in the context of segregation. Below are three examples of analytical applications where measures of segregation can be used to understand environmental health inequalities related to outdoor air pollution.

5.1. Criteria pollutants

Since the passage of the 1970 Clean Air Act and the establishment of National Ambient Air Quality Standards (NAAQS), the monitoring of criteria air pollutants (carbon monoxide, sulfur dioxide, oxides of nitrogen, particulates, lead, and ozone) has become ubiquitous in most metropolitan areas. Exceedances of the NAAQS can bring sanctions and public action to insure compliance. Monitoring is usually limited to a small set of strategically placed locations to assess the overall air quality across an entire metropolitan area. These data allow for studying the association between segregation and overall levels of criteria air pollutants, but not necessarily permitting the study of neighborhood level effects.

Table 2 shows the results of a regression analysis using metropolitan area-wide criteria air pollutants levels as dependent variables and segregation and other metro-level factors as independent variables. Criteria air pollutant levels for each available metropolitan area were obtained from the U.S. EPA's Aerometric Information and Retrieval System (AIRS) database, which contains annual metropolitan area-wide averaged levels of selected criteria air pollutants (EPA, 2004). Black-White Dissimilarity Index scores were calculated by the Mumford Institute using 2000 Census data (Mumford Center, 2000). Other potential metropolitan level explanatory variables, such as the percent of the total population living in poverty, total population, per capita income, percent of civilian labor force employed in manufacturing, and the percent of Black residents, were obtained from the U.S. Census. Controlling for these metro-level SES variables,

Black-White segregation was associated with increased metropolitan-wide levels of sulfur dioxide and ozone. Segregation was also associated with increased levels of PM_{10}, but this association was not statistically significant. Segregation was associated with decreased levels of carbon monoxide and oxides of nitrogen.

Table 2: Criteria Air Pollution and Black-White Residential Segregation

Pollutant	Number of Metropolitan Areas	Coefficient	95% Confidence Interval
Carbon Monoxide	130	-0.019	(-.0021, -.036)*
Particulate Matter	201	0.006	(-.054, .066)
Oxides of Nitrogen	94	-0.00002101	(-.0000093, .000051)
Sulfur Dioxide	135	0.00004713	(.000014, .000080)**
Ozone	197	0.000233	(.000097, .00037)**

* Significant at the .05 level
** Significant at the .01 level

Multivariate regression comparing metropolitan area average pollutant level with Black-White dissimilarity index
Regression controlled for metropolitan level percent of people living in poverty, total population, per capita income, percent of civilian labor force employed in manufacturing

Table 3: Relationship between segregation and inequality of exposure to air toxics

	Total Unweighted	Cancer Weighted	Non-Cancer Weighted
Asian - White Segregation	0.0034 (-.0018, .0086)	0.0011 (-.0058, .0080)	0.0015 (-.0032, .0062)
Hispanic - White Segregation	0.007 (.0029, .0111)**	0.0059 (.0017, .0192)**	0.0054 (.0018, .0091)**
Black - White Segregation	0.007 (.0048, .0091)**	0.0046 (.0021, .0071)**	0.0052 (.0033, .0092)**

** Significant at the .01 level

Multivariate regression comparing metropolitan area net difference score with dissimilarity index
Regression controlled for metropolitan level percent of people living in poverty, total population, per capita income, percent of civilian labor force employed in manufacturing and percent of subject group residents.

Source: Lopez 2002.

5.2. Air Toxics

Unlike criteria air pollutants, air toxics, also referred to as hazardous air pollutants (HAPs), have no set air quality standards and they are not routinely monitored. However, through its National Air Toxics Assessment (NATA) database, EPA has modeled annual ambient HAP concentrations for 1996 for each census tract in the continental U.S. based on emissions data and estimates of local land uses and population (US EPA, 2005). Lopez examined the relationship between total ambient air toxics levels and metropolitan segregation (Lopez, 2002). Three methodologies were used to assess cumulative exposures in this analysis: summation of all HAPs (total unweighted); summation of the estimated lifetime cancer risks for the metropolitan area average concentration of each HAP (cancer weighted); and the summation of the ratio of estimated metropolitan area average concentration of each HAP to its corresponding non-cancer reference concentration. The association between HAPs and Black-White segregation was assessed in a similar way to that used for the criteria air pollutants. Results showed that levels of Black-White segregation were associated with higher levels of total HAPs, cancer risks, and non-cancer risks after controlling for other potential metro-level explanatory variables (Lopez, 2002).

The study also applied a Net Difference Score methodology that describes the probability that a randomly selected Black person within a metro area lives in a census tract with higher levels of HAPs than a randomly selected White person, minus the probability that the Black person is living in a census tract with lower levels of HAPs than the White person. In almost every metropolitan area (out of 331 total) Blacks were more likely to be living in census tracts with higher concentrations of HAPs regardless of which cumulative summation

methodology was used. In addition, the level of inequality was associated with increased segregation, even after controlling for other potential explanatory factors. Results were similar for Hispanics and Asians who were also more likely to be living in census tracts with higher pollutant burdens (Table 3) (Lopez, 2002).

A second analysis of the 1996 NATA data examined whether segregation patterns across over 300 MSAs modified racial disparities in cancer risk burdens associated with ambient air toxics concentrations (Morello-Frosch and Jesdale, 2006). In this study, the generalized index of dissimilarity was used to capture concurrent segregation across multiple racial/ethnic groups (Iceland, 2004; Sakoda, 1981). Other covariates in this analysis included: state grouping based on six regional categories of the continental United States in order to account for geographical variation in racial/ethnic segregation levels and its historical causes; population density; MSA population size; county-level voter turnout as a proxy for community civic engagement; local area deprivation, as measured by the Townsend index (Krieger et al., 2003), and poverty level.

A population risk index (PRI), which estimates a population-weighted average of census-tract-level cancer risks associated with modeled ambient air toxics exposures (Morello-Frosch et al., 2001), was used to assess environmental inequities across segregation, poverty, and racial/ethnic categories. Figure 2 shows the racial distribution of lifetime estimated cancer risk burdens associated with ambient HAP exposures across three levels of multi-racial segregation. The y-axis on the graph shows a population-weighted individual excess cancer risk estimate for each racial and segregation category. As indicated in the figure legend, each line in the graph represents one of the five racial/ethnic groups. The graph shows two patterns: first, it indicates that cancer risks across all metropolitan areas increase with increasing segregation levels for all racial/ethnic groups and that overall, Hispanics and Asians, followed by African Americans,

have some of the highest estimated cancer risk burdens associated with ambient air toxics in metro areas with higher segregation, as compared to the average across all groups and compared to Whites and Native Americans. Figure 3 shows the racial breakdown of cancer risk burden across poverty levels. Although there is a persistent racial gap across all levels of poverty, there does not appear to be a gradient with rising area-level poverty, suggesting that the effect of segregation functions independently of poverty in terms of its association with exposure burdens across racial categories.

Poisson models were used to examine the relationship between segregation and estimated cancer risks by stratifying by race/ethnicity and calculating risk ratios for each level of segregation, using low segregation as the referent group. The model controlled for metro area regional grouping, metro area population size, tract-level poverty and material deprivation (Townsend Index), and tract-level population density. Results indicate that increasing segregation amplifies the cancer risks associated with ambient air toxics for all racial groups, although the effect appears to be strongest for Latinos and African Americans (Morello-Frosch and Jesdale, 2006).

Taken together, these three air pollution/segregation studies imply that metropolitan areas with higher segregation levels tend to have worse air quality compared to low segregation areas. In addition, increased segregation may also be associated with increased racial inequality in exposure and estimated health risk burdens. The health implications of criteria pollutants and air toxics have been well documented (Leifkauf, 2002; Neidell, 2004; Peel et al., 2005). Therefore, to the extent that these pollutants are associated with myriad adverse health effects, the overall increase in pollutant levels associated with segregation may be important for understanding factors that contribute to environmental health disparities.

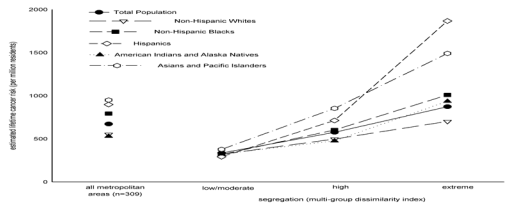

Fig. 2. Estimated cancer risk by race/ethnicity and racial/ethnic residential segregation, among residents of continental US metropolitan areas

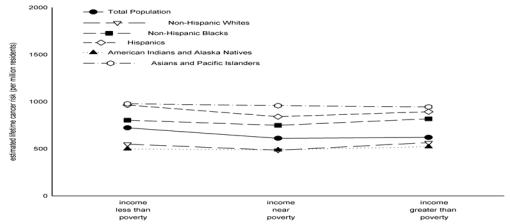

Fig. 3 Estimated cancer risk by race/ethnicity and poverty status, among residents of continental US metropolitan ares.

One way to better understand why segregation increases pollutant burdens and widens disparities in exposures would be to better link land use information with the location and density of major emissions sources. For example, research suggests that on average, mobile

sources of ambient pollution account for a significant portion of health risks associated with certain pollutants (particularly air toxics) (Morello-Frosch and Jesdale, 2006; Morello-Frosch et al., 2001; Reynolds et al., 2002), and that exposure to these sources are inequitably distributed across race and class lines (Gunier et al., 2003). Similarly, the relationship between neighborhood racial make-up and the siting of hazardous facilities has been long researched (Institute of Medicine, 1999; Pastor et al., 2001; Sadd et al., 1999; U.S. GAO, 1983; United Church of Christ, 1987). In general, these studies have found that both race and income are important predictors of disparate siting, although some have found that income is more important than race and others found the opposite (Fullilove, 2004; Pastor et al., 2004; Perlin et al., 1995; Sterling et al., 1993; Szasz and Meuser, 1997). Assessing whether segregation is associated with the proliferation of certain emissions sources (such as major traffic corridors) would help broaden understanding of the links between place-based inequality, land use patterns, and pollution distributions among diverse communities living in major metropolitan areas.

6. Applying segregation measures to study other environmental hazards and health outcomes

The techniques used to examine relationships between segregation and inequities in ambient air pollution exposures can be applied to other environmental health issues to elucidate socioeconomic drivers of environmental health disparities. Moreover, although the focus of this paper is on residential segregation, links between segregation and environmental health disparities can also be examined in other contexts, such as the workplace (e.g., to examine occupational health disparities) and in schools (e.g., to examine disparities in children's environmental health).

6.1. Lead & residential pesticide use

Childhood lead exposure is an environmental hazard for which there have been persistent disparities by race and income. While the prohibition of lead in gasoline and paint has resulted in a decreased risk of lead poisoning for most Americans, there is a continued problem of elevated lead levels for children living in older, substandard housing (Haley and Talbot, 2004; Mielke and Anderson, 1983). Increased levels of lead have been associated with an increased risk of a range of cognitive and behavioral outcomes (Needleman, 2004; Needleman et al., 1979). Certainly, there is evidence that low income communities of color, particularly African Americans, are concentrated in areas with older housing. This older housing is disproportionately likely to be contaminated with lead and more likely to be in such a state of disrepair, which increases the risk of lead exposure to residents, particularly children. These trends point to segregation as a distal cause of lead-related cognitive health effects that may disproportionately impact children of color. Nevertheless, no studies have examined this issue in terms of residential segregation. Yet the persistent racial and class-based disparities in childhood lead poisoning suggest that residential segregation may be concentrating communities of color, particularly African Americans, into poor inner-city neighborhoods with housing that has lead paint and lead contaminated soils (Breysse et al., 2004; Roberts et al., 2003). There are large disparities in elevated blood levels between Whites and Blacks, with Blacks being 13.5 times as likely to have blood lead levels above 20 μg/dL as Whites (Bernard and McGreehin, 2003). The role of segregation in causing these disparities, through increased likelihood of exposure to lead contaminated environments, could be investigated further as a way to understand some of the underlying social drivers that make the racial disparities in childhood lead poisoning persist.

Similarly, residential pesticide use is widespread in the United States, with approximately 80-90% of American households using pesticides (Landrigan et al., 1999; Whitmore et al.,

1994). Recent studies indicate that residential exposures to pesticides are associated with adverse birth outcomes (Eskenazi et al., 1999; Perera et al., 2003; Whyatt et al., 2002). Although little is known about residential pesticide use among minority populations in the United States, surveys suggest that frequency of use is more intense in public housing and in areas of high population density in housing (Surgan et al., 2002). Applying segregation measures to understand patterns of racial and class-based disparities in exposures to urban pesticides could also elucidate how consumer pesticides used to control pests in substandard housing or public housing projects may disproportionately affect certain minority groups.

7. Segregation in relation to health outcomes that may be environmentally mediated

There are profound racial differences in residential patterns and in environmental exposure burdens. Together, these may imply that segregation and the resulting inequality in the toxicity of residential environments may be contributing to racial differences in morbidity and mortality. The following suggest some of the potential associations and causal pathways between segregation and health outcomes that are environmentally mediated or that may enhance community vulnerability to the toxic impacts of contaminant exposures.

7.1 Adult Mortality

There is a growing body of evidence linking racial segregation to increased mortality risk among both Blacks and Whites, though the risk tends to be greater for Blacks (Collins and Williams, 1999; Cooper et al., 2001a,b; Polednak, 1996, 1997, 1991; Williams and Collins, 2001). Overall metropolitan levels of segregation were associated with increased total mortality and increased avoidable mortality (LaVeist, 2003). Controlling for individual risk factors, neighborhoods with high concentrations of Blacks have also been found to have higher levels of mortality (Schultz et al., 2002). The potential causes of these relationships are not well known,

but most likely mean increased exposure to social, economic and environmental risk factors (Bosma et al., 2001; Deaton and Lubotsky, 2001; Howard et al., 2000; McLaughlin and Stokes, 2002). In addition, the quality of health care and other services available to Blacks is lower (Leiyu and Starfield, 2001; Sheifer et al., 2000). In the context of segregation, these risk factors have the potential to act synergistically to raise allostatic levels of stress and simultaneously increase sensitivity to exposures, reduce the ability to access treatment and assistance, and reduce the ability to recover from environmentally mediated illnesses (Massey, 2004; Wallace and Wallace, 1998; Wallace, 1988). As a result, over time, mortality may increase (Fiscella and Franks, 1997; Kennedy et al., 1999). Further research on the health effects of segregation and adult mortality might include a better exploration of the health effects of individual pollutants, the study of how pollutants work synergistically with place-based measures of social inequality to increase adverse health outcomes, and modeling the impacts of exposures to pollutants in individuals with overstressed immune systems or who may be disproportionately vulnerable to the effects of pollution exposure due to place-based and individual-level factors.

7.2 Infant mortality and other birth outcomes

Since the first studies exploring the relationship between residential segregation and birth outcomes in the United States were published in the 1950s, the literature has been rather limited in scope and volume. It has focused almost exclusively on Black-White disparities in infant mortality rates, and has used a single dimension of segregation at a time, usually a measure of unevenness such as the index of dissimilarity. The research that does exist, however, has addressed the link between segregation and infant mortality from a few angles and at different levels of aggregation, from intra-city explorations of infant mortality rates by neighborhood (Yankauer, 1950, 1990; Yankauer and Allaway, 1958) to inter-city examinations of the variation

in Black-White infant mortality ratios (Jiobu, 1972; LaVeist, 1993). The literature over the past 50 years has established clear links between residential segregation, infant mortality, and Black-White infant mortality disparities. It is evident that racial inequalities in social environments engendered by racial segregation have put Black populations at a serious disadvantage relative to White populations, and have had a resounding impact on infant mortality rates among Blacks in the United States (Guest et al., 1998; LaVeist, 1989, 1993; Yankauer, 1990). These effects have consistently been shown to be at least partially independent of potential confounders, such as poverty levels (Bird, 1995). There are, however, a few serious gaps in the literature to date. First, the literature focuses solely on infant mortality, and has not assessed links between segregation and other birth outcomes, such as birth weight or preterm birth. Second, the literature only examines differences between Black and White infant mortality rates, and defines residential segregation as a Black-White phenomenon. Finally, no research has specifically examined the extent to which differential air pollution exposure may mediate and partially explain the relationships between broad social inequalities, neighborhood environments, and persistent racial disparities in birth outcomes.

Analyzing links between segregation, differential exposure to pollution, and birth outcomes among various racial and ethnic groups in the United States would be an important contribution to the literature. More specifically, differences in exposure to air pollutants due to residential segregation may be viewed as the physical manifestations of poor neighborhood environments that lead to poor birth outcomes. Preliminary research indicates that disadvantaged populations often experience a disproportionate amount of air pollution exposure (Woodruff et al., 2003). Other studies have linked air pollution exposure to negative birth outcomes (Dejmek et al., 1999; Dolk et al., 2000; Ritz and Yu, 1999; Ritz et al., 2000, 2002)and

found racial disparities in exposure burdens and in relationship to birth outcomes (Ritz and Yu, 1999; Ritz et al., 2000; Ritz et al., 2002; Woodruff et al., 2003). With one notable exception (Ponce et al., 2005), none of these studies have specifically examined place-based SES measures or segregation in conjunction with individual-level variables that may be associated with poor birth outcomes. Moreover, these studies have not assessed whether residential segregation amplifies observed associations between adverse birth outcomes and pollution exposures and how these dynamics play out across racial and ethnic groups. Examining this question, particularly in relation to birth outcomes that may be partially mediated by environmental factors might help elucidate how segregation contributes to environmental health disparities.

7.3 Asthma

Several factors related to the etiology of asthma may be associated with or exacerbated by segregation. Asthma rates are higher among Blacks than Whites (CDC, 2004; Grant et al., 2000), and the disease has been identified as the primary preventable cause of hospitalizations (Flores et al., 2003; Masoli et al., 2004; Pendergraft et al., 2004). The disparate risk of asthma is heightened by the dearth of access to health care in many Black majority communities. In addition to being less likely to have health insurance, hospitals in Black majority neighborhoods have been more likely to shut down than in other neighborhoods (Sager, 1983).

Asthma is often triggered by roaches, dust mites, and mold, all of which are linked to housing quality (Platts-Mills et al., 1995). Segregation, by limiting the housing options of communities of color and the poor, may lead to increased exposure to these triggers. Ozone, carbon monoxide, PM_{10}, and other pollutants have been implicated as asthma exacerbaters (Leifkauf, 2002; Loh and Sugarman-Brozan, 2002; Peden, 2002), and one study linked ozone exposure with the development of asthma among young children who play outdoor sports

(McConnell et al., 2002). If segregation is linked to increased levels of these pollutants, this may represent another pathway to ill health. The interplay of diverse factors leading to poor asthma outcomes might be better understood in the context of segregation including: attending schools in segregated districts with disparities in the quality of school facilities, living in poor quality housing, exposure to indoor and outdoor air pollution, and the distribution of preventive care and emergency care facilities.

8. Conclusions and implications for research and policy

Advocates working on environmental justice issues have urged scientists, policymakers, and the regulatory community to consider the junctures of socio-economic inequality, environmental protection, and public health. Certain disparities in exposures to environmental hazards may be related to or mediated by the degree of racial residential segregation, and these exposures may have important clinical and environmental health significance for populations across racial and class lines. Additional research, incorporating new models of exposure, should include segregation as a health risk factor. Moreover, while most research has focused on the health consequences of Black-White segregation in metropolitan areas, other minority groups may be similarly affected. Finally, the health impacts of rural segregation, particularly the experiences of Native Americans which were not addressed in this paper, should also be examined.

Although the literature on segregation and health has expanded significantly in recent years, studies that specifically address environmental health disparities are in their infancy. In general, most of this work has been limited to cross-sectional studies. Future research will require the development of longitudinal studies that look simultaneously at people and places—

that is, the trajectories of individuals in conjunction with the trajectories and evolution of the neighborhoods and metro areas where they live. These studies could also examine residential segregation in conjunction with segregation in other domains such as the educational system and the workplace.

A regional equity perspective is critical to understanding the interplay of individual factors and place-based measures of social inequality in shaping patterns of environmental health disparities (Morello-Frosch, 2002). Racial segregation and other SES disparities manifest themselves in major metropolitan areas along divides between the city core and the suburbs and across diverse neighborhoods (Gee and Payne-Sturges, 2004; Subramanian et al., 2005). Moreover, segmentation of housing markets, spatial mismatch of labor markets, and decentralization of metropolitan governance contributes to unequal access to economic opportunities, services, and the fragmentation of local control over land use and zoning in ways that affect community environmental health (Alshutler et al., 1999; Conley, 1999; Kain, 1992; Keister, 2000; Oliver and Shapiro, 1995; Preston and McLafferty, 1999). As discussed earlier, there is mounting evidence that various aspects of social inequality have contributed to the greater burden of environmental hazard exposure and health risks for communities of color and the poor. Social inequality, such as residential segregation, may affect the options that communities have to address environmental and health problems. For example, poverty may affect the likelihood of having health insurance, and linguistic isolation may hinder effective engagement with public officials. Therefore, it is necessary to incorporate these broad but significant indicators of place-based inequality and SES with individual-level factors into a comprehensive assessment of environmental health disparities. Ultimately, this enables policy makers and regulators to understand not only whether a community may be overburdened, but

also whether it has the capacity and resources to recover, reduce exposures, and protect public health.

How the regulatory community should address fundamental socioeconomic drivers of environmental health remains an open question. The capacity of environmental and public health agencies to proactively engage with these issues is constrained by legislative mandates that structure the priorities of their research, regulatory, and enforcement activities. Agencies that conduct research can begin to grapple with how to integrate place-based inequality measures and neighborhood-level SES measures with the individual-level factors that have traditionally commanded regulatory attention. Furthermore, research can begin to track the effects of segregation more systematically to determine the independent effects of segregation on environmental health, and how this form of place-based social inequality contributes to environmental health disparities. Indeed, segregation may disparately affect certain racial/ethnic groups more than others. It is also possible that segregation adversely affects the health of all racial and ethnic groups, even in areas where disparities might persist. For example, one study suggests that segregation affects physical inactivity risk, even for Whites (Lopez, in press). By developing indicators of social inequality and segregation and integrating these with environmental health data, regulatory agencies can generate the information necessary to inform regional authorities and community stakeholders about how to address some of the possible drivers of environmental health disparities and whether these relate to the built environment, transportation policies, fair housing, or land use planning. Although environmental and public health agencies may not be able to participate directly in these debates, they can generate the data and scientific information necessary to inform the discussion.

For example, suppose the body of evidence shows that segregation amplifies observed relationships between poor air quality and certain adverse health outcomes, and that segregation has worse health consequences for members of racial minority groups. Regulatory strategies such as air quality monitoring could be enhanced in segregated neighborhoods where poor air quality is a particular concern. Similarly, this information could help communities and local agencies understand how to target their efforts to reduce emissions from major sources. These targeted monitoring and emission source reduction strategies would have to be done in partnership with communities who would play a critical role in helping to identify smaller emissions sources that typically fall below the regulatory radar screen but that may be located near sensitive receptors (e.g., residential communities or schools). Communities can also help agencies balance the need for more effective regulation with the promotion of economic opportunities within a region. Previous agency-community collaborations of this sort include monitoring and source reduction efforts conducted by the California Air Resources Board and the communities of Barrio Logan in San Diego, and Wilmington in the Los Angeles area (Cal-EPA, 2003, 2004).

Rising interest within the regulatory community and the public about environmental health inequalities necessitates developing new analytical approaches that leverage existing data to sort through complex equity issues. Examining these issues through the lens of segregation can reveal connections between individual and place-based factors that shape health disparities, elucidate innovative methodologies to evaluate environmental justice concerns, and assess the viability of regional approaches to address racial equity in pollution control and prevention. NOTE: No human subjects or experimental animals were used in this study.

Appendix A: Summary Table of Measures for Five Segregation Dimensions

Measure	Dimension	Formula	Composition Invariant[1]	Multi-group Extension[2]	Spatial[3]
Index of dissimilarity	Evenness	$D = \Sigma[t_i \lvert p_{im}-P_m \rvert] / (2T P_m (1-P_m))$	yes	yes	no
Interaction index	Exposure	${}_mP_n^* = \Sigma[(t_i p_{im}/TP_m)(p_{in})]$	no	no	no
Duncan's delta index	Concentration	$DEL = \Sigma [\lvert (t_i p_{im}/TP_{m)} - (a_i/A) \rvert] / 2$	no	no	no
Absolute centralization index	Centralization	$ACE = \Sigma[X_{i-1p}A_i] - \Sigma[X_{ip}A_{i-1}]$ tracts sorted by land area $X_{ip} = \Sigma[t_i p_{im}]$, tracts from *0* to *i* $A_{ip} = \Sigma[a_i]$, tracts from *0* to *i*	no	no	yes
Spatial proximity index	Clustering	$SP = (TP_m P_{mm} + TP_n P_{mn})/NP_{tt}$ $P_{mn} = \Sigma \Sigma [(t_i p_{im}\, t_j p_{jn}\, c_{ij})/TP_m TP_n]$ $c_{ij} = e^{-d_{ij}}$ d_{ij} = distance between tract *i* and tract *j*.	no	no	yes

T = number of metro area residents
t_i = number of residents in tract *i*.
P_m = proportion of metro area residents of racial/ethnic group *m*.
p_{im} = proportion of tract *i*'s residents of racial/ethnic group *m*.
A = land area of metro area
a_i = land area of tract *i*.

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