Relationship between Premature Mortality and Socioeconomic Factors in Black and White Populations of US Metropolitan Areas

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SYNOPSIS

Objective. This ecologic study examined the association of mortality with selected socioeconomic indicators of inequality and segregation among blacks and whites younger than age 65 in 267 US metropolitan areas. The primary aim of the analysis was to operationalize the concept of institutional racism in public health.

Methods. Socioeconomic indicators were drawn from Census and vital statistics data for 1989–1991 and included median household income; two measures of income inequality; percentage of the population that was black; and a measure of residential segregation.

Results. Age-adjusted premature mortality was 81% higher in blacks than in whites, and median household income was 40% lower. Income inequality, as measured by the Gini coefficient, was greater within the black population (0.45) than within the white population (0.40; $p < 0.001$). To confirm that the proxy socioeconomic variables were relevant markers of population health status, regression analysis was performed initially on data for the total population. These variables were all independently and significantly related to premature mortality ($p \leq 0.01$; $R^2 = 0.74$). Income inequality for the total population was significantly correlated with premature mortality ($r = 0.33$). Black ($r = 0.26$) and white ($r = 0.20$) population-specific correlations between income inequality and premature mortality, while still significant, were smaller. Residential segregation was significantly related to premature mortality and income inequality for blacks ($r = 0.38$ for both); among whites, however, segregation was modestly correlated with premature mortality ($r = 0.19$) and uncorrelated with income inequality. Regional analyses demonstrated that the association of segregation with premature mortality was much more pronounced in the South and in areas with larger black populations.

Conclusion. Social factors such as income inequality and segregation strongly influence premature mortality in the US. Ecologic studies of the relationships among social factors and population health can measure attributes of the social context that may be relevant for population health, providing the basis for imputing macro-level relationships.

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It is generally recognized that the health of populations is determined primarily by the structure and organization of a society, including the level of scientific knowledge and technological capacity as well as operating social values. In the United States, the black population is at higher risk than the white population by most measures of morbidity and mortality, with disparities between black and white groups for some indicators reaching two- and three-fold.[1] Although these disparities have historically been ascribed to differences in socioeconomic status (SES), the complex mechanisms by which social advantage or disadvantage influence health are not well understood. As researchers attempt to unravel these complexities, it is important that the characteristics of individuals not be isolated from the social settings into which they were born and in which they have lived out their lives. While the effects of social processes may be mediated by individual factors and characteristics, they primarily operate at the local geographic level, where they are embedded in the socially structured relationships that affect the population as a whole.[2,3]

Unfortunately, conceptual clarity on the routine and uniform collection of SES data does not exist within the social science research community, creating difficulty in the interpretation of studies addressing SES. Likewise, variation in the strength of predictors of SES differentials in health and sometimes in the direction of the relationship have been found to be related to how SES is defined, the outcome of interest, the age and gender of the cohorts, and the geographic regions under study.[4–12] While the presence of a relationship between SES and health has remained consistent over time, notable changes have taken place in risk profiles and causes of morbidity and mortality across social strata. Given this complexity, SES should be measured at various levels and along several dimensions.

Racial/ethnic differentials cannot always be satisfactorily attributed to SES or social class as it is routinely measured through education, income, or occupation.[1] A number of researchers have argued in favor of including measures of interpersonal and institutional racism in studies of race and health.[13–15] As a concept, institutionalized racism facilitates the identification of structural impediments and promotes the exploration of disparities within a social structure. Racial segregation across a number of domains such as education, residence, and occupation has been the hallmark of institutionalized racism in the US. The landmark study by Yankauer demonstrated increasing mortality rates for blacks with rising residential segregation in New York City in the 1940s.[16] In the last decade, interest in the association of residential segregation and health has increased dramatically. McCord and Freeman found standardized mortality ratios for blacks in Harlem to be two to three times as high as those of whites, and significantly higher than for other blacks nationally.[7] A recent study based on survey data from 39 US states suggests that racism measured as an ecologic characteristic is associated with elevated mortality for both blacks and whites.[17] The white infant mortality rate in two of the most segregated cities in the country was found to be twice the rate for white infants in any other city,[6] and the black-white gap in infant mortality has been found to be narrower in less segregated cities.[6,9]

While there is no clear agreement on the basic social determinants of health, or on what intervening pathways produce their effects, there is a growing consensus that these broad social processes play a critical role in shaping population health. The present ecologic study examines the relationships among indicators of SES and institutionalized racism, including measures of income inequality and residential segregation, and their influence on patterns of premature mortality (< 65 years of age) for black and white populations of metropolitan areas of the US.

METHODS

Data sources

The data used for these analyses were obtained from a variety of US government sources, as described in previous publications.[12,18] The National Center for Health Statistics provided a data file consisting of data on deaths, population counts, the racial composition of neighborhoods, and household income for the years 1989–1991.

Data were initially obtained on the 282 Metropolitan Statistical Areas (MSAs), as defined by the Office of Management and Budget, which correspond to the largest cities and surrounding economic areas. These units ranged from the New York City metropolitan area (population = 18 million) to Enid, OK (population = 57,000); the average population size of the MSAs was 610,000 according to the 1990 Census. The population included in the 282 MSAs represented 73% of the US population in 1990. The complete set of variables of interest was available only for the "white" and "black" populations. Analyses of the total population were based on data for blacks, whites, and all other racial/ethnic groups combined.

A dataset was created containing the values at the MSA level for the study variables, i.e., mortality rates, median household income, indices of income inequal-

ity and residential segregation, and percentage of the population that was black. The data that were analyzed therefore represent an average (e.g., household income) or a proportion (e.g., percent black) for each of the MSAs. Inspection of the data revealed that 15 MSAs had missing or implausibly extreme values for the calculated variables, and the analytic dataset was accordingly reduced from 282 to 267 MSAs.

Exploratory analyses confirmed the general finding in the literature that the relationship between SES measures and mortality is greatly attenuated in the older age group; thus the mortality outcome indicator chosen was mortality of individuals younger than age 65, referred to as "premature mortality." Using US Census household income data, we measured economic inequality with two indices: the share of income going to the lower half of the income distribution and the Gini coefficient. These measures were highly correlated ($r = -0.93$ for whites and $r = -0.97$ for blacks) and gave equivalent results in all analyses. The Gini coefficient, based on a Pareto distribution, was chosen to be consistent with other studies in this field. The Gini coefficient ranges from 0 to 1 and represents the degree of unevenness with which reported income is distributed among households, with the lowest possible value (0) reflecting complete equality and the highest (1) inequality.

Residential segregation was measured using an index of dissimilarity, representing the unevenness of the black-white racial distribution of households by Census tract, and a single measure of residential segregation was generated for each MSA.[18] This indicator also has a range from 0 to 1, with the maximum value reflecting complete segregation.

Since we anticipated that the relationships among exposure and outcome variables might vary across groups of MSAs stratified by population size and location, variables for population size and location were kept in the final dataset (i.e., the proportion of the population that was black and the Census-defined geographic region of the US).

Analysis strategy

As noted, the primary purpose of the study was to identify the extent to which the observed relationships between premature mortality and socioeconomic indicators were consistent with institutional racism, as reflected in the known historical pattern of economic discrimination and housing segregation experienced by black Americans.[19] The analysis strategy, therefore, sought to capture the patterns relating variation in premature mortality to income inequality and segregation, i.e., to identify the extent to which these proxies for institutional racism were associated with premature mortality and whether these relationships were population-specific (i.e., more pronounced for the black population than for the white population). We recognized that each of these measures of inequality and segregation is a composite indicator, removed from its social context and causal processes by several levels of averaging and abstraction. Thus, residential segregation represents the average result of a range of social policies, from discrimination in the mortgage market to zoning laws. Likewise, the income variable is averaged at the level of the MSA. Each of these factors operates within a specific geographic context, where, for example, social services might mitigate or aggravate its effect.

Geographic variation across this sample of population units was expected to create some colinearity of the exposure variables. For example, MSAs with larger populations would be expected to have greater median incomes, and MSAs in the South would be expected to have both lower median incomes and larger percentages of black residents than MSAs in other regions of the country. To investigate this question, we first conducted multivariate regression analysis to determine whether the socioeconomic variables were significantly and independently associated with premature mortality. Subsequently, we examined the exposure variables individually to determine the pattern of the relationships between these variables and each of the other exposure variables and premature mortality. Regression analyses were weighted by the size of the population in 1990. We next performed race-specific analyses, e.g., using black premature mortality rates in a model that included median household income and the degree of inequality in the income distribution among blacks, as measured by the Gini coefficient. The same values for segregation and percent black were used in both the black and white race-specific models since they are attributes of the total population.

In correlation analyses, unweighted mean and proportions were used. At this stage, a secondary goal was to determine whether the black and white populations had distinctly different patterns of inter-relationships of the factors considered, or whether the combined population pattern was the determining process. The third step was to explore whether these inter-relationships differed across the strata defined by area size, percent black, and geographic region.

Calculations were performed in Stata, Version 6.0.

RESULTS

Findings from the descriptive analyses were as expected for all measures. Age-adjusted mortality among blacks

Table 1. Distribution of socioeconomic variables and premature mortality, 267 Metropolitan Statistical Areas, 1989–1991

	White		*Black*		*Total population*	
Measure	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
Age-adjusted premature mortality per 100,000 population, 1990	245	30	444	100	264	41
Median household income (dollars)	29,837	4,568	18,471	4,909	28,039	4,414
Income inequality[a]	0.40	0.02	0.45	0.04	0.41	0.03
Income share to lower half of population	0.22	0.01	0.19	0.03	0.22	0.02
Segregation index[b]	—	—	—	—	62	12
Percent black	—	—	—	—	10.5	9.9

[a]Gini coefficient

[b]Index of dissimilarity

SD = standard deviation

<65 years old in 1989–1991 was 81% higher than among whites (Table 1). The median income among black households for the study years in this sample of MSAs was 40% lower than among white households. As shown in Figure 1, the income data for black and white households clearly come from different underlying distributions. Income inequality, as indicated by the Gini coefficient, was higher within the black population than within the white population, as was the share of income received by households in the lower half of the income distribution. While the absolute racial differences for these indicators do not appear to be large, when the more stable white estimate is taken as the reference, the black-white difference corresponds to about two standard deviations, again demonstrating two separate distributions (Figure 2). The Gini coeffi-

Figure 1. Distribution of median income for black and white households, 267 Metropolitan Statistical Areas (MSAs), 1990

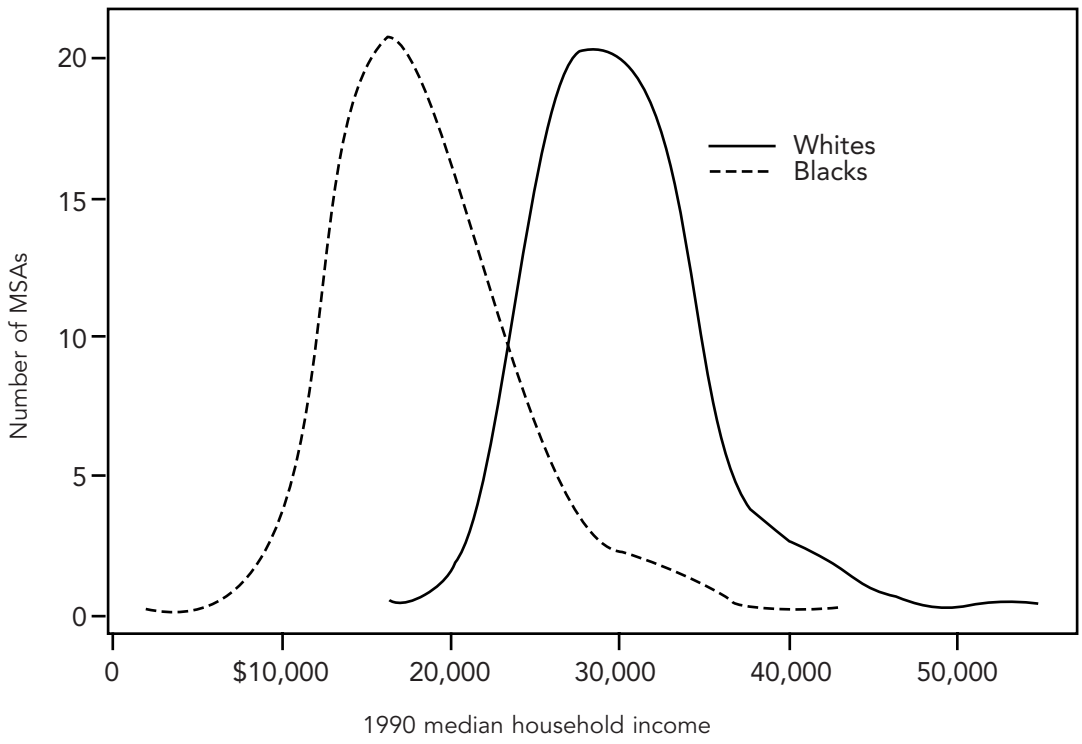
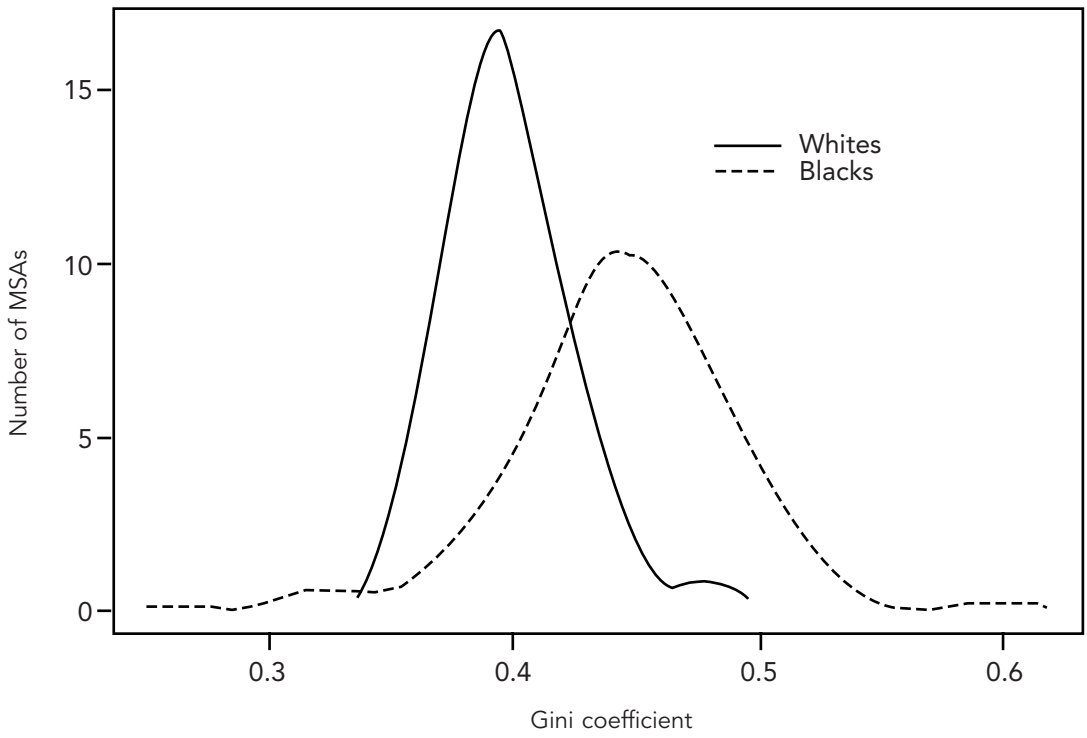


Figure 2. Distribution of income inequality, as measured by the Gini coefficient, for the white and black populations, 267 Metropolitan Statistical Areas, 1990



cient across all MSAs ranged from 0.35 to a maximum of 0.50.

The average dissimilarity index for racial housing patterns was 62%, with a range from 31% to 87%, confirming that most blacks in these MSAs live in highly segregated neighborhoods.

In a population-weighted multivariate model that included median household income, income inequality as measured by the Gini coefficient, degree of segregation, and percent black, each of these independent variables was significantly related to premature mortality for the total population, and the model R^2 was considerable, at 0.74 (Table 2). When whites and blacks were considered separately, however, with population-specific economic indicators used as the predictors, the magnitude and direction of the relationships varied. In models with premature mortality rates among whites as the outcome variable, the segregation index was no longer related to premature mortality ($p = 0.4$); the relationship between premature mortality and all

Table 2. Results of regression model using data on socioeconomic variables and premature mortality for 267 Metropolitan Statistical Areas, 1989–1991

Variable	*Coefficient*	*SE*	p
Median household income	–1.12	0.24	0.0001
Income inequality[a]	4.09	0.50	0.0001
Segregation index[b]	0.28	0.11	0.015
Percent black	2.98	0.15	0.0001

NOTE: Model $R^2 = 0.74$

[a]Gini coefficient

[b]Index of dissimilarity

SE = standard error

other predictors remained highly significant ($p < 0.001$). When premature mortality among blacks was the outcome, the association between premature mortality and median household income became positive ($p < 0.001$), while the associations with other predictors remained significant with the same sign as in the total population model. This inconsistency in the analyses among blacks resulted from concordance of higher mortality with higher income in the very large metropolitan areas. In all of the models, the relationship between percent black and premature mortality remained highly significant for both whites and blacks. The R^2 for the race-specific models declined from the value of 0.74 that was observed for the total population to 0.45 for blacks and 0.35 for whites.

The pattern of inter-relationships among the indicators was expected to be multi-dimensional and to include some effect of stratification by area size, region, and percent black. To avoid the constraints of assumptions that are required for multivariate modeling, pair-wise correlations of each of the relationships were examined separately. While this approach introduces the potential error that may arise from making multiple comparisons, the main goal was to identify patterns—i.e., sets of relationships—within and across the populations. Among these possibilities were that income inequality would be an important correlate of premature mortality for both whites and blacks while residential segregation would have a stronger association with premature mortality among blacks. The multivariate results were subsequently examined for consistency in relation to the multivariate models above and in the stratification analysis that follows. Correlations in which the absolute value of r was < 0.2 were ignored as inconsequential.

Premature mortality rates among blacks and whites were only moderately correlated (Table 3; $r \approx 0.4$). Variation in mortality in the two populations was associated with variation in population-specific median income ($r \approx -0.3$) and inequality ($r \approx 0.2$). While median household income among blacks and whites was reasonably correlated ($r \approx -0.6$), premature mortality among blacks was not associated with median income for white households. As anticipated, the pattern of relationships associated with the segregation index showed the greatest heterogeneity between races. The association between premature mortality and segregation was twice as strong for blacks ($r \approx 0.4$) as for whites ($r \approx 0.2$), and both median household income and the degree of income inequality were more strongly related to segregation among blacks than among whites. The percent of the population that was black, which in this dataset is a marker of both geographic region and population size, was associated with higher mortality for blacks and whites, greater overall income inequality, and greater segregation.

Some of the socioeconomic variables in this study are highly correlated with one another, as seen in the correlation matrix (Table 3). If these variables are also interdependent, using them in a large multivariable analysis in an attempt to adjust for confounding would likely result in an unstable model and fail to assign the proper value to the variables of interest. Given that population health is an interdependent and contingent state, rather than the sum of independent risks, the utility of adjusting for confounding in the analysis of broad social forces to determine the underlying process can be questioned. Multivariate modeling can control for confounding, but may be better suited to identifying residual effects than isolating primary causal pathways. This problem arises in part because some of the factors may lie upstream to others, or some may have different relationships in different strata, and these effects can be hard to identify in regression models. Stratification can help disentangle related processes using fewer assumptions. We first used the nine regional categories defined by the Census Bureau as the basis of stratification. We next divided the cities into tertiles based on the percent black and the population size to re-examine the pattern of relationships among the variables.

Inspection of the data stratified by region of the US suggested that the South varied from the rest of the country. When the regions were rank ordered on the basis of the percentage of the population that was black, thereby recapitulating the historical migration of the black population out of the South, a clear pattern emerged in the association between segregation and premature mortality (Table 4). In the regions where blacks represented more than 10% of the population, all of which were in the South, premature mortality and segregation were consistently related. These regional contrasts were also observed in the degree of segregation.

We next divided the cities into tertiles based on the percent black and the population size to re-examine the pattern of relationships observed in Table 4. A clustering of larger correlations in the set of variables that included income inequality, segregation, and premature mortality was again seen in areas with higher percentages of black residents; correlations among these three variables ranged from $r = 0.14$ to $r = 0.19$ in the lowest tertile and from $r = 0.34$ to $r = 0.55$ in the upper tertile. In all the metropolitan areas in the upper tertile, more than 11% of the population was black. A similar, albeit less consistent, pattern of correlations

Table 3. Correlations among socioeconomic variables and premature mortality, 267 Metropolitan Statistical Areas, 1989–1991

	Age-adjusted premature mortality per 100,000 population, 1990		*Median household income, 1990*		*Inequality*[a]			*Segregation index,*[b] *1990*	*Percent black*
Variable	*White*	*Black*	*White*	*Black*	*White*	*Black*	*Total*		
Age-adjusted premature mortality per 100,000 population, 1990									
White	—								
Black	0.44								
Median household income, 1990									
White	−0.32	0.04							
Black	−0.34	−0.30	0.56						
Inequality[a]									
White	0.20	0.21	−0.30	−0.27					
Black	0.15	0.26	−0.17	−0.64	−0.16				
Total	0.33	0.32	−0.26	−0.41	0.91	0.29			
Segregation index,[b] 1990	0.19	0.38	0.16	−0.25	−0.06	0.38	0.08		
Percent black	0.28	0.33	0.16	−0.23	−0.00	0.14	0.36	0.28	
Population size	0.01	0.14	0.45	0.23	0.12	0.01	0.11	0.22	0.09

NOTE: Overall $r \geq 0.12$, $p \leq 0.05$

[a]Gini coefficient

[b]Index of dissimilarity

was observed in the comparison of the lower and upper tertiles based on population size (data not shown).

DISCUSSION

Based on this sample of 267 MSAs, we confirmed earlier findings of the importance of median household income and income inequality as correlates of premature mortality risk, and extended these observations by including the impact of racial segregation and by conducting race-specific analyses that examined the black and white populations separately. Economic measures—namely, median household income and income inequality—were associated with premature mortality at the level of $r \approx 0.2$ to $r \approx 0.3$. Residential segregation was also significantly associated with premature mortality, although more strongly among blacks than whites; this association was strongly influenced by those geographic areas of the country that had proportionately larger black populations, i.e., the South and the larger MSAs. Both the percentage of the population that was black and income inequality were consistent predictors of premature mortality for blacks, whites, and both groups combined. The strong association between white mortality and the percentage of the population that is black has been noted before and invites further speculation as to its meaning.[19,20] The most straightforward interpretation would be that policies put in place that restrict social resources for blacks also influence whites; this interpretation, of course, cannot be supported by direct evidence from the present study.

Some variation was noted when the race-specific regression models were compared. The effect of residential segregation was not significant for whites. Also, as noted, blacks in larger MSAs had higher median incomes and higher mortality than blacks in other MSAs; when this association was weighted by population size the negative correlation between income and mortality in the sample of smaller MSAs was eliminated. The greater impact of segregation in the South and when more than 10% of the population was black

Table 4. Regional variation in the relationship between segregation and premature mortality, 267 Metropolitan Statistical Areas, 1989–1991

Region	*Percent black*	*Segregation index*[a]	*Correlation of premature mortality with segregation: black population*	*Correlation of inequality with segregation: overall*
South East	20	66	0.73	0.36
South Atlantic	18	64	0.53	0.41
South West	14	60	0.40	0.51
North East	9	70	0.51	0.33
Middle Atlantic	5	70	0.49	0.20
North West	4	59	0.31	0.08
New England	3	56	0.35	0.48
Pacific	3	52	0.26	0.07
Mountain	2	49	0.19	0.23

[a]Index of dissimilarity

is consistent with an effect of concentrated poverty, as suggested by Massey.[19] Collins and Williams observed a similar association between social isolation and mortality for blacks and whites that was more pronounced in MSAs with a high index of dissimilarity.[20] The strongly negative social consequence of segregation needs to be investigated directly in order to understand these observations.

We did not attempt to quantify the size of the impact of the social factors on premature mortality. Inferring the attributable risk for premature mortality associated with low income and segregation from cross-sectional data would be difficult. However, the findings are consistent with ongoing national trends toward increasing educational and geographic disparities as SES inequalities increase.[1,21,22] US vital statistics, for example, demonstrate that the rate of decline in all-cause mortality over the decade from 1985 to 1995 was 15% among individuals with education beyond high school, compared to 7% among those with only a high school degree or less.[23] Likewise, the largest black:white mortality ratios were found in federally designated core urban areas (i.e., a mean of 1.6 for core urban areas vs a mean of 1.4 for rural areas in 1995–1997).[23] As recently reviewed by Massey, these large US cities are now uniformly characterized by hypersegregation, a phenomenon that had been restricted to rural areas of the South in the first half of the 20th century.[19] The cross-sectional relationships we report are therefore likely to be a major component of the process shaping current SES differentials in health.

Increased interest in the relationship between health and social factors has highlighted the importance of using appropriate methodological approaches. Analytic models should represent to the degree possible the dynamics of the processes generating the data, and the selected variables, whether individual, multi-level, or ecologic in character, must correspond to the research question being asked for a particular level of analysis. Ironically, while ecological studies are often thought to suffer from significant group-level confounding, and are therefore viewed with skepticism, the primacy of the "individual" in epidemiologic research has allowed the social construction of variables that are attributed to individuals to be ignored. The individualist framework sees race and ethnicity (as well as class and gender) as individual attributes rather than characteristics embedded in institutional structures.[24]

Given that the individual and ecological context levels are distinct, their properties and characteristics cannot be entirely predicted by each other.[25–27] In studying social, economic, and cultural features of Glasgow neighborhoods, MacIntyre and her colleagues demonstrated that although individual social class may be correlated with where one lives, the specific features of the areas themselves are independently related to population morbidity and mortality. Among others, these features included: networks of social support, crime rates, quality and tenure of housing stock, and neighborhood reputation.[28] Social context has also been found to exert independent effects in studies relating to violent crime,[29] initiation of intercourse and contraceptive behavior,[30] and physical activity,[31] among other subjects. At the state level, in analyses adjusted for health service variables, structural characteristics across social, economic, and political dimensions were found to account for a significant proportion of the variance in infant, neonatal, and postneonatal mortality rates.[32] Kunst and Mackenbach found that variations in premature mortality within

and between countries were partially explained by differences in the egalitarianism of social and economic policies, as indicated by measures of education and occupation.[33] After controlling for baseline health status, a follow-up study in the United States found significantly elevated risk of mortality across several causes of death for people ages 25–54 years residing in poverty areas.[34] These studies contribute to the mounting evidence that health outcomes cannot be explained solely by individual characteristics and present challenges to further identify and disentangle the underlying causal social forces.

In the present study, income inequality and segregation were taken as proxies for a complex set of social relationships that are structured by and reflective of daily experience. While the specific characteristics of these relationships change with evolving social conditions, their essential purposes remain. For example, as noted, intense residential segregation was once a feature primarily of the rural South but was recreated under new conditions in urban centers of the North.[19] The social role of racial discrimination in these regions varies but shares the same function of undergirding high levels of economic exploitation. Some component parts of these relationships are apparent on the surface, corresponding to the distribution of income, schools, and housing opportunities; others are hidden beneath the surface of the social system, e.g., the fact that white privilege is inherent (regardless of class) and that meritocracy remains a myth within our social structure.[35,36] Although it has been argued for more than a century that racism is a major contributor to the poor health of the black population in the US, scientific tension continues to revolve around etiology and around whether an individual or a societal approach would be most effective in redressing these disparities.

What value do the observations in the present study have to public health? It has been suggested that the method of inquiry in social science is different from that used in biology.[37] Social scientists, in this view, proceed from a theoretical framework, make observations about historical events, and attempt to give these events meaning. In biology, on the other hand, the scientist uses empirical methods to gather facts, in order to discover truths about the natural world. On further reflection, however, it is clear that the distinction is illusory, created not by inherent properties of these disciplines but by their socially defined function. Biological facts and associated generalizations do not exist independent of our ability to observe them, the questions behind the experiments that create them, or their utility as tools or explanations. The interpretation of social forces is likewise contingent on our descriptive resources and prior assumptions and hypotheses. In both cases, the observed "truth" or the imputed "meaning" do not belong to some external reality but signify relationships in a socially determined context.

Discrimination and racial segregation are the hallmarks of institutionalized racism in this country, which has been one of the most important historical determinants of the distribution of resources, power, and privilege.[1,3,38–40] We found that the percentage of the population that is black and income inequality were significant predictors of premature mortality in both blacks and whites, and segregation was strongly associated with premature mortality for blacks. These relationships conform to the view that racism is a noxious influence on the health of all members of society. Unfortunately, the US debate about the causes of racial differentials in health is all too often focused on individual-level attributes; this overly restrictive focus often suggests that change must occur primarily at the individual level. Clarifying the theoretical assumptions and hypotheses that shape this discourse will help distinguish between inquiry that justifies existing conditions and inquiry that broadens our understanding of these observations to promote change and improve population health.

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